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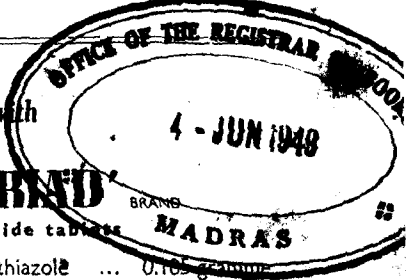
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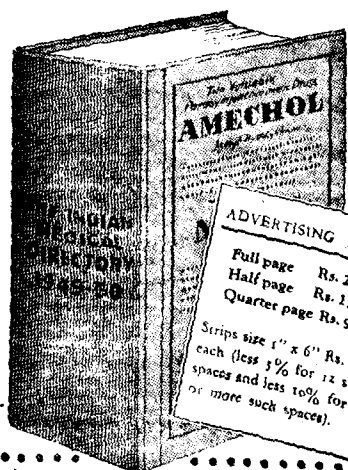
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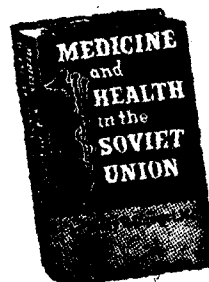
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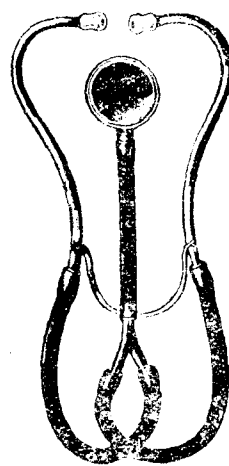
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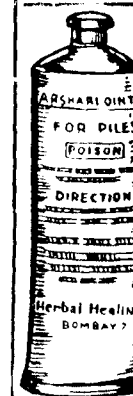
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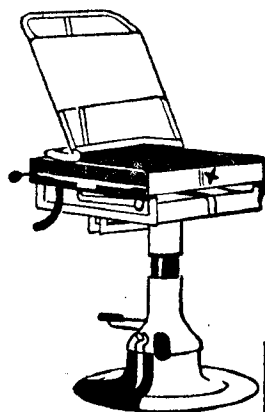
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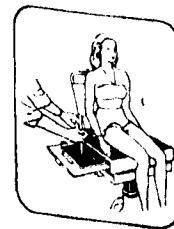
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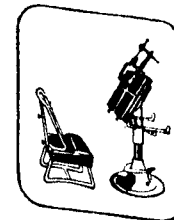
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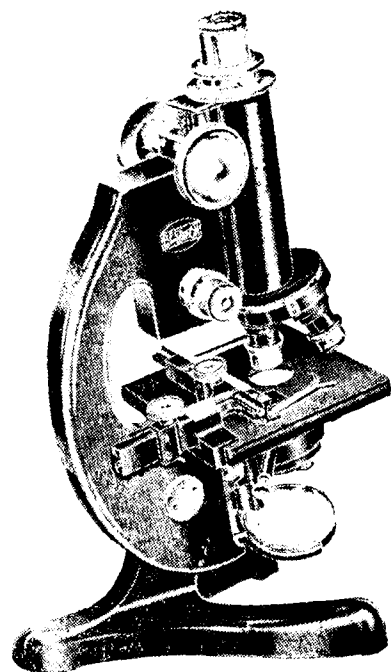
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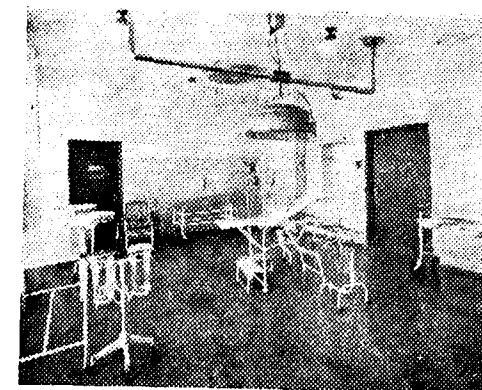


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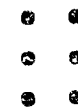
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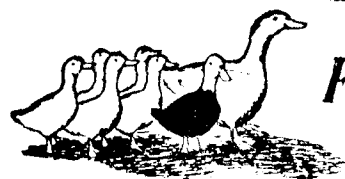
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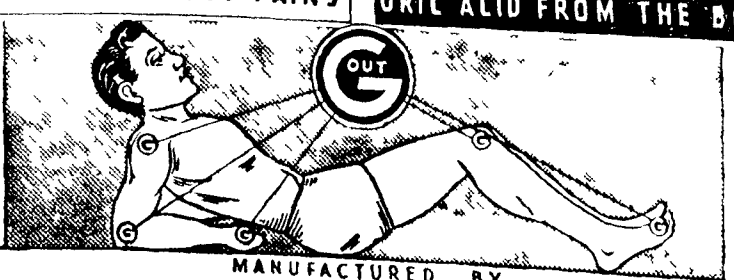
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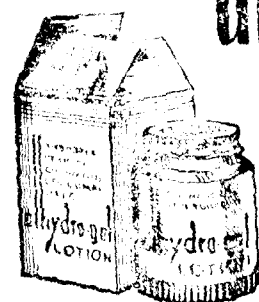
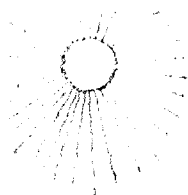
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Original Articles

Movements of the Eyelids in Blinking*

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Introduction

IN a paper read recently at the Tenth Conference of the All India Ophthalmological Society held at Madras, the author (1949) made some observations on the mechanism of eyelid closure in blinking. Some additional data have since been gathered and this paper is based on this material dealing with some aspects of the blinking act.

Closure of the eyelids might be either a forceful occlusion of the eye, or a gentle closure of the lids. In forceful occlusion, a sphincteric screwing up movement takes place and the whole orbicularis oculi muscle, including the orbital and palpebral parts, contracts. Apart from willed action, forceful occlusion takes place as a protective reflex when the offending stimulus is sufficiently strong. Gentle closure of the eyes is called blinking and it involves only the palpebral part of the orbicularis. Blinking may occur (a) as a voluntary action, (b) as a protective reflex, and (c) as a periodic involuntary action. The word 'wink' is used for voluntary closure of one eye to express disdain or connivance, and it does not come within the scope of our discussion here. In blinking, on the other hand, both eyes take part in the action. Protective blinking is caused by offending stimuli transmitted by the V nerve, or a sudden bright light or loud sound or proximity of objects portending danger to the eye. Involuntary blinking takes place as a periodic movement in all normal persons at all times except during

* Specially contributed to THE ANTISEPTIC.

sleep or complete relaxation. This occurs not only when the lids are kept open, but also in a latent manner occasionally in the form of a twitch of the closed lids, if the lids are kept gently closed and the person is awake and mentally alert. The precise cause of involuntary periodic blinking is not fully known and explained.

Scope of the Investigation

There are some aspects of the blinking act on which previous work has been done. The time taken for each blink, the periodicity, its variations in different conditions of age, mental state, disease, drug action, etc., have formed subjects for investigation. Studies have also been made on animals (Blount, 1928). We know that blinking is protective in its function and secondarily serves to moisten and cleanse the cornea and to help the circulation of aqueous and tears.

Authors have also speculated from time immemorial on the primary causation and significance of periodic involuntary blinking. In the *Ramayana* dating prior to the first millennium B.C., the Indian sage Valmiki expounds that due to a benign agent entering into the eyes as a nerve impulse all living beings blink their eyes, often, for recovery from fatigue. It is known that the Berger rhythm in the electroencephalogram is disturbed by the opening of the eyes. Ponder and Kennedy (1928) consider that in all probability the blink constitutes a kind of relief mechanism whereby nervous energy otherwise unutilised passes into this highly facilitated path. But the writer has to confess his inability to comprehend what is nervous energy, how it is produced and stored, and how it could pass or overflow into any facilitated channel. In this paper it is not proposed to tread on this hypothetical ground. The scope of this investigation is limited to a study of the duration of the blinking act in the normal subject, and to the mechanism of eyelid closure as observed in still photographs called from motion picture films taken to analyse blinking movements.

Observations

Duration of the blink.—The time taken by a blink was determined by a kymograph. A strip of adhesive tape attached vertically to the upper eyelid was connected to a string passing over pulleys and moving a lever writing on smoked paper on a revolving drum. The time tracing was made by a tuning fork of 100 frequency. The average duration of a blink was 0.23–0.25 second. Garten (1898, cited by Duke-Elder, 1942) gives for each blink 0.3–0.4 second. It was not considered feasible to apportion very precisely the lowering time, the closed time and the time for raising the lid as the wave had no well marked apical plateau. Approximately these times were 0.1, 0.025 and 0.12 second, respectively. But emphasis need not be laid on the accuracy of the time moities for the three components of the blinking act, because, in any

there is considerable difference between the right eye and the left eye in the same subject as is explained below.

Mechanism of lid closure in blinking.—The analysis of the mechanism of lid closure is made from stills obtained from specially taken motion picture films. Four subjects were filmed and many blinks for each subject came on record. The subjects were normal intelligent people and were aware that the photographs were for a study of blinking. Hence whether a particular blink in the film was voluntary or involuntary, is more than one could say. But I presume it makes no difference as far as the present inquiry is concerned. It appears that the mechanism of gentle closure of the eyelids is manœuvred by the palpebral part of the orbicularis in a similar manner, both in voluntary and involuntary blinking, though part of the nervous pathway is admittedly different in the two cases. Garten in 1898 and Weiss in 1911 (cited by Duke-Elder, 1942) studied photographic records for noting the time for each blinking movement, and the shortest interval between consecutive voluntary blinks. I have not had access to their original papers; from the citation (Duke-Elder, 1942) it does not appear that these authors were concerned with the particular aspects of the mechanism of blinking which have attracted my scrutiny. The movements making up a typical blink are reproduced in fifteen stills in Plate I. (*vide picture*).

It is noticed that the lower eyelid moves only very slightly in blinking. Assuming that the outline of the nose and forehead is relatively immobile, tracings were taken on transparent paper from the pictures of the eye when it is open, partly closing and completely shut. On superimposing these tracings it was noticed that the margin of the lower eyelid has only very slight migration upwards in gentle closure of the eyelids.

Regarding the movement of the upper eyelid which takes the major share in the blinking movement, its movement has been compared to the dropping of the visor of a helmet (Whitnall, 1921). This comparison is very suggestive and explains the movement in an easily understandable manner. In the first phase of blinking the writer can confirm that this movement takes place. When the eyelids are open the tarsal part of the upper eyelid is telescoped deep to the septal part at the superior palpebral fold, and during closure the tarsal part of the upper eyelid is let down like the visor of the helmet. But this mechanism does not fully and completely explain the action that takes place.

For if this should be the complete action we should expect the palpebral fissure to be gradually converted to a narrowing slit of identical linear dimension and to be totally obliterated at one instant. But the stills from cinefilm reveal that towards the terminal phase of lid closure the narrowing palpebral fissure gradually becomes foreshortened *i.e.*, less long, due to a progressive contact

of the upper and lower lid margins proceeding from the lateral angle medially; so that finally a small slit remains near the medial part of the ciliary portions of the lid margins before total closure is effected. This terminal movement, I compare, for easy understanding, to a 'zip' movement (of course without the sliding can) creeping from the lateral to the medial side along the narrowing palpebral fissure.

This movement is cutaneously beneficial, as it can produce a gradual displacement of any unfixed foreign body, step by step, towards the medial angle (the lacus lacrimalis) with successive acts of blinking.

As regards an explanation for this terminal stage action from principles of muscle mechanics, I can only hazard a suggestion. The concentric elliptical muscle fibres of the orbicularis palpebrarum do not contract all fibres together at one instant, but a wave of the ones situated nearer the lid margin. Further the muscle fibres at the lid margin, both the sub tarsal and ciliary parts of the orbicularis palpebrarum, also help in negotiating this manoeuvre.

One can take a parallel from the action of the orbicularis oris muscle round the mouth. It has, however, to be remembered that the orbicularis of the eye has a fixed bony and ligamentous attachment medially and its palpebral fibres are only partial ellipses lateral angle of the eye can well be compared to the lateral angle of the mouth. But while the eye has a stable medial angle, perhaps the median point of the oral lip margins corresponds to the stable point of the mouth. Voluntarily one can produce a 'zip' movement in the case of the mouth, for example, a cigarette can be held at or near the angle of the mouth while keeping the central part of the oral aperture patent for phonation. In normal closure of the mouth also a kind of 'zip' action takes place, progressing from either lateral angle of the mouth, and finally finishing itself at the median point. Further expatiation on this point is not relevant and necessary to the subject on hand. It is suggested that a similar closure of the palpebral margins takes place progressing from the lateral to the medial side up to the lacus lacrimalis.

Movements of the Eyelids in Blinking

(Continued from page 323)



PLATE I. Still photographs from film of normal blinking. Read each vertical row from above downwards, and the vertical rows from left to right.

[Vide page 323.]

being injured by filings from a machine or a splash of corrosive fluid, run by the left eye when compared with the right eye, in normal righthanded people.

It might also be mentioned that the part of the eyeball most liable to injury by exposure, *i.e.*, the part last being covered and earliest exposed in the case of both the eyes is the sclera medial to and below the lower edge of the cornea.

I express my extreme thankfulness to Dr. C. Venkataramiah, Professor of Physiology, for help in taking the kymograph tracings, and to Mr. K. Ramnoth, Cinephotographer, and Dr. M. N. Guruswamy, Assistant Professor of Pharmacology, who took the various motion films that have formed the material for this study.

Summary. The movements of eyelids in blinking have been studied. A blink lasts 0.23-0.25 second on the average. The mechanism of lid closure is analysed by cinematograph recording of the action. Stills from moving film are presented as illustrations. The author finds that during gentle closure of the eyelids the lower lid moves only very little, that the migration of the upper lid during the first phase of the movement is like the dropping of a visor of a helmet, and that during the terminal phase of the blinking act the narrowing palpebral fissure becomes obliterated by what may be called a 'zip' action *i.e.*, closure gradually proceeding from the lateral angle of the eye towards the medial side. It is also observed that though both the eyes blink together, one eye, usually the left one, lags behind in the closure, remains shut for a shorter while, and opens earlier. This is probably correlated with righthandedness.

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Penicillin in Dentistry*

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SIR ALBERT FLEMING first reported on Penicillin prepared from a mould, *Penicillium Notatum*, in 1929, and Florey applied it clinically in 1939 and established the Oxford units. It was only in 1942 that the American reports of this new drug appeared and after the War in 1945 was Penicillin released for unrestricted sale and only then was it possible for our general practitioners and dental surgeons to try it out on their routine cases. Due to advances in production, purified Crystalline Penicillin is now used in all preparations and refrigeration is no longer required. The one difficulty in the use of Penicillin is its rapid excretion which has necessitated its administration at three hours' intervals. Rapid elimination of Penicillin by the kidney tubules, primarily accounted for the inability to maintain high plasma concentrations of the drug. To eliminate the necessity of rapid injections Romansky developed the preparation of Penicillin in oil and beeswax. Such a preparation of 300,000 units of Penicillin maintains an effective Penicillin plasma level for a period of approximately 24 hours. Recently, Caronamide has been discovered which reduces the rate of its elimination. With its addition into Penicillin it is possible to maintain high Penicillin plasma concentrations and beeswax and oil are not necessary. Wyeth Bros. have recently put out Wycillin which is Crystalline Penicillin G and Procaine for aqueous injection. 300,000 units of this mixture added to 1 c.c. of sterile water maintains the blood levels for 24 hours. The Procaine eliminates any post-operative pain. Then there is Intracillin by Warner, which is Crystalline Potassium Penicillin G and Epinephrine in oil which does away with the unpleasantness of the wax. These new preparations of Penicillin coming into the market do away with Romansky's oil and beeswax which is the cause of some pain at the site of injection. Virulent organisms can be effectively handled and relatively resistant strains of bacteria come under the bacteriostatic power of Penicillin. I give below a list of dental diseases (*vide page 327*) prepared by Kolmer in which Penicillin is potentially effective.

Dosage and methods.—In the early stage when Penicillin was not easily available, for most acute infections 10,000 units every three hours intramuscularly was considered adequate and 20,000 units every three hours was deemed a high dose. With the greater experience and supply of Penicillin on a diminished price we were able to get a better knowledge of the blood levels obtainable for the raised dosages and the better appreciation of the therapeutic effect. The aim now is to get the acute infection under control in

one or two days. The usual dosage employed is about 30,000 units for every three hours for milder cases and 50,000 units every three hours for the first 24 hours or 100,000 units as the initial dose followed by 30,000 units at three hourly intervals. Other things being equal these elevated doses are in line with good therapy. Adequate doses must be given, failing which there is danger of a possibility of producing Penicillin resistant organisms in the system.

Disease	Route of Administration
Actinomyces	Intramuscular
Anthrax	Intramuscular
Burns of face	Topical; intramuscular
Cavernous sinus thrombosis	Intravenous; intramuscular
Compound fractures	Topical; intramuscular
Gonorrheal stomatitis	Topical; intramuscular; oral
Ludwig's angina	Intramuscular
Maxillofacial injuries	Intramuscular; topical
Osteomyelitis of jaws	Intramuscular; topical
Fusospirochetal gingivitis	Topical, intramuscular, oral
Syphilis	Intramuscular
Wounds of face	Intramuscular, topical
Alveolar abscess	Intramuscular, oral
Apical periodontal abscess (acute)	Intramuscular, oral
Cancerum oris	Intravenous; intramuscular
Canker sores (Secondary infections)	Topical (troches)
Dento-alveolar abscess	Intramuscular; oral
Dry sockets	Topical (dental cones)
Infectious stomatitis	Topical (troches)
Membranous stomatitis	Topical (troches) intramuscular; oral
Mikulicz's disease (achrocytosis)	Intramuscular
Pericoronitis	Submucous injections
Periodontal abscess	Intramuscular; oral
Pulpitis (acute, early)	Intramuscular; oral
Replantation of teeth	Sterilization (solution)
Ulcerative stomatitis	Topical (troches)
Root canal sterilization	Topical (paper points); cream

Besides the intramuscular injection, the most prevalent method of administration now is to give Penicillin orally. When oral Penicillin is administered it must be given in doses four times greater than the amount injected to produce blood levels identical with those obtained with parenteral therapy. Therefore, at least 100,000 units every three hours must be given to keep the blood levels comparable with those of 30,000 units injected intramuscularly every three hours. Whereas one daily injection of 300,000 units of Penicillin is recommended for most infections, Romansky advises that injections be given at 12 hours' intervals for overwhelming bacteremias or any particularly virulent infections. He had

cited a case in which one patient used as much as 180 consecutive doses of Penicillin in beeswax and oil without ill effect. In oral surgery and extractions of impacted teeth the usual dose should be 300,000 units 24 hours prior to the operation and the same dose for approximately three to four days following surgery. This procedure is particularly necessary in patients with rheumatic heart disease.

Oral diseases.—Penicillin in any of these forms will give a marked improvement in most oral diseases, Vincent's infections, herpetic, stomatitis, periocoronitis, canker sores, alveolar abscesses, and dry sockets. Finely powdered Penicillin used in conjunction with an atomizer gives out a Penicillin mist which is extensively used now with good results for sinus infection. Penicillin ointment or cream, 100,000 units in one ounce of Albucid soluble Dental cerate expelled into an infected antrum caused by perforation *via* the buccal cavity is known to resolve the infection rapidly and also seal the antral opening into the buccal cavity.

Vincent's infection.—The drug is very effective in acute stages. MacGregor and Long published a report on the use of Penicillin in the form of pastilles on 25 patients with an acute ulcerative gingivostomatitis of Vincent's type. Within 24 hours, symptoms in all the patients had disappeared; in forty-eight hours, the gingivae showed signs of healing and treatment was halted in six days. There were no relapses in 3½ months of observation. Similar results have been achieved with a dosage of 50,000 unit troches three times a day, allowing the troche to dissolve over a period of two hours. A Penicillin mouth wash is also effective or a topical application of 500 units of Penicillin per c.c. of sterile water with spray is also of good. Additional intramuscular injections increase the efficiency of the drug. Two Penicillin tablets containing 25,000 Oxford units buffered with aluminium hydroxide, calcium carbonate and magnesium oxide every waking hour for a minimum of 12 hours daily has also been known to reduce the acute stage. These 600,000 Oxford units are given in lieu of intramuscular injections. If 25,000 units of Calcium Penicillin is added to 10 grains of denture adhesive powder and is blown on the gingivae with the aid of an atomizer, the Jelly-like matrix prolongs the contact of the Penicillin with the infected area and brings down the inflammation. This treatment can be given once every four to six hours for a 24 hours period. For Vincent's infection, this drug injected intramuscularly and applied topically or given orally acts as an efficacious spirocheticide, but the decrease in Vincent organisms is more marked after intramuscular administration. Epithelial stimulation marked after intramuscular than normal. In inflammation and bleeding of the gums we have the same good results.

Treatment of pyorrhœa.—Penicillin Sodium Lozenges of 25,000 units each buffered with Magnesium Oxide Aluminium

Hydroxide and Calcium Carbonate to counteract gastric acidity given in a daily dosage of 350,000 units, has been used for the treatment of periodontal disease. There seems to have been some improvement with the disappearance of suppuration.

Oral surgery.—In spite of the marvellous result of the Penicillin therapy we must consider the drug an adjunct to, and not a substitute for, surgical care and treatment. In the management of suppurating surgical infections caused by susceptible bacteria, Penicillin therapy has had a strikingly beneficial effect on mortality and morbidity but the fundamental surgical principles of early accurate diagnosis, early treatment, rest, adequate external drainage, correction of altered physiology and vigilant supportive treatment remain as important as ever. The prophylactic use of Penicillin in oral surgery is of major importance. What promises to be one of the most valuable uses of this drug is its power of rendering the blood stream hostile to a bacterial shower. When a tooth has to be removed, it is a great comfort to know that an injection of 50,000 to 100,000 units of Penicillin in Procaine injected into the buttocks 10 minutes before the operation will control any spread of infection in so far as the infection is Penicillin-sensitive. Particularly is this indicated in cases of simple non-infective endocarditis. A boost dose of Penicillin in cases of bacterial endocarditis a couple of hours before the extraction of a tooth will prevent any spread of infection. Penicillin drips into bone-graft wounds of the jaw have been used for a period of post-operative time as a prophylactic measure, with excellent results. There are cases of osteomyelitis of the mandible treated with oral Penicillin and local injection that resolve without the need of sequestrectomy or removal of the teeth. Acute alveolar abscess, cellulitis of the jaw, periocoronar infections and Ludwig's angina are other conditions which are dramatically aided by Penicillin therapy. Treatment of Ludwig's angina with Penicillin has been a boon, but not a panacea. Where Penicillin does not give the expected result, Sulfadiazine should also be administered along with it. In acute periocoronar infection 25,000 units of Penicillin in a two percent solution of Procaine given once or twice daily at site into the inflamed tissue is enough to bring it back to normal. Combining Penicillin solution with one to two percent Procaine Hydrochloride eliminates pain on injection and does not interfere with the absorption of the drug. An ordinary 2 c.c. ampoule of 2% Procaine can be injected into the phial of Penicillin and drawn back and that can be used for giving either intramuscular injections or even into the site of the operation. It is clinically possible to use the combined Penicillin-anesthetic solutions without affecting the potency of either constituent element. Penicillin has been used for direct implantation into a socket after the removal of impacted third molars. 100,000 units of Crystalline Sodium Penicillin in cream implanted directly in that area reduces

under: (1) Swarasa, half a pala; Nisoshita and Agnisidha, each one pala; (2) Putapaaka, one pala; (3) Kalka, one karsha; (4) Kwaa-titha, two palas according to some and one pala according to others; Seetha kashaaya, Phaanta-kashaaya and Mantha kashaaya, each two palas; (5) Choorua, one karsha or quarter of a pala; (6) Yataka, quarter of a pala is the maximum (but greatly variable); (7) Confections or Avalehas, one pala; (8) Gritha or thaila, each one pala; and (9) Aasava or Arishta, each one pala. A Vaidya pala, it is said, weighs four rupee-weight; and it is accepted that one pound (avoirdupois) of today is equal to the weight of forty rupees. Therefore, one vaidya-palam is equal to one-tenth pound or about one and a half ounces in weight (or one and a half fluid ounces in volume, making allowance for the specific gravity etc.) Conversion ratios above as under: (1) Swarasa, three fourth fluid ounce; Nisoshita and Agnisidha, each one and a half ounces; (5) Choorua, three-eighth ounce or 180 grains; (9) Aasava and Arishta, each one and a half ounces.

Dosage and age:—The doses mentioned above are for ages between 16 and 70 years—i.e., the standard dose for average adult; for ages below 16 and above 70, the doses are correspondingly less. According to Sarangadhara, infants up to one month of age may be given one gunja of the drug at a time; and this should be given as linctus, with the drug macerated in breast-milk, honey or ghee and mixed with sugar. From the second month, the dose can be increased by one gunja for every month of age till the first year so that a child of one year gets 12 gunjas of medicine at a time. Thenceforth the dose is increased by one maasha for every year of age till the 16th year when the dose becomes 12 gunjas + 15 maashas of 16½ maashas which make nearly one karsha, i.e., the adult dose. This is continued till 70th year after which it falls in the same gradient as the rise from birth to 16th year. The doses mentioned above are for Kalkas and Choornas—in fact, for any preparation whose adult dose is one karsha. The age-variations in the corresponding adult dose as the basis. Thus, the dose of Gritha (whose adult dose is one pala or four karshas) is four gunjas up to one month of age and is increased by four gunjas for every month till first year and by four maashas for every year from first year to sixteenth year when it becomes the adult dose of one pala [12 + 4 gunjas or 6 maashas + 15 × 4 maashas = 66 maashas = 4½ karshas; and four karshas make one pala.]⁵

5. "Baalasya prathame maasi deya bhesaja-rakthikaa
Avalahee-kritna-eka-eva kshera-kshoudra-sitha-grithai:
Vardhaye-thaavadi-ekaikaam yaavath-bhavaatha-rathasara:
Maashav-vidhis-thad-oodhvaam syaad-yaavadi-shodasa-rathasara:
Thatha: sthithina bhaveth-thaavadi-yaavadi-varshani sapthathi:
Thatho baalakaram-maatrai kraasanwega sanais-sanaai:
Marthreyam kalka-choornanaam kashaayanam chatur-guham."
(Sarangadhara, Poorvakhanta, Chapter 6, verses 14-17).

B. Times of administration of medicines.—Savants of old are all agreed that drugs, especially kashaayaas (kwaththaas), are best administered in the early hours of the morning when the stomach will be empty and at a time when the activity of kalpa has passed its phase of ascendancy; this is more so when the patient and the disturbance are both powerful⁶ and 7. There are, however, conditions when exhibition of drugs will be called for at other periods of the day, and with or without relation to meals.⁸

The five periods in the day for giving drugs:—Physicians of old have prescribed five periods for administration of drugs—"the Pancha-vidha kala." And these are—(1) During early morning (a small time after sunrise) and in an empty stomach; (2) at the time of and in relation to morning meal; (3) at the time of and in relation to evening meal; (4) frequently and at repeated intervals; and (5) during night and in an empty stomach.⁹

1. *Early morning:*—This is the best time for giving medicine for purgation, emesis, and attenuations ('lekhana') and where pitha or kapha is vitiated. Stomach should be empty then.¹⁰

2. *In relation to morning meal:*—(a) Where the 'Apana Vaayu' (the vital gaseous energy controlling the vital processes in lower abdomen and pelvis) is dysfunctioned—in other words, where the intestines and pelvic organs are not functioning properly—, administration of medicine should be closely before the meal. (b) In anorexia, appropriate but agreeable drugs should be given in the course of the meal which should consist of spicy, tasty and varied dishes. (c) In cases of dysfunctioning of 'Samana Vaayu' (the vital gaseous energy that controls digestion and occupies the region of umbilicus or nearabouts jejunum, pancreas and celiac plexus) and in cases of dull digestion, in other words, in cases of alimentary asthenia (secretory or motor)—, select digestants and stomachics should be given along with the meal, and preferably placed within the boluses. (d) Where 'Vyana Vaayu' is excited, medicines are best given immediately after the morning meal [Vyana Vaayu is the gaseous energy that pervades the entire body and has a lot of functions to perform]. (e) In hiccup, and affections accompanied

6. "Bhaishajyam-abhi-avaharel prabhaathe praayaso budha:
Kashayagams-thu viseshena thathra bhedasithu darsitha."
(Sarangadhara, Poorvakhanta, Chapter 2, verse 1).

7. "Kaphodreke gathe-anannam balino roga-roginio."
8. "Yajnyaat-anannam-annaadaw madhye anthe kabala-anthare
Graha-grahase mahas-sa-annam samulgam nisi ch oushadham."

(Ashtanga Hridaya, Soothrasthhaana, Ch. 13, verses 36 and 37).

9. "Agni panch-vidhi: kals bhaishajya-grahane nriyam
Kinchal-Surg-ud-aye jathe thaththa darsa-bhojine
Sanyathane bhojane ch mahas-ch-epi thaththi nisi."

10. "Prayati pithi-kaphodreke virek-samam-arththago:
Lekk-mi-arththam-chi bhaishajya-prabhaathe an-annam-avaharel
Eva syad prathimam: kulo bhaishajya-grahane nriyam."
(Sarangadhara, Poorvakhanta, Chapter 2, verses 2, 3, 4).

by jumps and jerks (as in disseminated sclerosis or post-influenzal nerve affections), drugs should be exhibited before and after the meal.¹¹

3. *In relation to evening or night meal:* (a) In cases of excitement of 'Udam Vaaṅ' (the vital gaseous energy that controls life processes mainly round and nearabout the neck) with consequent laryngeal, pharyngeal and other throat troubles, medicines are to be given with each bolus or placed within each bolus of food (cf. 2 (c)). (b) In affections born of vitiation of 'Prana Vaaṅ' (the vital gaseous energy that controls the very breath and is engaged in everything connected with respiration), drugs should be administered immediately after supper.¹²

[Note. Vagbhatta is of a different view. He says that in affections of Udam Vaaṅ excitement, drugs should be administered immediately after supper; while in troubles of Prana Vaaṅ vitiation, medicines should be given with meal and placed within the bolus¹³.—Just the reverse of what have been mentioned above.]

4. *At repeated intervals:* In excessive thirst, incessant vomiting, continued hicough, severe dyspnoea and grave poisoning, drugs must be administered frequently and mixed with food¹⁴.

5. *During night:* Medicines intended for affections of head, as also medicines used for their fattening, attenuating, reducing or soothing effects are to be exhibited during late at night and when the stomach is empty.¹⁵

The above are the five periods of the day for administration of medicines. It is to be noted, however, that where no time is particularised, the drug should be given in the early hours of the morning—('Kaale vande prabhavatham,' Saravagadhara).

11. "Bhishajyama bhajam...
Aracham kshitha bhajam...
Somaam...
Daly...
V...
H...
E...

12. "Udam...
Grasse...
Prane...
Ushadham...

—Saravagadhara, Poorvachanta, Ch. 2, verses 5-8.

13. "V...
G...
(Ashdanti Hridaya, Sootrasthithama, Chapter 13, verses 38, 39).

14. "Mahur...
Sa...
Sa...

15. "O...
Pa...
I...

(Saravagadhara, Poorvachanta, Chapter 2, verses 10, 11, 12).

C. Medicines and Meals. It will have been observed that medicines are mostly prescribed to be taken before, during or after meals, (and, in certain cases, before, during and after meals); also, there are occasions when medicines are advised to be taken incorporated with or put within the boluses of food. The following from Chakradatta on the indications for and the effects of administration of medicines in relation to meals will be read with interest.

(1) Medicines given alone and in an empty stomach will have a greater effect and bring about a speedier result than when given otherwise. But, such should be exhibited with caution in the very young, the fair sex, the aged and the delicate, as medicines given alone to these are liable to cause weakness and bring about exhaustion.¹⁶

(2) Medicines given with meals will be assimilated rapidly, will not regurgitate, and will not affect the patient's strength adversely. This course is indicated in the very young, the fair sex, the aged and the timid, fastidious or delicate. The effect of giving medicine before food is the same as the effect of giving it with food.¹⁷

(3) It is important that no medicine should be given when there is undigested matter in the stomach, and also important that no food should be given till all the medicine has left the stomach. In either case, the medicine will not only not have the desired effect on the disease, but is also very apt to cause fresh disorders.¹⁸

D. Assimilation of Medicine.—It has been said that no food nor medicine should be given in disease when the stomach contains something un-digested. It is necessary, therefore, to know if a medicine administered has been or has not been assimilated.

Signs of restitution of disordered vital gases (Vaaṅs); feelings of comfort, hunger, thirst, cheerfulness and lightness; normal evacuations; and healthy eructations—these are the signs of the drug having been assimilated.¹⁹ On the other hand, a feeling of dulness, heavy sensation all over the body, fatigue, giddiness, fainting, headache, anorexia, restlessness and exhaustion—these

16. "Veerya...
H...
Th...
G...

17. "Seegham...
D...
P...
D...

18. "Oushadha...
Na...

(Chakradatta, Swaraadhikara, verses 53, 57, 58).

19. "Anuloma...
L...

indicate non-assimilation or mal-assimilation of the drug by the system.²⁰

[*Note*.—The raging practitioner who indulges in racing with incongruous drugs in experimental stage may, with advantage to all, well assimilate what have been said under 'Assimilation of Medicine.' The allergic, anaphylactic and other untoward reactions obtained during the exhibition of sera, vaccines and 'chemo-drugs' are more often than not due to their exhibition at a time when the cells are loaded with "undigested" matters—and "undigested" matters that are distinctly incompatible with the drug exhibited. It may be interesting to know the exact number in the very small fraction of medical men and women who trouble themselves about the Opsonic Index of the patient's blood during vaccine administration. It is no doubt very convenient to put all forward reactions to 'idiosyncrasy.' But, in many a case, the honest can find no honest answer to the sufferer's question 'whose?']

Chapter V.—(A) Groups of Therapeutic Agents

A complete therapeutical classification of drugs is now here possible. The physicians of yore have mentioned but the more important among such groups, and cited one or two examples under each.

A. Deepana—Paachana Oushadhas.—'Deepana' means 'promoting digestion'; and 'Paachana' means 'cooking'.

1. *Deepana-oushadha*:—A drug that has in itself no power to digest (cook) but kindles what does digest (cook) is a *Deepana-oushadha*. 'Misi' is an example. [Misi is either *Peucedanum Graveolens* or *Pimpinella Anisum*. The former, according to Dr. Nadkarni, is *Misariya* or *Satapushpi* in Sanskrit, Dill in English, *Shatakupii* in Telugu and Tamil, and *Chatakuppa* in Malayalam. The latter, according to him, is *Madhura-misi* or *Satapushpa* in Sanskrit, Anise or Sweet Fennel in English, *Sompu* in Telugu and *Sombu* in Tamil; no Malayalam equivalent is given—perhaps, it is the 'Aasaali' of Malabar physicians. It is worthy of note that Dr. Nadkarni has described *Foeniculum Vulgare* or *Anethum Foeniculam* as Indian Sweet Fennel in English, *Madhrika* or *Methica* in Sanskrit, *Sopu* in Telugu and *Sombu* in Tamil].

2. *Paachana-oushadha*:—A drug that merely digests but does not produce or promote what does digest is a *Paachana-oushadha*. *Naagakesara* is an example. [This drug is *Mesua Ferrea* botanically, *Cobra's saffron* in English, *Nagakesara* in Telugu, *Cheru-nagapu* in Tamil, *Nagachampakam* in Malayalam

20. "Klamo daaho-amgasadanam bhramo moorchchaa siro-rujaa
Arath(k)ir-balahaanis-cha sa-avasesha-oushadha-aakrithi."

(Chakradatta, *Jwarandhikara*, verses 54, 55).

and *Naga-sampige* in Canarese (Nadkarni). This drug is allied to and causes confusion with another drug known as *Punnaga* in Sanskrit, *Ochrocarpus Longifolius* botanically, *Alexandrian Laurel* in English, *Surapoonu* in Telugu, *Naggessur-pu* in Tamil, *Gardundi* in Canarese and *Punnaga-pu* in Malayalam.]

3. *Deepana-Paachana-oushadha*:—A drug that both digests and helps to produce what digests is *Deepana-Paachana-oushadha*. 'Chitraka' is an example. [This is *Plumbago Zeylanica* botanically, *Ceylon leadwort* in English, *Agnimantha* in Telugu and Sanskrit, *Chittira* in Tamil, *Bile Chitramula* in Canarese, and *Kotuveli* or *Vellakotuveli* in Malayalam.]¹

[*Notes*:—(a) A *Deepana-oushadha* may be compared to a stomachic like a bitter or a carminative which, increasing the vascularity of the stomach and augmenting secretion or motility, promotes digestion. (b) A *Paachana-oushadha* may be compared to pure digestives like *Papain*, *Pepsin*, *Diastase* etc., all of which are agents that actually contain digestive principles and have direct action on the digestion of foods. (c) A *Deepana-Paachana-oushadha* may well be said to be represented by *Vitamin-B* complex of today, especially the *Nicotinic acid* element in it.]

B. Samana-oushadhaas.—'Samana' means 'Allaying.'

A drug that does not affect normal metabolism nor removes the dys-metabolised products, but merely restores the normal functioning in cases of dys-functioning is a *Samana-oushadha*. 'Amritha' is an example. [This is *Cocculus Cordifolius* botanically, *heart-leaved moonseed* in English, *Gulantha* in Bengali, *Tippateego* in Telugu, *Amratavalli* in Tamil and Canarese, and *Chittamrith* in Malayalam.]²

[*Note*:—*Samana-oushadha* may be compared to present-day 'Alternative' of the type of arsenic, iodine, mercury etc., a class of remedies which give relief in diseased conditions without producing manifest effects on any organ but by some as-yet-unknown process of alteration of the morbid process to normality].

C. Sodhana-oushadhas.—('Sodhana' means 'Correction' or 'excrement'; it means the latter in the context). These are remedies possessing 'purging' effects on the body, and mainly consist of cathartics and emetics. The cathartics are of different types depending on the nature and intensity of their action on the alimentary contents and walls, and correspond to laxatives, simple purgatives, drastic purgatives and cholagogue purgatives of present-day classification of aperients.

1. "Pachen-na-aamam vahnikrich-cha deepanam thad-yaththaa-misi:
Pachathi-aamam na vahnim-cha kuryaad-yath-thad-hi paachanam
Naaga kesara-vad vidyaad chithro deepana-paachana."

2. "No sodhayathi yad-doshaan samaan-na-udeerayathi-api
Sameekarothi vishamaan samanam thad-yaththa-amrithaa."
(Sarangadhara, *Poorvakhanda*, Chapter 4, verses 1 and 2).

1. *Anulomana-oushadha* :—(‘Anuloma’ means ‘in natural direction’ or ‘in regular order’). An agent that acts on undigested matter, if any, in the alimentary canal, and, loosening the contents, pushes them downward ‘in natural direction and regular order’ is an *anulomana-oushadha*. ‘Hareetaki’ is an example. [This is *Terminalia chebula*³ botanically and chebulic myrobalan or ink nut in English].

[Note:—Hareetaki is a safe, mild, cholagogue aperient, and may be said to resemble Rhubarb in action but devoid of all griping or with but a minimum of it].

2. *Sramsana-oushadha* :—(‘Srams’ means ‘to cause to move’ or ‘to cause to slip down’). An agent that, without any digestive action on the contents in alimentary canal, sets up peristalsis and drives the softened and adherent faeces down to the anus is *Sramsana-oushadha*. ‘Kritha-maalakam’ is an example. [This is *Cassia Fistula* botanically, and Indian laburnum or Purging Cassia in English]⁴.

[Note:—Kritha-maalakam seems to resemble Figs, Prunes, Sulphur or Manna in action, and appears to be a simple laxative like the latter. Sir William Whitla, in his *Materia Medica*, says ‘Cassia Pulp’ is a mild laxative, like manna, and it probably acts by stimulating the peristaltic movements of the intestines’].

3. *Bhedana-oushadha* :—(‘Bhed’ means ‘disuniting’ or ‘evacuating the bowels’). An agent that breaks hard fecal matter that may exist en masse or in lumps in the intestinal canal and directs the softened contents to the anus is *Bhedana-oushadha*. ‘Katukee’ is an example. This is *Pierorrhiza Kurroa* botanically, and Katukarohini in Indian languages]⁵.

[Note:—*Pierorrhiza Kurroa* was once a B. P. drug, and in action resembles Cascara or Aloes.]

4. *Rechana-oushadha* :—(‘Rechana’ means ‘emptying’ or ‘evacuation’; ‘Virechana’ means ‘evacuation of bowels’ or ‘purgative’). An agent that liquefies digested and undigested matters in the intestines, and helps the bowels to evacuate them is *Rechana* or *Virechana* *oushadha*. ‘Thirivithaa’ is an example. [This is *Ipomoea Turpethum* or Indian Jalap, and is a drastic purgative]⁶.

3. “Krithavan paakam mala naam yad-bhithava bandham-adho navel
Thuch-cha-anulomanam jnegam yathitha prokthaa hareethaki.”

(Sarangadhara, Poorvakhanda, Chapter 4, verse 3)

4. “Pakthavvam yad-apakthava-eva shishtam koshte mala-andikam
Nepathi-adha: sramsavum thad-yathitha syant kritha maalaka.”

5. “Mala avlikam-abandham yal baltham van pinlitham malui:
Bhithava-adha: paathayathi thad-bhedanam katukee yathithaa.”

6. “Vipakvam yad-apakvam eva mala-adi dravatham navel
Rechayathi-api thad-jnegam rechavam thirivithaa yathithaa.”

(Sarangadhara, Poorvakhanda, Chapter 4, verses 4, 5 and 6).

5. *Vamana-oushadha* :—(‘Vama’ means ‘to eject by mouth’ or vomit). An agent that forces up and ejects *via* mouth pathological mucus and bile is *Vamana-oushadha*. The fruit of ‘Madana’ is an example. [Madana, according to Nadkarni, is *Randia Dumetorium* botanically, and Emetic Nut in English.]⁷

6. *Sam-sodhana-oushadha* :—(‘Sam-sodhana’ means ‘correcting or purifying’ thoroughly). An agent that ‘displants’ conglomerate accumulations of waste in the alimentary canal and throws them out *via* mouth and anus is *Deha-sam-sodhana-oushadha*—‘Deha,’ meaning the body. The fruit of ‘Deva-daali’ is an example. [Let those who can distinguish Deva-daali enlighten others!]⁸

7. *Chchedana-oushadha* :—(‘Chchedana’ means cutting, dividing, destroying or removing). An agent that energetically divides or ‘dissolves’ and completely removes or destroys firmly rooted pathological formations in the body is *Chchedana-oushadha*. ‘Kshaara,’ ‘Maricha’ and ‘Silajit’ are examples. [(1) Kshaaraas are corrosives, and correspond to caustics. (2) Maricha is Black Pepper; its ‘Chchedana’ action resembles that of Soda Bicarb on mucous in chronic gastritis or that of Iodides on gumma in syphilis. (3) Shilajit, according to Nadkarni, is Asphalt. In Malayalam (and possibly in other Indian languages too) it is known as ‘Kam-madam’, the real term being ‘Kal-madam’ meaning ‘born of frenzy or ‘heat’ of rock’; in fact, Silajit is the molten product erupted by rocks rich in minerals during very hot summer when the rock becomes extremely hot and its contents are molten. The ‘Chchedana’ qualities of Silajit are exhibited in its lithontriptic action on calculi (*cf.* Lithium), in its absorption of pathological fibrous tissue in cirrhoses (*cf.* Iodine and Fibrolysin), and in various other directions]⁹.

8. *Lekkana-oushadha* :—(‘Lekkan’ means scraping, making thin, attenuating). An agent that thins the tissues and wastes (Dhatus and Malas)—*i.e.*, an agent that brings about reduction or resolution in hyper-formations—is a *Lekkana-oushadha*. ‘Kshoudram’, (honey), ‘Neeram-ushnam’ (hot water); ‘Vachaa’ (*Acorus calamus*); ‘Yavaa’ (Indian Barley)—are all examples^{10 & 11}.

7. “Apakwa-pitha-sleshmanow baland-oordhavam mageththu yal
Vamnavum th-adhi-vjnegam madanasya ppakam yathithaa.”

8. “Sthithamud bahir-maged-oordhavam-adho eva mala-sanchayam
Deha-sam-sodhanam th-ad-syaal-deva-daalec-ppakam yathithaa.”

(Sarangadhara, Poorvakhanda, Chapter 4, verses 7 and 8).

9. “Shishtam kappu-andikam dosham-unmooleyathi yad-baland
Chched-nam thad-yathitha-kshaara marichani silajitu.”

10. “Dhaathom malaan eva dehasya visoshya ullekhasyescha yal
Lekkavam thad-yathitha kshoudram neeram-ushnam vachaa yavaa.”

(Sarangadhara, Poorvakhanda, Chapter 4, verses 9 and 10).

11. Note.—Bhaavamisra in his Bhaavaprakasha (verse 234, Misra Varga Prakarana) annotates yavaa in the context as Indra-yavaa (*Holarrhena Anti-dysentrica*)—“Yavaa-Indra yavaa:”. But, it should be noted that while the action of yava or Indian barley is to excrete, more urine and faeces (Barley is a diuretic and laxative) and hence yava is a lekkana-oushadha the action of *Indrayavaa* is to bind or confine, and, as we shall see, is a ‘*Sthimbana-oushadha*.’

[Note:—Lekkana-oushadha seems to have the virtues of Dessicants, Detergents, Discutients and Resolvents].

9. *Pramaaththi-oushadha*:—('Pramaaththi' means 'churn', set in motion, or destroy). An agent that acts on pathological formations and accumulations in circulation and expels them is a *Pramaaththi-oushadha*. 'Maricham' (Black pepper) and 'Vachaa' (*Acorus calamus*) are examples. Sufficient has been said on these under 'Chchedana' and 'Lekkana' oushadhas.¹²

D. *Bhandana oushadhas*.—('Bhandana' means 'binding' or 'confining'). These constitute the reverse of *Sodhana-oushadhas*.

1. *Graahi-oushadha*:—('Graahi' means 'holding'). A drug that is both a stomachic and digestant and by virtue of its desiccant properties absorbs fluids is a *Graahi-oushadha*. Examples are 'Sunttee' (*Zingiberus*), 'Jeerakam' (cumin seed), and 'Gajapippali' (which last, according to Nadkarni, is *Scindapsus Officinalis* botanically, *Enuga pippalu* in Telugu, *Anaitippali* in Tamil and *Aththi thippali* in Malayalam).

[Note:—Acid Sulph. aromatic and Acid Nitro-Hydrochlor Dil may be said to correspond to *Graahi-oushadhas*].¹³

2. *Sthambhana-oushadha*:—('Sthambhana' means 'making stiff', 'arresting' 'binding' etc.) A drug that is sharp (or harsh), stiff (or cold), and astringent, but light and agreeable on digestion, and that, inducing reverse currents, impedes the movements of the contents and confines them is a *Sthambhana-oushadha*. Examples are 'Vatsaka' (*Holarrhena anti-dysentrica* or *kurchi*) and 'Tundika' (*Oroxylum Indicum* or *Colosantes Indica* botanically, according to Nadkarni; this is one of the drugs in the famous combination of *Dasamoola*).¹⁴

[Note:—Astringents of the type of catechu, kino and Ipeca-cuanha (?) may be put to resemble *Sthambhana-oushadha* in action].

3. *Abhishyandi-oushadha (or Dravya)*:—('Abishyanda' means 'great increase, excess' in the context). A substance that, due to its sliminess and cumbrousness, causes occlusion of vessels carrying

12. "Nija-veeryena yad-dravyam srothobhyo dosha-sanjayam
Nirasjathi pramaaththi syaal thad-yaththa maricham vachaa"

(Sarangadhara, Poorvakhanda, Chapter 4, verse 23)

13. "Deepanam paachanam yuth-syaad-nishnathuvaa drava-soshakam
Graahi thal cha yaththa sunttee jeerakam gaja-pippalee"

14. "Roukshyaal saithyaal kashaayathuvaa laghu-paakaal cha yad-bhavel
Vathakril sthambanam thasyaad-yaththa vatsaka tundukow"

(Sarangadhara, Poorvakhanda, Chapter 4, verses 11 and 12)

Note: "Vathakril" in verse under footnote-reference 14 literally means "inducing peristalsis" with reference to intestines. Such a drug cannot confine intestinal contents, and in his *Bhavaaprakasa* that "Vathakril" in the context means 'Prathiloma vathakril' i.e., means 'in natural direction', or 'in regular order'; and 'Prathiloma' is the antonym of 'Anuloma'.

'rasa' (chyle), thereby producing stagnation and heaviness is *Abhishyandi*. Curds is an example.¹⁵

E. *Vaajeekarana oushadha*.—('Vaajeekarana' literally means 'Performance of a fiery steed', and means in the context 'Excitement of amorous desire by aphrodisiacs').

Vaajeekarana oushadhas are of two types: (1) Those that in any wise improve the quantity of 'Sukla' (vital fluid); and, (2) those that stimulate its emission. The former are called 'Sukla-janaka' *Oushadhas* or *Suklalas* while the latter are termed 'Sukla-pravarthaka' or 'Sukla-rechaka' oushadhas.

Aswagandha, *Musali*, *Sarkara* and *Sataavari*; *Dugdha*, *Maasha*, *Bhallaatha-ppala-majja* and *Amla*;—all these, by virtue of their being tissue-builders increase the quantity of *sukla* and thereby augment sex-impulses. [Aswagandha is *Withania coagulens* (vegetable rennet) or *Withania Somniferans* (Wintercherry)—according to Nadkarni; *Musali* is *Cureuligo Orchoides* botanically, Black *Musali* in English, *Naelatadi-chettu* or *gadda* in Telugu, *Neladali* in English, *Naelatadi-chettu* in Tamil and Malayalam; *Sarkara* (Canarese, and *Nellapana-kilangu* in Tamil and Malayalam; *Sarkara* is crystalline White Sugar, *Sataavari* is *Asparagus Racemosus* botanically; *Dugdham* is milk; *Maasha* is black gram; *Bhallaata-ppala-majja* is the seed or nut of *Bhallaata* fruit (*Bhallaata* is *Semecarpus Anacardium* botanically, *Marking-nut* in English, *Jeedivittulu* in Telugu, *Shenkottai* in Tamil, *Chermara* in Malayalam and *Gerkayi* in Canarese); *Amla* is *Emblia Officinalis* botanically, *Indian Gooseberry* in English, and *Nellikai* in South Indian languages].

Naagabala and *Kapikachhu-beeja* excite the psychological centre of sex-emotion while *Brahathi-ppala* stimulate the physiological centre of sex-impulse and ejaculation. 'Woman', however, combines both the influences, constituting the most potent aphrodisiac.¹⁶ [Naagabala is *Sida Spinoso* botanically; *Kapikachhu-beeja* is the seed of *Kapikachhu*, which is *Mucuna Pruriens* botanically, the cowhage or cowitch plant in English, *Pilliadagu* in Telugu, *Poonakkali* in Tamil, *Naikuruma* or *Chorivalli* in Malayalam and *Nayisonagu-balli* or *Nasuganni* in Canarese; *Brahathippala* is the fruit of *Brahathi*, and *Brahathi* is *Solanum Indicum* botanically, *Indian Night Shade* in English, *Tellamoolaka* in Telugu, *Karimull* in Tamil, *Kiriguligida* in Canarese and *Cheruchunda* or *Cheruvazhuni* in Malayalam—this is one of the ingredients in the famous

15. "Paichchillyaal gowravaal dravyam rudhavan rasa-vahaa: siraa:
Dathhe yad-gowravam thal-syaad-abhishyandi yaththa dalki."

(Sarangadhara, Poorvakhanda, Chapter 4, verse 24).

16. "Sthree, smarava-keerthava-darsava-sambhaashana-sparsava-
Chumbava-adlingava-nidhuvana: samasthai: vyasthai: cha
Suklasya pravarthinee - pravarthithi-karinee."

(Bhaavaprakash, Poorvakhanda, Misravarga-prakarana, verse 238).

Dasamoola. (The appellation 'Brahathi' applies also to Solanum Jacquini known as Wild Eggs plant in English, Kantakari in Canarese, Kandaikattari in Tamil and Vellottuvallutina in Malayalam.)

Jaati-ppala (nutmeg) is a 'Sukla-sthambana' delaying ejaculation and emission during coition. It is therefore useful in cases of premature ejaculation.

Hareetaki (chebulic myrobalan or ink nut) is said to dry up (inspissate?) sukla. It is difficult to annotate 'Soshance' in the context as 'exhauster.'

[Note.—It is a common belief that Hareetaki (Kadukkai) suppresses or depresses sex-impulses. Pseudo-sanyasins consumed a lot of it, especially, during their preparatory period. And the 'wolves' fleece the 'sheep' through unholy 'Atanga Nigraha' preparations which contain but Hareetaki with a sprinkling of nutmeg, opium or some like substance. Let it be clearly understood that Hareetaki is a 'Kalpam' and a 'Rasayan'. Kalpams and Rasayans, being vitalisers, cannot injure sex nor exhaust sukla; perhaps, Hareetaki, long continued, sublimates the inspissated sukla and elevates the baser sex impulse.]¹⁷

F. Rasaayana-oushadha.—('Rasaayana' means and 'Elixir Vitae'). A medicine that keeps one in bloom and cuts short (chronic?) ailments is a Rasaayana-oushadha. Amritha (Long pepper in the context), Guggulu (Indian Bdellium in English and Babsamoden-dron Mukal botanically), Hareethaki (chebulic myrobalan or ink nut), and 'Rudandhi' (?) are examples.¹⁸

(B) Miscellaneous Groups of Drugs

1. Yoga-vahi:—A substance, when compounded with other substances, imbibes the medicinal virtues of the latter is Yoga-vaahi-dravyam. Madhu (honey), Jalam (water), Thaila (oil), Sootha (mercury) and Loha (iron) are examples.¹⁹

17. "Yasmandrayad-bhavel sthreeshu haratho caajeeekaram-cha-thal
Yaththa nambakayana: syur-bheejam-cha kapikachichujam
Yasmand suklaya eridhi: syual suklalam-cha thad-uchyathe
Yaththa-aswagandham mus-dee sarkara-cha sathavaree
Dugdham-maashana-cha bhallatha-ppala-majja-amalaani-cha
Pravarthikani kuththimthe jarakanani-cha rethasa:
Pravarthnam sthree suklasya recham-bhriththe-ppalam
Janthe-ppalam sthambanam-cha soshance-cha hareethakee"

18. "Rasayam-cha-thal jeejam-yaj-jara-eyadhi-maashanam
Yaththa-amritha, rudanthee-cha gugguluscha hareethakee"

(Sarangadhara, Poorvakhanda, Chapter 4, verses 14-17).

19. "Grihyanthi yoga-sadhi dravyam sansargi-nistha-gucream
Pachy-maashanam-yath-etham-maashu-jala-theil-ujja-sootha-loha-sadi"

(Bhavaprakash, Poorvakhanda, Misravarga-prakaranam, verse

2. Sookshma-dravya:—A substance that passes through minute pores in the body and traverses the entire body is a Sookshma-dravya. Saindhava (Rock-salt), Kshoudra (Honey), Nimbathaila (Neem oil) and 'Ruvoodbhava' (castor oil?) are examples.²⁰

3. Vyavaayi:—A substance that, immediately on its intake circulates as such in the body exerting its action on the tissues, and then gets split up manifesting its secondary actions is a Vyavaayi. Bhanga (Ganja or Cannabis Indica) and Phena (opium) are examples.²¹

[Notes:—(a) It would appear that, according to Ayurvedic savants of yore, the vast majority of drugs exhibit their actions only after their having been acted on in the alimentary canal and their active principles liberated. Drugs of Vyavaayi group behave differently. (b) It is noteworthy that opium and Cannabis Indica like many others acting on the brain (especially cerebrum) have a two-fold action; they first stimulate causing excitement and exhilaration, and then depress producing dulness and stupor.]

4. Vikaasi or Vikaashi:—A substance that devitalises the tissues, and unnerves and unfastens the 'joints' making them loose and weak is a Vikaasi. 'Kramuka' (Arecanut or betel-nut) and 'Kodrava:' (Varaku in Tamil) are examples.²²

5. Manda-kaari:—Also known as Mada-kaari. A substance (continued use of which) dulls the intellect, and excites the coarse and base in man is a Ma(n)da-kaari. 'Sura' and other spiritous liquors are examples.²³

6. Visha:—A substance that combines the qualities of a Vyavaayi, a Vikaasi, and a Ma(n)da-kaari; mixes easily with other substances; and is fiery, penetrative and destructive to life is Visha.²⁴

20. "Dehasya sookshma-chidreshu viseththal sookshmam-uchyathe
Thad-yaththa-saindhavam kshoudram nimbathailam ruvoodbhavam"

21. "Poornam vyavayi-akkilam kaayam thetha: paakam-cha guchchathi
Vyavayi thad-yaththa bhanga phenam-cha-ahi samudbhavam"
(Sarangadhara, Poorvakhanda, chapter 4, verses 18, 19).

22. "Smathi-bhutha-cha-suthi-bhutha-karothi vikaashi thal
Vishlesha-oja-cha dhaathubhyo yaththa kramuka kodrava:"

23. "Budhim lunpathi yadrayam manda-kaari thad uchya
Thamo-guna-pradhnam-cha yaththa madyam surandikam"

24. "Vyavayi-cha vikaashiyam sookshmam chchedi mada-avatham
Angueyam jeevitha-haram yoga-vahi smritam visham"
(Sarangadhara, Poorvakhanda, Chapter 4, verses 20, 21, 22).

(To be continued).

Relapsing Typhoid Fever*

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A succession of cases of relapsing enteric fever, observed lately by the writer, appears to be worthy of record. While in the leading text-books of medicine the relapse rate of typhoid is stated to be varying between 4% and 16% in different epidemics, the series of enteric fever cases described in this paper shows a much higher relapse rate.

During 1947-48, 23 cases of enteric fever were admitted at the Barsi Light Railway Company Hospital at Kurduwadi, Sholapur District. Among these cases there were not less than 9 genuine relapses, characterised by the re-appearance of the typical clinical picture after a period of comparative well-being, with normal or practically normal temperatures of several days' duration. There were further, among the same series, 3 cases, showing a repetition of all or some characteristics of the initial attack after a typical fall of the fever, but before the temperature had actually reached normal.

The course of the whole epidemic—if the term epidemic is admissible to the spasmodic occurrence of typhoid cases all the year round—was moderately severe with two fatal cases among the total of 23.

Age incidence.—All relapsing patients were young people, the eldest one being 27 years old. The others were below 20, 5 of them being in their teens, while the remaining 3 were under 10 years of age. The youngest one was 3½ years old.

Sex.—Six patients were males, 3 females.

Race and community.—Two patients were Europeans, seven were Hindus, belonging to different communities of the Bombay Deccan.

Character and course of disease.—The clinical picture in all nine cases was rather severe and gave rise to anxiety in at least three cases during the whole duration of the disease. The diagnosis, in all cases, was made in the first instance from the clinical picture, and was confirmed by a positive and rising Widal.

The interval between the first proven rise of temperature and the occurrence of the feverish stage varied from 16 to 49 days.

The Table in the next page gives particulars as to the fever period, the afebrile stage and the relapse in all 9 cases. Case No. 9, whose temperature chart is shown below, relapsed not less than three times.

* Specially contributed to THE ANTISEPTIC.

present, and during this time the patient should be carefully observed for symptoms of alkalosis, such as lassitude, dizziness, headache, vomiting, and muscular cramps or tetany. The intake and output must be carefully observed, and it is thought that an intake of 2,000 to 2,500 cc. each twenty-four hours is necessary to wash the hemoglobin out of the kidneys. If, however, the patient becomes oliguric and edema appears, the intake should be reduced to prevent further formation of edema. When fluid is not tolerated orally, it can be given intravenously. Since damaged kidneys handle salt poorly, it should be withheld and 5 to 10 per cent dextrose in distilled water administered.

The retransfusion of compatible blood is important to counteract the severe hemolytic anemia and shock associated with these reactions. It is believed by some that the renal vasospasm already mentioned is also counteracted by the immediate transfusion of compatible blood. As plasma possesses a high salt content which is poorly handled by damaged kidneys, it is advisable to remove the plasma and resuspend the red blood cells in normal saline solution.

It is also advisable to administer albumin for the restoration of the albumin-globulin ratio rather than to use commercial plasma which has a high salt content.

General supportive measures should not be neglected. In order to maintain the normal concentration of the constituents of the blood from day to day, it is absolutely essential that careful, detailed studies be made daily. Recovery from anuria of from 10 to 12 days' duration and an elevation of the non-protein nitrogen to 200 mg. per 100 cc. of blood has been reported. Since the glomeruli suffer only secondarily, renal function may be restored once the tubules become patent.

Hypertonic dextrose is thought by some to be helpful in promoting diuresis. Administration of penicillin is advisable to forestall pulmonary infection which is so prone to develop in these patients who are on the borderline of pulmonary edema or in a semiconscious state. The terminal hypertension associated with uremia can be moderated by the intravenous use of magnesium sulfate.—*Digest of Treatment*, Feb. 1949.

OBSTETRICS and GYNÆCOLOGY

Sedimentation rate in pregnancy.

—(*Jour. Amer. Med. Assoc.*, 4-12-1948, p. 1070).

There is a progressive increase in the sedimentation rate during normal pregnancy. But there is no alteration in this rate associated with the toxæmias of pregnancy. During the puerperium the rate returns to normal in the majority of cases. The change over begins after the second day *post-partum*.

Duration of Pregnancy	Av. Sedimentation in mm.
5 months	.. 14.0
6 "	.. 19.0
7 "	.. 22.0
8 "	.. 22.5
Full term	.. 24.0
Second day <i>post-partum</i>	.. 27.5
Seventh day "	.. 25.5
Six weeks "	.. 12.0

There is a pronounced rise in the basic metabolic rate during pregnancy; sometimes reaching 35 per cent according to Stauder and Peckham.—(*Bull. John's Hopkins Hosp.*, 38.227).

Angina in women.—(*Br. Heart. Jour.*, 10, Jan., 1948).

Dr. Summers has given his findings in a series of 87 cases of angina pectoris in women. This is really a valuable study in view of the comparative infrequency of this disease in women, as compared with men. The age distribution in Dr. Summers' patients showed that 54 of the 87 were of the ages 40 to 60, only 3 being under 40, and 12 over 70. No relationship was detectable between parity or menopause and the incidence of angina. Hypertension (over 150 mm. Hg. and 90 mm. Hg.) was found in 58 patients. Definite history

of myocardial infarction was had in 34 and a less definite one in 14 patients. X-ray evidence of coronary disease was obtained in 57 cases. 83 per cent of which was in patients of 60 to 70. 2 patients under 40 were completely relieved of their anginal symptoms when treated successfully for anaemia. This study confirms the view that the underlying pathology is the same in both sexes, *viz.*, coronary artery disease.

T.N.S.R.

The management of obstetric emergencies.—F. L. Schade, M.D., from *Minnesota Medicine*, Sept., 1947, and abstracted in the June, 1948 issue of *The General Practitioner*.

Nowhere in the entire field of medicine can dire emergencies develop as suddenly and acutely as in obstetrics. When we, as practising physicians, accept the responsibility of taking care of a woman through her pre-natal care and labour, it behoves us to be adequately prepared for any emergency that may arise. The phrase, "Too little and too late," applies all too often to tragedies occurring in the delivery room.

Placenta praevia:—This condition occurs in at least one of 300 obstetric patients. When the placenta is implanted in the region of the internal os, partial detachment with haemorrhage results from the tissue re-alignment incident to formation of the lower uterine segment. The initial haemorrhage appears at or before the onset of labour, and every patient with placenta praevia must be considered as a candidate for fatal haemorrhage: thus—

1. A patient with suspected placenta praevia should be immediately hospitalized.
2. Her blood should be typed and cross matched.
3. A donor should be selected and ready for immediate blood transfusion.

Cystography in the diagnosis of placenta praevia was introduced in 1934 by Ude, Wenn, and Urner, who outlined the soft tissue space between foetal presenting part and bladder by filling the latter with an opaque medium. The technique used is as follows:

1. Empty bladder with a catheter.

2. Introduce 40 c.c. of sodium iodide solution into bladder, clamp catheter.
3. Anteroposterior film with tube centred on bladder and table tilted, feet down ten degrees from horizontal.

4. Unclamp catheter, allow bladder to drain. Remove catheter.

The interpretation of the roentgenogram in vertex presentation may be difficult, and in breech and transverse presentations it is virtually impossible.

Dipple and Brown in 1940, were able to visualize the placenta with a single roentgenogram in about 90 per cent. of 200 pregnant women by using a soft tissue technique. Interpretation and X-ray technique require experience for accuracy. The X-ray method represents a distinct advance in diagnosis, but for accuracy cannot displace sterile vaginal examinations. There is only one absolute diagnosis of placenta praevia, namely, digital palpation of the placenta through the cervical os. When the gloved finger introduced through the os cannot feel placenta, the patient does not have placenta praevia.

A note of warning! Digital examination must never be performed until the delivery room is completely set up for emergency delivery, since palpation of the placenta occasionally produces more bleeding.

Delivery is mandatory after repeated bleeding. The method of delivery may be vaginal or abdominal and the choice of procedure depends largely upon the probability of foetal salvage.

If the foetus is viable, a caesarean section should be performed on all cases of complete placenta praevia. If the placenta praevia is marginal or incomplete or if the foetus is not viable, one may resort to vaginal delivery. Acceptable vaginal methods include:

1. Spontaneous labour with or without rupture of membranes. Spontaneous labour may be brought about by medical means and artificial rupture of membranes.
2. Scalp traction. Personally I have never resorted to this means of delivery.
3. Insertion of a hydrostatic bag.
4. Braxton-Hicks version. This, of course, should never be attempted before full dilation of the cervix.

Except for the spontaneous labour with or without antecedent rupture of membranes, all these methods increase the foetal hazard.

Premature separation of the normally implanted placenta (Ablatio or abruptio placenta).—These terms are reserved for the separation of all or part of the normally implanted placenta occurring after the twenty-eighth week of gestation and prior to the onset of the third stage of labour. The incidence of premature separation is variously reported between 1:100 and 1:500.

Symptoms.—A moderate amount of vaginal bleeding usually associated with pain indicates partial placenta separation. If the placenta is implanted on the anterior wall of the uterus, there is frequently a circumscribed area of uterine tenderness.

Vaginal examination, again under sterile precautions, revealing no evidence of placenta praevia, confirms the diagnosis by exclusion.

Treatment.—As with placenta praevia, the value of blood transfusion cannot be over-emphasized. Even with mild vaginal bleeding, the physician must make preparation even though subsequent events render transfusion unnecessary. In every case, management depends upon the rate of bleeding and the condition of the cervix. Minimal bleeding requires little or no treatment. When bleeding is at a rate demanding delivery for hemostasis, whether we use the abdominal or vaginal route depends upon the condition of the cervix. With an uneffaced, firm and tightly closed cervix, caesarean section is mandatory.

Cervical effacement and partial dilation generally motivate vaginal delivery. Artificial rupture of the membranes institutes labour and the bleeding is controlled by the contracting uterus. If necessary, small doses of pitocin may be administered with relative safety. Never give more than one minim of pitocin for the initial dose! If these measures do not control the bleeding, insertion of a Voorhees bag to produce compression on a possible low-lying placenta, and the occasional employment of an abdominal binder, to raise intrauterine pressure to parity with the blood pressure, may be beneficial.

Uteroplacental apoplexy or the cour-relaire uterus.—This rare accident is too dramatic to omit, although the average practitioner will probably never encounter it. Retroplacental haemorrhage is sudden, extensive and forceful, and produces acute symptoms of profound shock and board-like uterus. The typical board-like uterus is not simulated by any other condition. Immediate caesarean section is the treatment of choice; despite known presence of a dead baby. Why caesarean without foetal salvage? In this condition blood infiltrates directly into the uterine muscle, dissociates the muscle fibres and inhibits uterine contractility. If the uterus will not respond to oxytocic drugs, a hysterectomy must be done.

Postpartum haemorrhage.—One just cannot discuss obstetric emergencies and omit postpartum haemorrhage. As we are bringing the toxemias of pregnancy and the puerperal infections under control, haemorrhage is assuming a relatively more important role in maternal mortality.

It is unfortunate that accumulated experience is not as impressive as personal experience and that some women must die to make some of us "haemorrhage conscious."

The emergency tends to appear at a time the attendants are relaxing. The baby is born, the labour is all but concluded, when suddenly a desperate fight to save the patient's life ensues.

Preparedness and the prompt institution of treatment can all but eliminate one of the most frequent causes of maternal mortality. Deaths from haemorrhage are largely preventable.

As a prophylactic measure, especially in hospitals without blood banks, the routine practice of typing all obstetric patients pre-natally is recommended. Also, the Rh status of each obstetrical patient should be established and special precautions taken should the patient be Rh negative.

Prolapsed cord.—Prolapse of the cord in vertex presentation is usually an indication for immediate delivery, provided the child is alive and the cervix is dilated enough. This implies six to seven centimetres dilation in multi-gravid and eight to nine in primigravid.

women. With dilations less than these, hasty extraction of the child usually leads to severe cervical lacerations and seldom results in a living child. Delivery by forceps is justifiable only if the head lies at mid-plane or below. With higher stations, the procedure of choice may be podalic version and extraction.

If the cord has prolapsed and the proper dilation has not been obtained, the patient should be placed in a deep Trendelenburg's position and closely watched until the cervix is dilated sufficiently to permit the necessary manoeuvres. Replacement of the cord through a partially dilated cervix is almost universally unsuccessful and I believe should not even be attempted. Prophylactic doses of penicillin or one of the sulpha drugs should be given to each patient with cord prolapse to prevent ascending infection.

Prolapse of the cord occurs once in approximately 150 deliveries and its occurrence depends upon three factors: the presence of an excessively long cord; inadequate filling of the pelvic basin by the presenting part; and ruptured amniotic sac.

Tetanic contraction of the uterus:—Tetanic contraction of the uterus is a possible sequel to administration of pituitary extract. Doses as small as one minim may produce tetanic contractions enduring as long as five minutes. Other factors, including long or obstructed labours, are operative in causing this condition unless the uterus can be relaxed. Two dangers become imminent, rupture of the uterus and asphyxia of the child. The time-honoured remedy is ether anaesthesia. Magnesium ions have a relaxing effect and the intramuscular administration of 2 c.c. of a 50 per cent aqueous solution of magnesium sulphate may be of benefit.

Baud's ring or contraction ring:—The upper active contractile segment of the uterus and the lower passive segment are separated by a physiologic ring of muscle which normally is of little significance. In mechanically obstructed labours, the upper segment progressively becomes thicker and the lower thinner. This produces a gradual elevation of the physiologic ring above the symphysis.

In rare instances pathologic constriction of this physiologic ring about the child occurs. In the cases a specific stricture of uterine muscle is produced. Diagnosis is made by internal palpation. The ring will probably not disappear but may be sufficiently relaxed to permit delivery following the intravenous injection of five minims of 1:1,000 epinephrine solution. Deep ether anaesthesia also has a relaxing effect.

Uterine rupture: Rupture of the uterus occurs once in 2,000 to 3,000 cases and may be incomplete or complete. Despite its infrequency, discussion of the accident is included because of its dramatic nature and potential dangers.

The foetal mortality approaches 100 per cent and the maternal risk is considerable.

The principal aetiological factors include: previous caesarean section; previous cervical operation or repair; obstructed labour with over-distended uterine segment and operative procedures, especially internal version.

The diagnosis of uterine rupture is based upon sudden, acute and lancinating pain, with abrupt cessation of uterine pain and cessation of labour, and the rapid development of hemorrhage and profound shock.

The rupture of a previously intact uterus may cause a dire obstetric emergency from shock and hemorrhage, and death within the hour unless heroic measures are applied.

Two methods of treatment are current; conservative and radical. Conservative treatment consists of either uterine and vaginal tamponade, or laparotomy with suture of the rent. Hysterectomy represents the radical method of treatment. The latter is much preferred and is the generally accepted treatment. Its advantages are numerous and offers the best chance of recovery for the mother. Nowhere in the entire field of medicine is there such vital need for rapid mobilization of facilities and early administration of plasma and blood as in the emergency of acute uterine rupture with severe hemorrhage. Sometimes rapid hysterectomy is demanded despite falling blood pressure.

Plasma and blood must be given while the operation is being performed.

Inversion of the uterus: Inversion of the uterus is a rare obstetric complication, but mention is made here because it appears with sufficient frequency to make it necessary for anyone doing obstetric practice to familiarize himself with therapeutic procedures for this condition. Reported incidence in various parts of this country has ranged between 1:740 to 1:16,000 deliveries.

Attempts to expel the placenta before its separation either by pushing from above or by pulling on the cord may be a predisposing cause, although the fundamental aetiological factor is unknown.

The symptoms are shock and hemorrhage associated with absence of the uterine body from the abdominal cavity and its presence in the vagina as a rounded tumour, a mass whose upper end is constricted by the encircling cervix.

Active and long-continued shock therapy is all-important! The immediate replacement of the inverted uterus under deep anaesthesia has given the unfavourable mortality rate of 10 to 43 per cent. A few writers have pointed out that this is a dangerous and unnecessary procedure.

Recent treatment has been to deal

with the inversion *per se* at any suitable time after the patient is restored to normal health, preferably four to twelve weeks following the acute episode. The inverted uterus may be left entirely alone unless the extent of the bleeding from it is such as to require the application of a tight vaginal pack. Treatment of the shock is the all-important thing. If it is not present, it should be anticipated. Multiple blood transfusions may be required not only to combat the shock but also to hasten the restoration of normal hemoglobin levels. Systemic chemotherapy should be instituted at once as a prophylactic agent. Local infection should be further combated with daily application of local antiseptic vaginal packing. After four to six weeks in the hospital, the well-involved uterus may then be restored surgically to its normal position or removed.

In conclusion, I wish to place emphasis on being prepared for the emergencies that may be expected in an obstetrical practice. We, as general practitioners, conduct the vast majority of childbirths. Emergency is a sudden condition—calling for immediate action, and time is an all-important factor. By its very nature, we cannot place the care of emergencies in the laps of specialists.—*The General Practitioner.*

Book Reviews

A Text Book of Bacteriology—By N. G. PANDALAI, M.D., D.T.M., F.R.C.P. (Edin.), Professor of Bacteriology and Principal, Andhra Medical College, Vizagapatam, 1948, 744 pages. The Bangalore Printing and Publishing Company, Ltd., Mysore Road, Bangalore City. (Price: Rs. 18)

This is one of the very few books on the subject written by an Indian. The existing text books written by foreigners are either too exhaustive or are written to foreign students with special reference to foreign conditions. Dr. Pandalai's effort is therefore none too early. Drawing from his vast experience as a teacher of the subject in one of our biggest Universities, the author has produced a book which is eminently useful to our undergraduates. It is written on the usual orthodox lines. The first Fifteen Chapters are devoted to general aspects of bacteriology such as the general characteristics of bacteria and their physiology, the preparation of culture media, infection, immunity, hypersensitive-

ness and bacterial variations. The next Fifteen Chapters are devoted to a detailed description of the various bacteria, their habitat, growth requirements, cultural characters, biochemical activities, variation, serological classification, typing and pathogenesis. There are chapters on filterable viruses and on bacteriophage. Each section of the book is complete in itself and special prominence is given to the application of bacteriology to the clinical and preventive aspects of medicine. We congratulate the author on his book and echo his hope that this work will help in stimulating the production of Indian publications. The printing and general get-up are good. We recommend the book to all medical undergraduates and post-graduates.

O'Meara's Medical Guide for India and the Tropics—Completely revised and greatly enlarged—By H. W. WILLIAMSON, O.B.E., M.D., M.R.C.P., F.R.C.S. (Ed.) Lt. Col., I.M.S., Late Director of Medical Services, Jaipur

State, India, 1947, 108 pages. (Butterworths & Co. Publishers Ltd., London.)

This little book is one of the popular medical guides to India and the Empire. The various diseases and ailments have been treated in an alphabetical order—A for Abscess, B for Yellow Fever. The form of information given and the manner in which it is given are special features. Among the chapters, those on Gynaecology, Fracture, Anaesthesia, Infectious Diseases, Heart Disease and Venereal Diseases are particularly good. Among the other chapters are those on the Eye, the Ear, Sinus, Lung, and Muscles, and on the Deformities, Nerve, Lung, Brain, Throat, and Women, are especially well done. The Chapter on Gynaecology written by Lt. Col. V. B. Green Armytage and revised and added to by the author contains very useful information. The Chapter on Civil Health Administration in British India written by Dr. K. C. K. E. Raja is well written. The Pharmacopoeia which appeared in previous editions has been omitted. The book contains a mine of information and will be very useful to every medical man. The printing and general get-up of the book are good.

The Beginnings of Modern Medicine in Madras—By Dr. D. V. S. Reddy, 1947, 244 pages, Franker Spink & Co. (1933), Ltd., Calcutta. [Price: Rs. 5]

Dr. Reddy is a pioneer in India as a medical historian. He has spent many years in the study of the History of Medicine on a scientific basis. His well-known internationally through his publications, the "Medicine in Mournful Times", "Medical Love in Ancient Indian Literature", "Medicine in Medieval India", "Medicine at the Madras Courts", etc. He now presents this fascinating and authentic account of the beginnings of modern medicine in Madras ever since the first English ship touched the Indian coast land (1600 A.D.)

It is pleasing to read that as early as the 15th century India possessed men of knowledge, professional men of high ethics, and an extensive system of medical relief. Contemporary writers mention that at this time Hindu and Mohammedan physicians practising in India were far superior in their specialities to those in other lands at the time.

Before the advent of the East India Company the Portuguese had already introduced European medicine and culture in India; it is said that hospitals and schools were established at Goa and Santhome

(Madras). Books written by the Portuguese Physician Garcia De Orta and French Physician Bernier give an idea of 17th Century Indian Medicine and Medical Practice in India about the 18th century. The author describes with illustrations and extracts available from processions of the East India Company through various sources, the growth of Medical Relief in this Presidency from the early days of the Ship-Surgeon and Wares of the E.I.C. to the opening of the Medical School in Madras. Madras is an institute that has grown to a great size now with its various colleges, laboratories and colleges for nurses and the General Hospital has become responsible for the training of thousands of doctors, saving the lives of millions of people of this Presidency and safeguarding the health of the teeming poverty-stricken millions of men, women, and children of all creeds and castes during peace and war.

It is encouraging to note that Madras has been unique in having taken and utilised the benefits of modern medicine much earlier than other places in India and has developed to what it is today. Surely, in the words of the author "India was neither a vast dark continent like Africa nor was devoid of medical aid or medical practitioner as may well be learnt from Kautilya's Arthashastra, Asoka's edicts, Chola inscriptions and numerous other historical records read and to be read.

The method of recruitment of the Company's Surgeons, the supply of drugs and equipments, the cost of maintenance, the life and work of the medical men engaged by the Company, the original hospital buildings within the Fort St. George and its ramblings in and outside the Fort necessitated by the change of condition and its final site at the Peddymayekam Pettah in 1773 where the nucleus of the foundation of the present General Hospital was laid, are given in a very interesting manner. With the increasing popularity of the hospital, the need for more medical men for service and war purposes in the Carnatic prompted the Company to train local men to the post of dressers. Soon the defect of this system of training in the art was discovered and a medical school on more rational basis was founded in 1835.

We congratulate the author for having produced this interesting document on the growth of medical science in Madras and wish him all success and renewed vigour to unearth several medical and historical documents scattered all over the country and bring to light the glory that was India.

Correspondence

Paludrine Tablets—New Sizes

To The Editors, THE ANTISEPTIC, Madras.

Sirs,

In July of last year you favoured me with the hospitality of your correspondence columns to announce certain changes in the dosage of 'Paludrine'. Owing to these changes in

dosage it has become convenient to make available to the Medical Profession two new sizes of 'Paludrine' tablets. The availability of these new tablets is being announced this month in the medical press, but I shall be glad of the privilege of using once again your correspondence columns to inform the Medical Profession of the reason for introducing the new sized tablets.

MAY '49]

CORRESPONDENCE

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The matter will be better understood, if I first recapitulate briefly the recommendations with regard to 'Paludrine' dosage.

(1) For protection against malarial tertian, and suppression of benign tertian, malaria 0.3 Gm. once weekly, or 0.1 Gm. twice a week at 3 and 4 day intervals.

(2) For radical cure of malarial tertian malaria 0.3 Gm. twice daily for 10 days.

(3) In benign tertian malaria, the greatest hope of a radical cure with 'Paludrine' is offered by the dosage regime of 0.3 Gm. daily until the fever subsides, and then 0.3 Gm. once a week or 0.1 Gm. twice weekly at three and four day intervals for about 6 months.

(4) For "clinical" cure of all types of malaria 0.3 Gm. daily until the fever subsides.

(5) For children over ten, the dosage for adults is indicated both for treatment and prophylaxis. Children under ten should be dosed according to age and size, but the minimum given to any child, however young, need not be less than 0.025 Gm.

It is clear from the dosage schedules enumerated above that the most convenient dosage unit of 'Paludrine' is 0.3 Gm. for adults and 0.025 Gm. for young children. Tablets containing these quantities of 'Paludrine' Hydrochloride have therefore been made available to the Medical Profession.

Medical Services Dept., Yours faithfully,
I.C.I. (India), Ltd., J. M. MCGAVIN,
Calcutta 1. M.B., B.Ch. (Cantab.)

Epidermal Anaesthetic

To the Editors, THE ANTISEPTIC, Madras.

Sirs,

May I crave the hospitality of your esteemed journal to publish the following:

For many years I was in the quest of an anaesthetic, which applied on unbroken skin will produce local anaesthesia like cocaine on mucous membranes. The idea was to make hypodermic injections and incisions on skin painless.

Looking for a vehicle that will penetrate through the skin, I came upon chloroform. A four percent solution of cocaine which dissolves in chloroform solved the problem. I have named the solution "Surfacin". Applied on a pellet of cotton, 4% solution of cocaine in chloroform produces the necessary degree of numbness of the skin without any untoward reactions. For hypodermic injections it serves as a steriliser and anaesthetiser in the same stroke.

Johnson Hotel, Yours sincerely,
Sadar, Karachi, Major N. Z. KHAN,
3/9/48. M.B. (Cal.) I.M.S. (Ex.)

A Request

To The Editors, THE ANTISEPTIC, Madras.

Sirs,

A lady patient, aged 28, with high myopia but otherwise healthy, complains of pain in the

eyes and watering while working with spectacles, minus 16 each eye. Without glasses she feels very uncomfortable. Minus 20 gives better vision but she cannot tolerate it. The present glasses though not sufficient for good vision make her uncomfortable. She is rather desperate. Very slight astigmatism is also there. Compound lenses are not suitable. Dark room examination shows fundus congested, probably as a result of straining. Will any fellow brother explain the causes?

United States of Rajasthan, Your faithfully,
Chandhvir Hospital, PRAYAG SINGH,
P.O. Bijolian (Vewar) Ophthalmic Surgeon.

Profession and Practice

To The Editors, THE ANTISEPTIC, Madras.

Sirs,

I was very much interested in Dr. A. S. Patnaik's reply to Dr. P. A. S. Raghavan's address in your issue of Feb. 49.

I have great faith in the Indian Medicine as practised by our ancestors. While Dr. Patnaik talks so much of Indian Medicine and praises it to the skies, I have not come across a single practitioner of Indian Medicine practising his line of teaching. On the other hand, he competes with the allopathic practitioners and uses Western medicines only without knowing fully what they are, how they act and what are their ill-effects.

I know people, so-called Pandits, B class practitioners, who can neither write nor read English using Western medicines, injections etc. indiscriminately. When such being the case why talk loud of Indian Medicine but use Western Medicine? It is high time the Government places some sort of ban towards this sort of quackery.

Hegde Clinic, Yours faithfully,
Coondapoor, S. R. HEGDE,
19-2-49. C.M.P. (Regd.)

Recurrent Mania

To the Editors, THE ANTISEPTIC, Madras.

Sirs,

A male and a female are under treatment for the last 12 years. Both of them (brother and sister) are suffering from chronic mania (insanity). The male got the first attack at the age of 20 years, in 1935; subsequently, he got the attacks in 1938, 1942, and 1946. The female got the first attack in 1935, second attack in 1939 (July) and the third attack in May 1948. She is still suffering from it and is not cured as yet. Both of them, during the attack, were treated by Dr. W. C. Roy's pills, and they became perfectly well all the same. But the female who was attacked for the third time in May '48, has still not responded to Dr. W. C. Roy's pills this time. Nothing abnormal could be found in both cases in the system.

Will any fellow brother throw light on this subject, and let me know what line of treatment should be followed? The 5th attack in the male case is expected in 1949 or 1950,

What medicines should be prescribed, so that the fifth attack can be aborted, as every attack certainly damages the brain, and it will be a difficult job to treat during the 5th attack? Vitamin B has been found useful in many nerve diseases. Can Vitamin B therapy help in aborting such attacks also?

I shall be obliged if some one will throw light on this subject and also as to why relapses occur at such intervals?

Sincerely,
S. O. MATHUR,
Dibrugarh, Assam.

Liver Extract in Typhoid Cases

To the Editors, THE ANTISEPTIC, Madras.
Sir,

There are certain cases of typhoid, whose temperature does not come down to normal even after twenty-one days. I generally give small doses of liver extract by mouth, one drachm twice a day in half ounce water before mosambi juice. Even temperatures like 99° or 100° F takes a very long time to come to normal due to the low vitality of the patients. The temperature of these patients came down earlier than the expected time by giving this drug. I request my fellow practitioners to try this harmless drug and note its result.

Yours obediently,
R. J. ADHIVAYU,
T.V. Rly.

How to Attract Medicos to Rural Areas?

To the Editors, THE ANTISEPTIC, Madras.
Sir,

I read with interest the letter of Dr. G. N. Sinha—"The Problem of Rural Medical Aid in India", that appeared in the ANTISEPTIC of February last.

We agree with him in various points. In our opinion it is not only in the dearth of the qualified personnel that the rural health scheme could not be undertaken, but it is due to the utter negligence of the Government who do not care to utilize the available doctors.

We know of hundreds of unemployed doctors and some of them are doing business of tube wells, insurance agents, banking and even private tutors! Is it not disgraceful? Is it not a national loss and wastage? Why is this so? Because the doctors are not given adequate salaries for their maintenance, they are not encouraged to settle in a rural area. The problem of shortage of doctors cannot be solved only by producing more doctors who will naturally crowd in the towns and cities. It will rather increase the number of unemployed.

What do we see in the towns? The compounders and quacks are allowed to practise though plenty of qualified practitioners exist. This is due partly to the ignorance and partly to the great economic distress of the people. We have seen on several occasions that the lay people would call a compounder or a quack with a fee of Rs. 1/- or -- 8/- only even when

M.B.'s are available on Rs. 2/- only. We are going to raise the standard of medical education! But we are blind to the fact that quackery is in full force even in towns which are overcrowded with qualified medics. This is the real picture of medical profession in India. We only think of the statistical number of doctors while we neglect their utility!

Lastly, we fully support the suggestive scheme brought forward by Dr. Sinha for a rural dispensary and we have no doubt that hundreds of qualified men will be available for rural health centres if attractive terms are offered as suggested by him. We regret to say that Rs. 60/- is being offered to a Medical Officer of a District Board when the cost of living has increased by 500 per cent. No one can expect sincere and efficient work from an ill-paid and half-starved employee who will always be anxious for maintaining his growing family.

Yours faithfully,
Dr. S. C. SEN.

Trigeminal Neuralgia?

To The Editors, THE ANTISEPTIC, Madras.
Sir,

I shall be highly obliged if you can permit me to say something on the article on "Trigeminal Neuralgia" published in your Vol. XLVI, No. 2, Feb. '49.

In this the writer has mentioned that the case was relieved by Mag. Sulph.

The writer has not mentioned the signs which he found. He has mentioned only the symptoms. My questions are:—

- Was there hyperaesthesia?
- Was there hyperalgesia?

Was there muscle-spasm—during the paroxysm of pain.

Was there history of scratch or wound or any relation to dusty business?

I emphasize these points because there is a condition known as tetanus dolorosus in which only sensory part is affected. In this condition—

- (1) There is absence of motor spasm.
- (2) There is hyperaesthesia.
- (3) There is hyperalgesia.

In trigeminal neuralgia you have no objective signs.

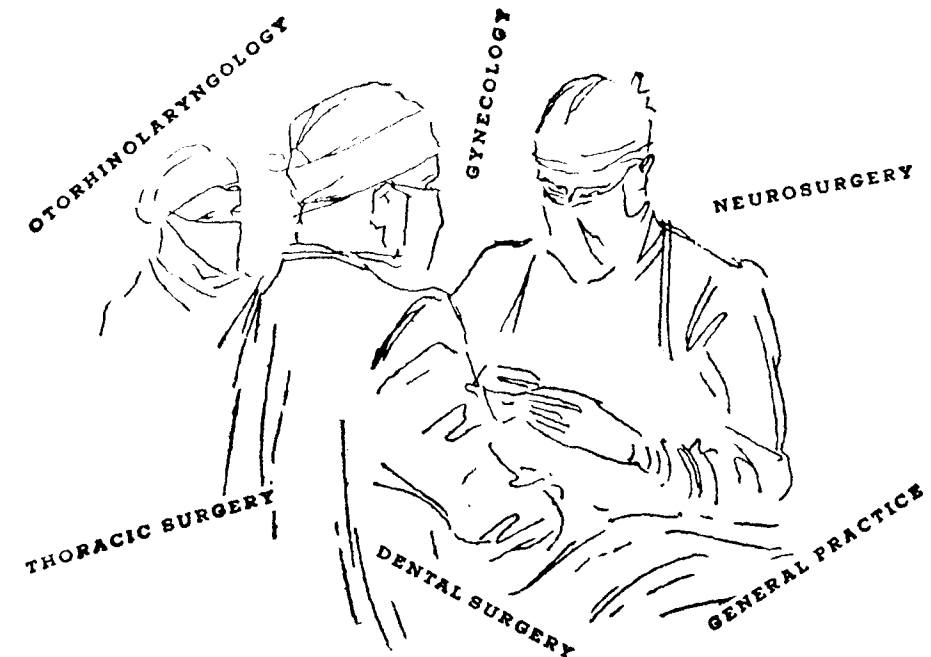
Unless these points are cleared, I insist that it was a case of tetanus dolorosus, which I have met in my practice and which, too, was relieved by Mag. Sulph.

I shall be much obliged if he can provide me with the information which I require.

(Ref. Text Book of Pathology by Banerjee).

Ex-Govt.
Medical Officer,
King's Gate 1542,
Ahmednagar.

Yours truly,
LACHMANDAS CHATRU-
SINGH PARDESHI, L.M.P.



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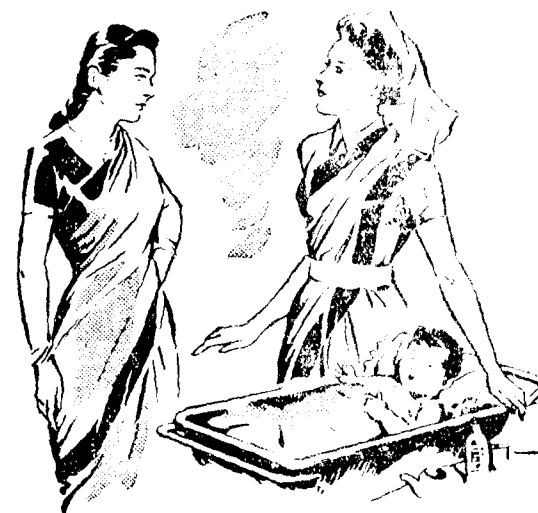
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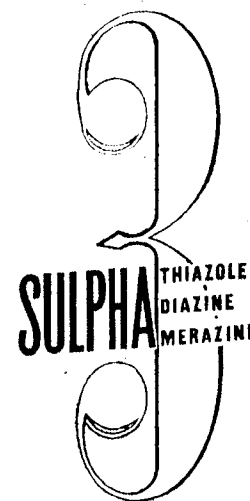


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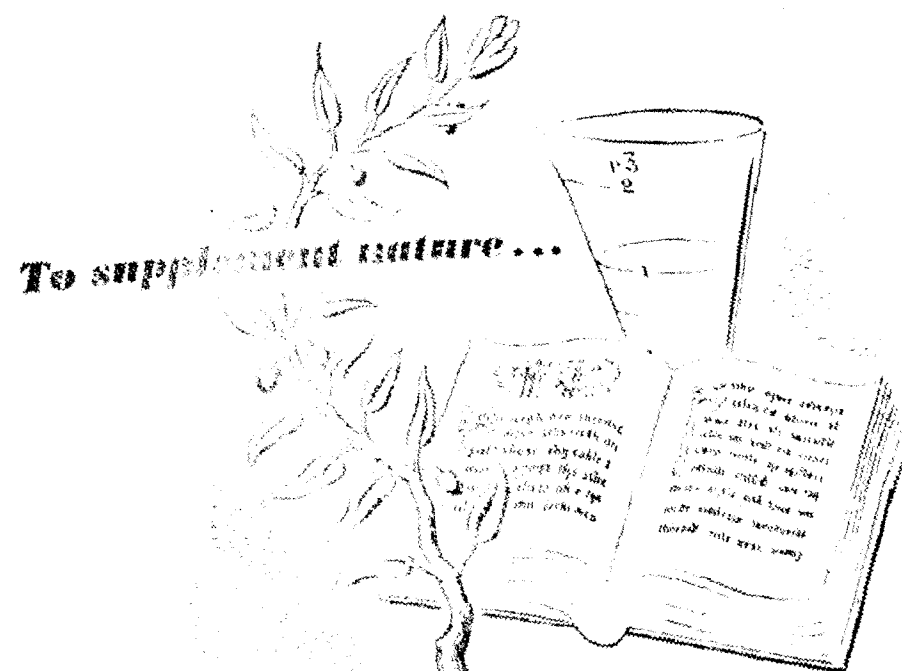
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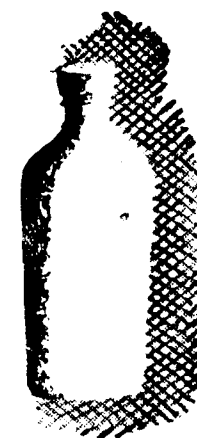
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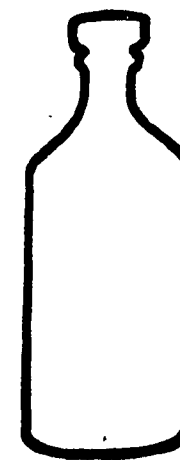
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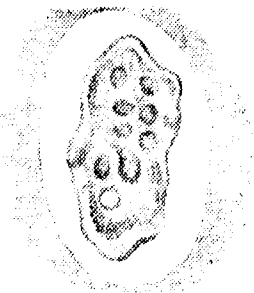
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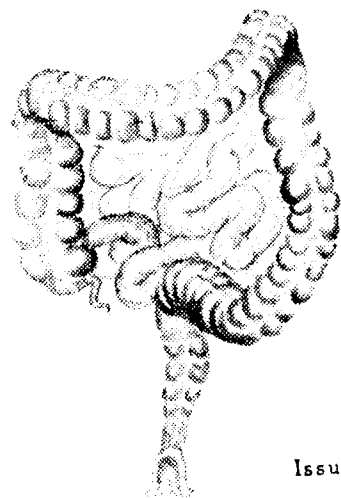
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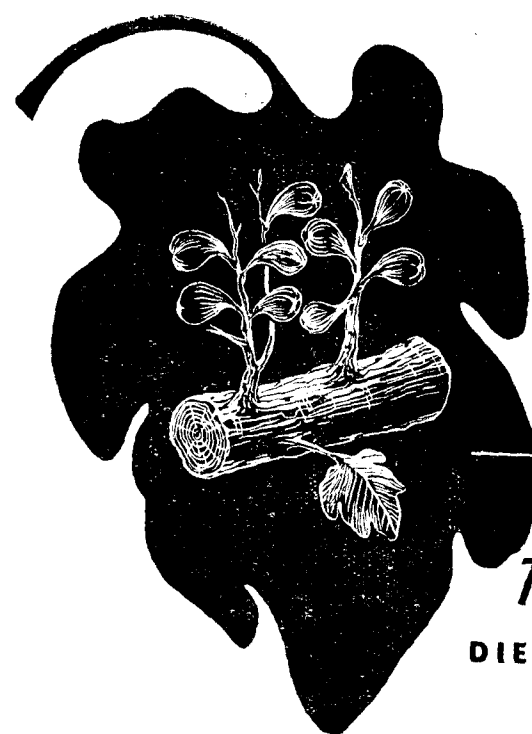
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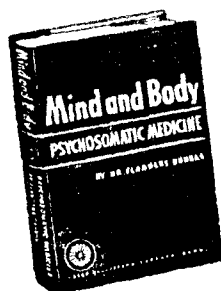
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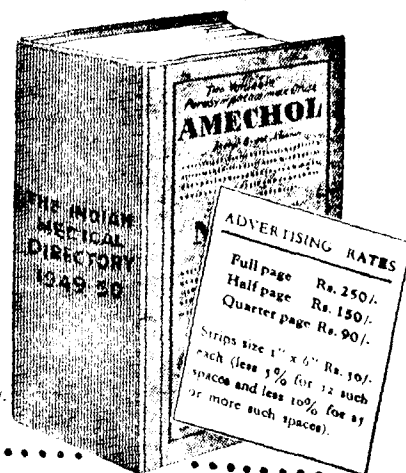
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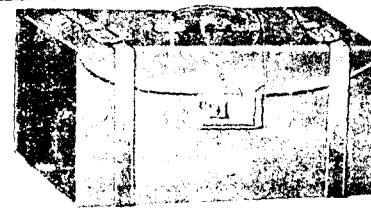
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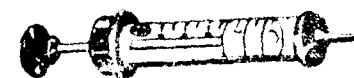
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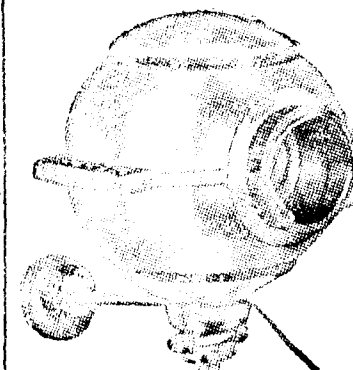
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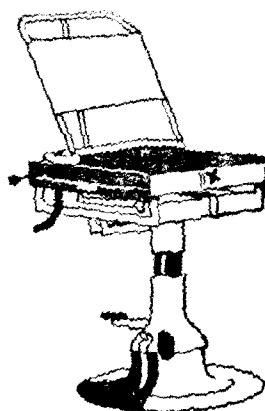


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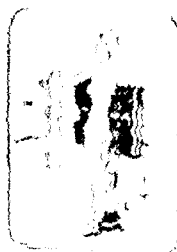
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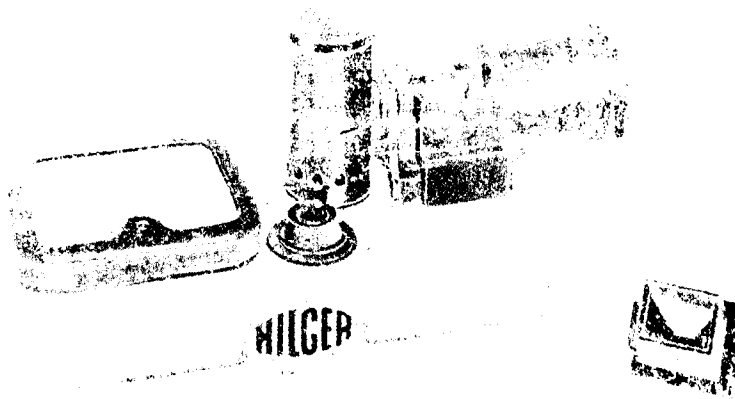


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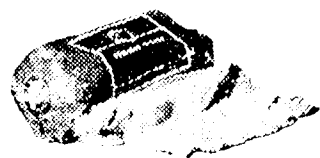
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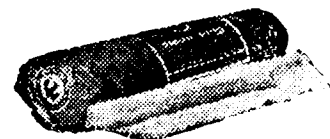
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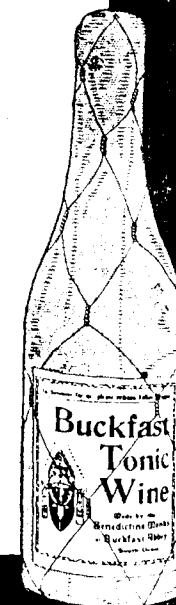


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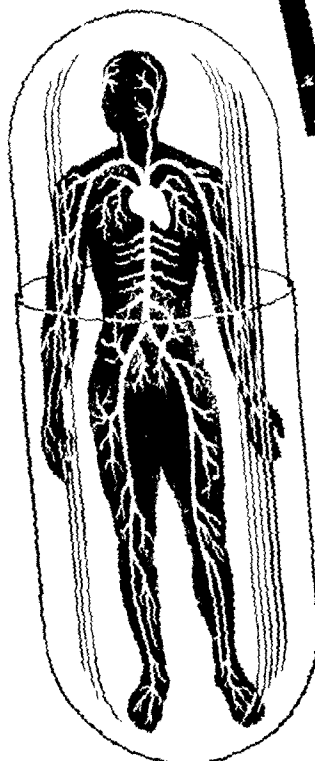
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TABLE

	Time between onset of fever and relapse	Duration of afebrile stage prior to relapse	Duration of relapse
Case 1	16 days	3 days	4 days
" 2	23 "	5 "	17 "
" 3	27 "	4 "	12 "
" 4	31 "	15 "	9 "
" 5	22 "	4 "	16 "
" 6	20 "	2 "	16 "
" 7	49 "	13 "	8 "
" 8	19 "	4 "	10 "
Case 9 1st relapse	30 "	5 "	16 "
" 9 2nd relapse	52 "	6 "	17 "
" 9 3rd relapse	75 "	6 "	18 "

In the majority of cases the relapse was an abridged and somewhat mitigated repetition of the initial stage. However, in at least 3 cases it equalled or even exceeded the original attack in severity. The spleen was palpable during the relapse in all cases.

A fresh crop of rose spots during the relapse was distinctly visible in one case only.

Fever.—The temperature exceeded the 105°F mark during the relapse in 3 cases; 4 cases showed temperatures ranging from 103°F to 104°F. In the two remaining cases the highest temperature was 102°F.

Variations in course.—In one case profuse multiple hæmorrhages occurred in coincidence with relapse. The patient was a boy of 6 years, whose primary attack of typhoid fever appeared to be only moderately severe. The disease seemed to take an uneventful course, with the temperature subsiding about a fortnight after onset. However, after 4 days of well-being the child became very ill and toxic again, the temperature rose, and at the same time a tendency to bleeding became manifest. Hæmorrhages occurred first from the bowels; a few hours later hæmaturia was observed which was followed by free bleeding from several small furuncles of the scalp which had been incised a few days before. Cutaneous petechiae and conjunctival suffusions were present.

The condition was obviously critical, but came finally under control after treatment with Vitamin K, Multivitamin, Coagulen in large doses and Calcium Gluconate injections.

One case relapsed three times, the disease lasting altogether 115 days. The patient was a Hindu girl of 14 years. Each time the temperature came down to normal it looked as if the child had shaken off the disease completely and it became difficult to keep her in bed. However, the relapses were severe, with high temperatures and much constitutional disturbance. All 3 relapses resembled each other closely. They were shorter than the primary attack, but the clinical picture of each of them was grave. The febrile

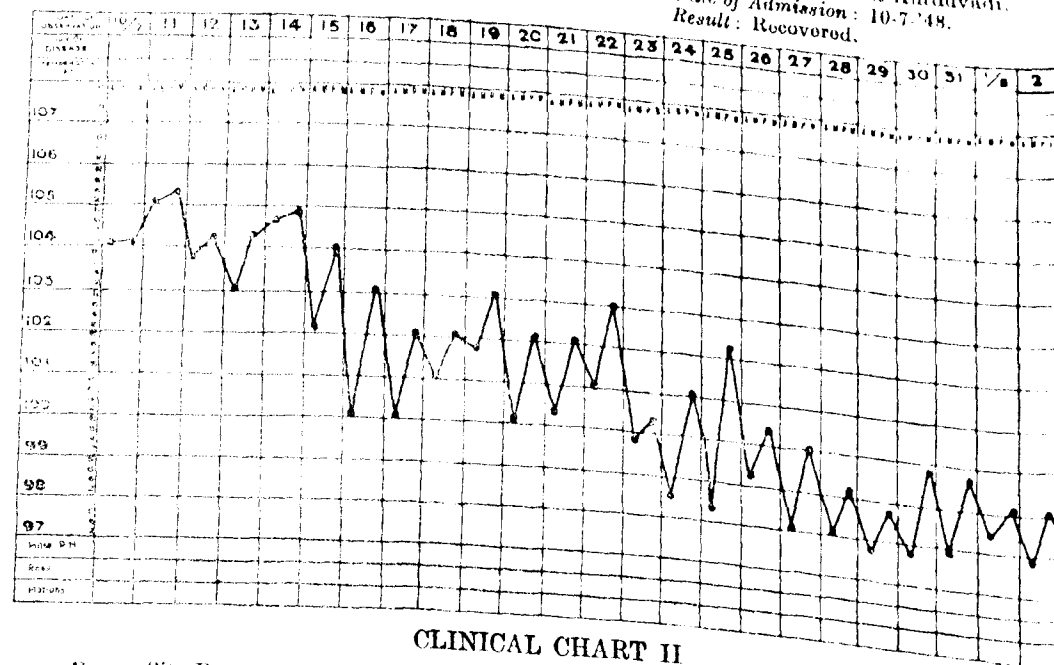
period of the relapses lasted about 17 days, and the free interval between the attacks about 6 days. This periodic recurrence of the whole cycle is certainly most remarkable. The Temperature Chart of this case is given below:

CLINICAL CHART I

Name: Sita Dattu.
Disease: Enteric Fever.
Date of Discharge: 29-10-'48.

Age: 14.

B. L. Rly. Hospital at Kurduvadi.
Date of Admission: 10-7-'48.
Result: Recovered.

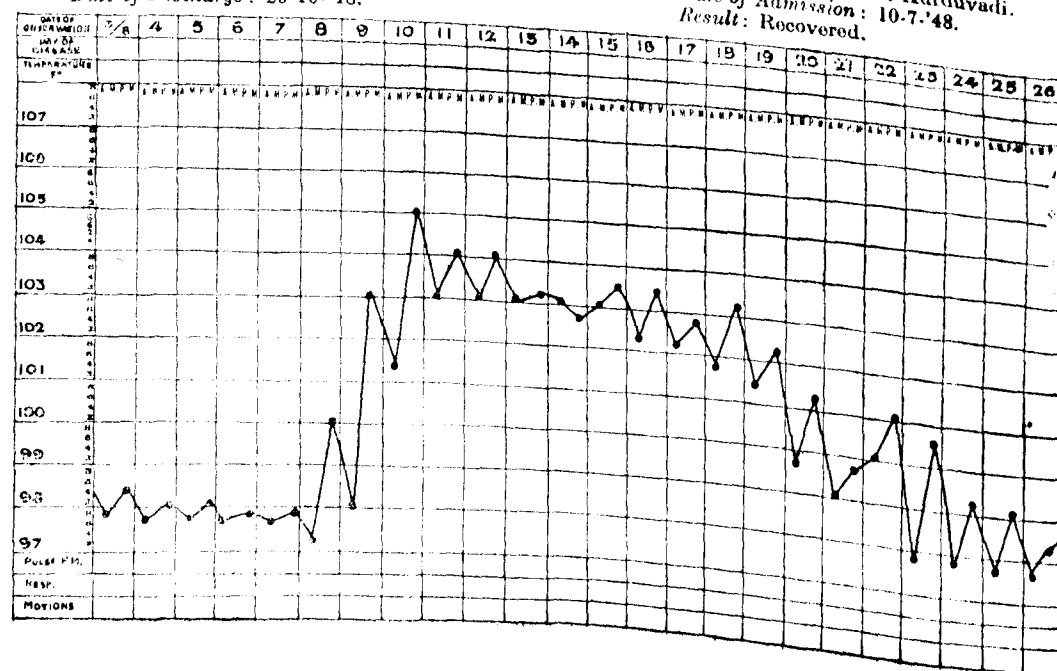


CLINICAL CHART II

Name: Sita Dattu.
Disease: Enteric Fever.
Date of Discharge: 29-10-'48.

Age: 14.

B. L. Rly. Hospital at Kurduvadi.
Date of Admission: 10-7-'48.
Result: Recovered.

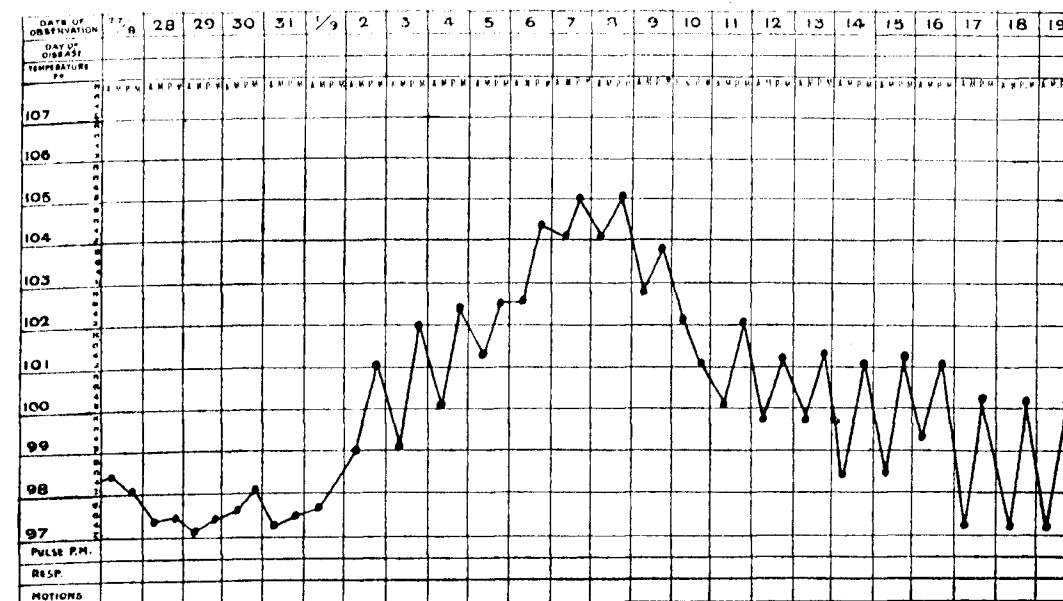


CLINICAL CHART III

Name: Sita Dattu.
Disease: Enteric Fever.
Date of Discharge: 29-10-'48.

Age: 14.

B. L. Rly. Hospital at Kurduvadi.
Date of Admission: 10-7-'48.
Result: Recovered.

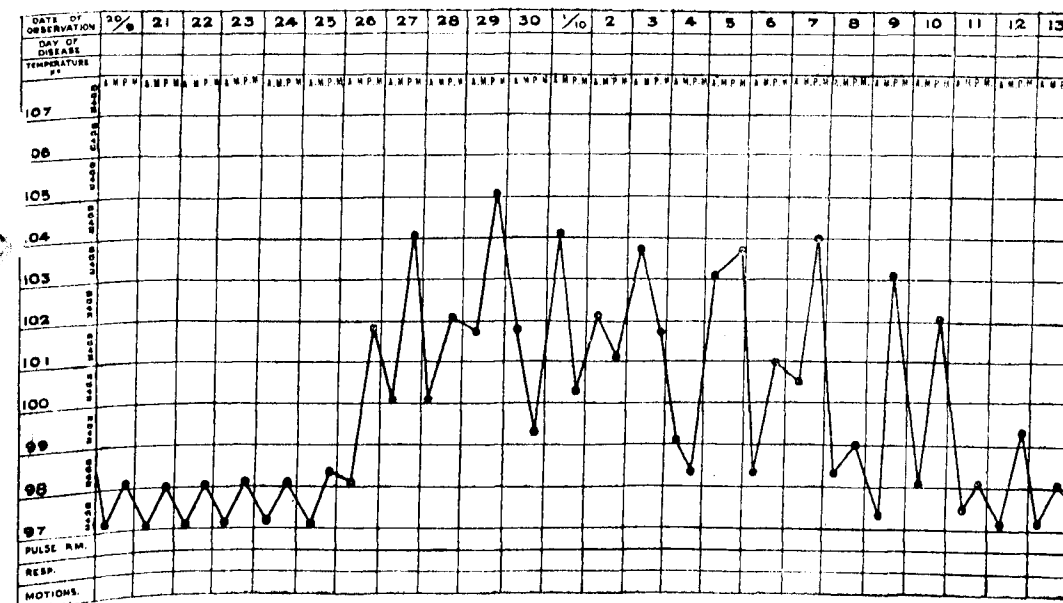


CLINICAL CHART IV

Name: Sita Dattu.
Disease: Enteric Fever.
Date of Discharge: 29-10-'48.

Age: 14.

B. L. Rly. Hospital at Kurduvadi.
Date of Admission: 10-7-'48.
Result: Recovered.



CLINICAL CHART V

Name: Sita Dattu.

Age: 14.

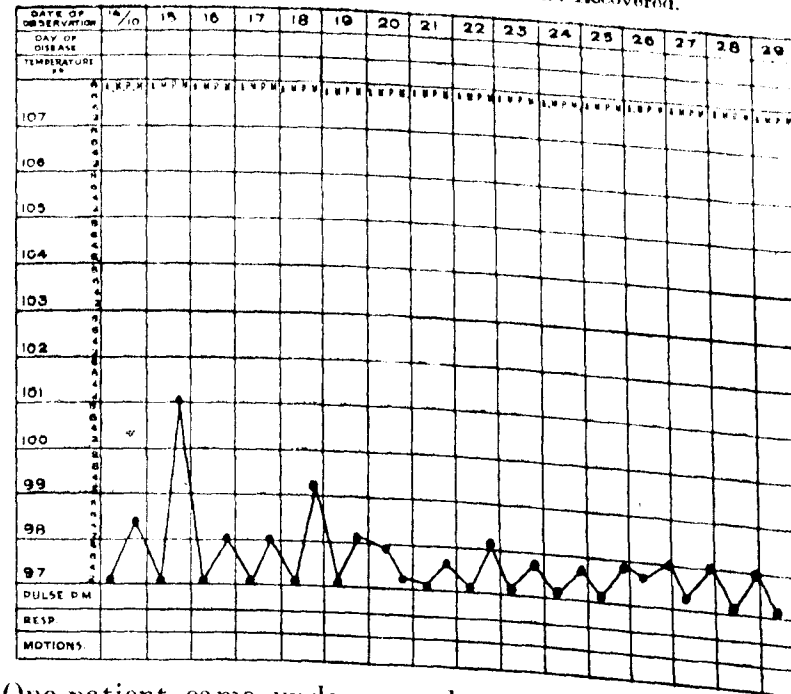
B. L. Rly. Hospital at Kurduvadi

Disease: Enteric Fever.

Date of Admission: 10.7.48.

Date of Discharge: 29.10.48.

Result: Recovered.



One patient came under our observation only at the onset of the relapse. He was an European of 15 years, who had been treated in Bombay for an attack of typhoid fever. About 2 weeks after the temperature had come down to normal he had been released from treatment and sent to Kurduvadi to recuperate. On his arrival here he fell ill with a typical attack of typhoid fever with splenic enlargement and a fresh crop of rose spots.

None of the 9 relapsing cases had ever had a T.A.B. inoculation.

As mentioned before, we have been treating also 3 cases who showed a recrudescence of fever after an incomplete lysis. These cases were not included in our series of relapsing cases as it is open to discussion whether they were true relapses. It is, however, probable that they were closely related to our group. One of these 3 cases proved fatal. The patient was a woman of 28 whose temperature chart after an acme of about 10 days duration showed the 20th day of illness. The highest temperature on that day was only 99.2°F. From the 21st day onward there was again a gradual rise of fever upto a new fastigium of 104-105°F. The patient died of heart failure on the 59th day of hospital treatment. The temperature remained high upto the end.

The 2 other cases of this group showed a rise of temperature during convalescence, of short duration only, and with few other symptoms and signs.

Treatment.—The treatment of relapsing typhoid fever is, in principle, the same as in uncomplicated cases. In the present series, much attention was paid to dietary measures, and a high caloric, high proteins diet was aimed at in all cases. There can be no doubt that in relapsing cases with prolonged pyrexia and consequently increased demand upon the protein stores of the body a high protein, high caloric diet is of supreme importance. In our endeavour to provide a seriously ill patient with a suitable diet, containing 3,000-4,000 calories, we made extensive use of a buttermilk—sweet oil—and attah mixture (O.A.B.J.) which had served us before as an infant's food. The particulars of preparation of this food have been described elsewhere¹. The actual recipe is as follows:

2-3 oz. jaggery (or sugar which, however, is dearer) dissolved in 1,000 c.c. hot buttermilk. A few grains Sod. Bicarb may be added to the mixture.

2-4 oz. gingelly oil or karadi oil and 1-1½ oz. attah are mixed and heated until a brown paste results. Instead of the oil ghee which is more expensive may be used as well. The buttermilk is then slowly added to the paste and the whole mixture boiled for 2-3 minutes.

It contains about 30 calories per ounce, but the caloric value can be considerably increased by concentrating the amount of oil and attah to double strength.

In the majority of our cases it has been possible to give a patient 1,500 to 2,000 c.c. of this mixture in small sips, distributed all over the day. Our experiences with that food were rather encouraging. It was well accepted by the patients and always well tolerated. In addition to it the patients were allowed to have any light food they felt inclined to eat; they were especially encouraged to take liver soup, eggs, rice, milk, glucose etc.

Most of our patients were also given Multivitamin tablets. Drug treatment was resorted to only as an auxiliary measure. In all cases, where the pulse was rising and of higher frequency than would be expected from the body temperature, Digitalis was given in efficient doses. Usually 6 tab. Digoxin a day were given to an adult, and correspondingly less to a child, but occasionally even higher doses were prescribed. We have gained the impression that Digitalis has a definite beneficial influence on the heart in those cases, and in our opinion it should be resorted to,—provided it is used with discrimination.

Discussion.—Comparison of our figures with other statistics shows, as already mentioned, a higher relapse rate than previously recorded.

Charles R. Box¹ estimates the actual number of relapses as being 5–15 per cent. Other writers arrived at similar conclusions: Curschmann,² according to Brugsch² reckons with 6–12% relapses and Strumpell³ states that the relapse rate in different epidemics varied from 4 to 16%. He himself saw, among 600 cases, 9% relapses. Among his patients he saw only once a case who relapsed 3 times.

As far as we can see, no relapse rate higher than 16% has been described. Our present figure, however, amounts to almost 40% relapses, if the 3 doubtful cases are excluded, and if they were accepted as true relapses our figure would surpass 50%.

The age incidence of our relapsing cases is certainly of interest. However, it is impossible to draw any far-reaching conclusions from the fact that it affected only young people.

It seems significant that none of the relapsing cases had ever been inoculated against T.A.B. and it would appear that relapsing typhoid fever is a feature of the disease in the un-protected.

As the relapses in our cases concerned patients of different races and communities—Indians as well as Europeans—it seems likely that the tendency to a relapse is a feature of certain epidemics and not of any individual or group predisposition.

Summary.—Among a total of 23 cases of enteric fever, treated in 1947–1948, there were 9 relapsing cases, i.e., almost 40 per cent of all cases.

Among the same series there were further 3 cases of recrudescent fever which were, however, not listed as relapses.

The percentage of relapses in our series is considerably higher than the figures given in text-books.

Some factors, probably responsible for the condition, have been discussed.

Acknowledgement.—I am indebted to the Medical Staff of the B. L. Rly. for their interest and collaboration in the preparation of this paper.

References:

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2. Brugsch, T.—*Innere Medizin*, Urban and Schwarzenberg, 1930.
3. Curschmann.—Quoted by Brugsch.
4. Koonigsfeld, E. G. A. (1948)—*Journ. I.M.A.* 17:8.
5. Strumpell, A.—*Spezielle Pathologie und Therapie*, 28th Edition, 1929.

Thread Worm Infestation *

M. C. RAMA AYYANGAR, B.A., M.B., B.S.,

Civil Assistant Surgeon, Tellicherry.

ENTEROBIUS (or *Oxyuris*) *Vermicularis* commonly known as thread worm or pinworm belongs to the nematode sub-group of helminths and is a cosmopolitan, infesting many members of the same family especially children. It is white, small and resembles a bit of thread; the male is smaller (3–5 mm.) and is seldom seen as it does not migrate out to the anus like the female which is larger and measures 10 mm. $\times$ 0.6 mm.

The disease spreads by *autoinfestation* essentially. The ova are also air borne. One may however get infested through ingesting food (improperly washed raw vegetables and fruits) which has become contaminated with the ova found in the dust of the houses in which the infested persons live, or rarely through inhalation of the eggs from the dust, infected garments etc. The habitat of the worms is the large gut (caecum, rectum) and in some cases the appendix or small intestines. Their eggs cannot develop into adults unless they leave the host. As soon as the ova are ripe the fertilised female worm begins to migrate to the anus of the host at nights so as to deposit the eggs—there are as many as 10 to 15 thousands in each worm—in the natal folds and in the perianal region. This causes itching (*pruritus ani*) at nights and the host (usually the child) scratches, crushing the worms and setting free the ova. Even otherwise the ova are deposited in the perianal skin. Later they may be conveyed in the finger tips to the mouth or may become spread all round the house in the dust; thus the eggs are ingested or inhaled and infestation occurs. The eggs are colorless, thin shelled, oval, flattened on one side (*plano convex*), $50\mu \times 20\mu$, and contain almost fully-formed, coiled embryo but are rarely seen in faeces. These, when they enter the host, hatch out in the duodenum and the liberated larvae develop in the small gut. As they grow older they pass into the large intestines where they become mature, live in the submucosa and mate. This marks the end of the male's life but the female travels downwards towards the rectum.

Symptoms.—1. *Itching of the anus or vulva* (in children) at nights is the most common and cardinal symptom. This *pruritus ani* is annoying and even intolerable and is caused by the migration of the female worms in anal canal.

2. The worms may produce a *mucous colitis* (catarrhal) and at times recurrent attacks of *subacute appendicitis*.

3. Restless sleep, frequency of micturition, bed wetting, vulvitis may also be caused.

* Specially contributed to THE ANTISEPTIC

4. Some authors believe that these worms may be responsible for poor appetite, loss of weight, fidgettiness, fretfulness, irritability and even fits.

Diagnosis.—The newer methods of recovering the ova from the perianal skin by scraping it with Hall's NIH Anal-Swabs or by using the adhesive cellulose tape or paste are far more reliable and give positive results in more than 90% cases especially if numerous swabs are taken repeatedly, while the examination of motions under the microscope gives only positive results in 1% of the infected cases and the floatation method in 10% cases. The ova are rarely seen in faeces, but the adult female worms are seen especially after an enema.

Prophylaxis.—The most important measure is to prevent *autoinfestation* by enforcing general measures of personal cleanliness, by insisting on the scrupulous washing of the hands especially before meals very well, by having the finger nails of the infected persons pared short, by making the children wear cotton gloves and by allowing them to sleep in cotton drawers or knickers on separate beds. It should be insisted that the clothing is exclusive and separate for each inmate of the family and that the bed clothing and dress are changed often and washed or dry cleaned periodically. It is also necessary to wash the raw vegetables (cucumbers etc.) that are consumed as such, and the fruits thoroughly before eating them. All these precautions are necessary for the success of any treatment as well.

Treatment.—Till recently it has been difficult, almost impossible, to eradicate the infestation with thread worms. This is reflected in the many remedies that have been used for this simple ailment. The response to treatment varies with the severity and duration of the condition. Light infections may be cured in a few weeks by rectal injections alone while heavy infections will require many months for a complete eradication by means of enemata and oral medicaments.

1. *Hygienic measures* must be enforced before therapy by drugs is attempted. The first step in treatment is scrupulous attention to cleanliness of hands and buttocks. The perineum should be washed well with soap and water twice daily especially after the stools. All bed clothing and underwear should be boiled daily.

2. The chief emphasis in treatment must be laid on the *prevention of autoinfestation* and on the complete and comprehensive *treatment of all the individuals of the family or household simultaneously*.

3. To achieve the former, it is necessary to *prevent or treat itching at anus*. *Nupercainal* (Ciba) or *Scuroform* anaesthetic ointment (M & B) or *Benzocaine* ointment may be applied to the anus,

Pruritus ani can also be relieved by applying *Ung. Hyd. Ammon* (B.P.) 5% to the anus. But it must be understood that these local applications do not affect the ova which have an impervious chitinous shell but the mercurial ointment may destroy the worms. A specific treatment for the *pruritus ani* is a small enema of 2-3 oz. of tap water which will remove the worms, give great relief from itching and thus prevent auto infestation.

4. If the rectum could be kept free from worms for a sufficient length of time the patient will get cured in a majority of cases as re-infestation is prevented. The gravid females are best eliminated by *retention enemata* which wash out the large gut of the worms. First an aperient is given; then the bowel is washed with Soda Bicarb solution. And then any one of the following medicinal enema is given:

(a) *Hot hypertonic saline enema* (1 tablespoonful of NaCl. to half pint of water) at 110°-115°F. and retained for 10 minutes.

(b) *Inf. Quassia* is an excellent anthelmintic. After the saline enema, either half to one pint of a fresh infusion of Quassia 2.5% (Quassia chips 5 ii steeped in half a pint of water) is injected slowly high up the rectum with the foot of the bed raised, or Sod. Chloride 3 i Inf. Quassie Recens. #T is given daily for 4 or 5 days. The worms are killed but not the ova; 4 to 6 oz. can be injected in children. Instead half a pint of infusion of Calumba, Chiretta or Gentian or other vegetable bitters may be used similarly.

(c) *Hexylresorcinol* has been used with good effect in America and can be tried if the other enemata fail. First a soap and water enema is given and then Hexylresorcinol 1 in 2,000 solution (1 Gram dissolved in 1 quart of water) is given as enema—2 pints in adults and 5-10 oz. in children—and retained for 10 to 20 min. As soon as the enema is expelled the buttocks should be cleaned well with soap and water to avoid any irritation to the skin which may result. The enema is given once daily for 6-10 days.

(d) Previously the following have been also tried. Ol. Turpentine (1 oz. in one pint of Mucilage Amyli), Tannic acid solution (1 in 500), Decoction of Oak Bark, Decoct. Aloes, Tr. Ferri Perchlor (60 m) in half a pint of water, Copper Sulphate 1 in 2,000, Thymol 1 in 2,500, have been all used as rectal irrigations with varied success. While enemata wash out the gravid females from rectum, the young worms while still in the small gut or caecum can be killed only by oral medication. This is done especially in cases which resist local injections or in conjunction with them. Two new drugs are now available and have been found recently to be very effective.

1. Diphenan (B. P. C.) is claimed to be an active and least toxic oxyuricide. It is P-Benzyl, Phenyl Carbamin acid ester. It is colourless, odourless and almost tasteless. The proprietary preparations are Diphenan (BDH), Butolan (Schering) and Oxytan. It is remarkably free from toxic effects and hence useful for treating children. Rarely some gastric disturbance may occur but it passes off in a few days. The drug is active *in vitro* and *in vivo* against the adult worms and not the ova and hence *reinfestation must be prevented and the whole household treated*. It is preferable to Gentian Violet which may give rise to undesirable toxicity. The dosage for the adults is one tablet of $\frac{1}{2}$ Gm. ($7\frac{1}{2}$ grains) three times a day *after meals* for 7 days and for children $\frac{1}{4}$ Gm. (4 grains) t.d.s. It may be necessary to repeat this course after a week's interval and in resistant cases to give *Diphenan and Gentian Violet Eucscals during alternate weeks for a total of 4-6 weeks and this may be combined with the retention enemata* (already mentioned) for greater success.

2. Gentian Violet or Crystal Violet (B.P.C.) is an aniline dye derivative recently introduced and tried in oxyuriasis. It is also called Methyl Rosaniline and it is Hexa-Methyl Para-Rosaniline Hydrochloride. It is efficient in the control and eradication of the Pin Worms (Faust) and cheaper but the chief drawback is the frequency of nausea caused by it, though, in many, no side effects are observed. Rarely some get vomiting, mild abdominal pain or diarrhoea, while a few others headache, dizziness and lassitude. The first group of symptoms are minimised by administering *the drug as Eucscals* (enteric coated tablets) 30 to 60 minutes before meals. The tablets should not be bitten or chewed. By the enteric coating (using a combination of oils, waxes and fatty acids with high melting point or a mixture of Salol, Shellac and Resins or Kertain coating) the drug is protected from being acted on by the gastric juice and also its effect is delayed, the eucscals beginning to disintegrate gradually in 6-8 hours in the small gut only. The toxic symptoms subside, however, if the drug is omitted temporarily for a day or two and the therapy can be resumed a few days later without the ill effects. But the drug should not be administered to patients with severe cardiac, hepatic, renal or gastro-intestinal diseases; Ascariasis is another contraindication. Diphenan may be used instead. The dosage is 3-20 grains daily in 3 divided doses for each year of age upto a maximum of 3 grains daily (for adults) orally before meals for 8 days followed by a second similar course after a week's interval. The proprietary preparations are (a) Eucscals Gentian Violet No. 8 (Eli Lilly Co.) (3-20 gr.) and Eucscals G.V. No. 9 ($\frac{1}{2}$ gr.) $\frac{1}{2}$ gr. and 1 gr. (b) Gentian Violet Pills (B.D.H.) Enteric coated, each of $\frac{1}{2}$ gr., 1 gr., (adults) Dragees (Dr. A. Wanders, Switzerland).

3. *Phenothiazine* or Phenovis or Thiodiphenylamine is another recent addition to our armamentarium; it is effective but

too toxic for routine use. It is used in tablet or granular form in doses of 100 grains daily in divided doses for adults for 7 days, 30 grains daily for children between 5 and 12 for 5 days and 15 grains daily for 5 days for children below 5. It may be necessary to repeat the course a second time after an interval of 10 days. The urine is stained red; aplastic anaemia may be caused and so this drug is rarely used.

4. Still another recent remedy is *Lubisan*. For adults 6 capsules (each containing 3 grains) are given each morning on empty stomach and food withheld for 3 hours more; it is repeated for next 3 days and rest is given for 4 days; again the capsules are given for another 3 days. Proportionately smaller doses can be given for the children. Toxic symptoms due to the drug are rare.

5. The time honoured anthelmintics have also been used with beneficial results in oxyuriasis and they are Hexylresorcinol, Santonin, Ol. Chinopodium and Carbon Tetrachloride, Tetrachlorethylene and Betanaphthol. Any of these may be tried with advantage in resistant cases or when toxic symptoms are produced by the newer drugs.

(a) *Hexylresorcinol* or Caprokol is 1:3 Dihydroxy-4 Hexyl Benzene. Brown has found this effective. Only a light meal is given the previous night and no breakfast on next morning. The drug is given in doses of $1\frac{1}{2}$ grains for each year of age upto 10 years and 15 grains for all above ten years; it is administered orally as pills or gelatine capsules or as enteric coated tablets, swallowed whole and not chewed up, with a glass of water on an empty stomach. A saline purge is given after 3 hours and food is withheld for another 2 hours. This has to be repeated twice or thrice at intervals of 2 weeks or 10 days. The drug is mildly toxic. Wright and Brady hold that this drug gives better results as enema.

(b) *Santonin* (1-3 gr.) along with Calomel in 2 divided doses followed by a Saline purge can be given as in ascariasis once a week for 2-3 times.

(c) ℞ Ol. Chinopodium	.. m xv
Carbon Tetrachloride	.. m xxx
Liquid Paraffin	.. ℥ i

This is given to adults (half at 5 a.m., the other half at 6 a.m., and Mist. Alba ℥ iii at 8 a.m.) as in hookworm disease. But it is toxic for children.

(d) ℞ Tetrachlorethylene	.. m lx
--------------------------	---------

Saturated Solution of Sod. Sulph ℥ iii. Half is given at 6 a.m. and half at 8 a.m. and in children the dose is 0.2 c.c. per year of

age. The drug may also be taken in gelatine capsules (e.g. *Tetracup* (B. W. & Co.) and followed by a Saline purgative.

(c) *Betanaphthol* (B.P.)

Dose is 3-10 grains; it may be given in cachets or in an ounce of castor oil. Other useful remedies which may be tried are:

1. *Methylene Blue* is efficacious when given for 7 days in doses of gr. 3, three times a day. It is non-toxic.

2. *Storarsol* (M & B) the pentavalent arsenical is also useful in many cases. In young children one $\frac{1}{2}$ gr. tablet is given b.d. in older children one tablet t.d.s. for 7 days and the course is repeated after a week's interval. In adults one 4 grain tablet is given on first day, two tablets on second day, three on third and four on each of next 4 days and after a week's interval a second similar course is given. In obstinate cases the courses may have to be repeated more times.

3. *Carbarsone* (Leucarsone M & B) is administered orally in doses of one tablet of 4 grains twice daily after meals for 10 days; a further course may be given after an interval of 10 days.

4. *Allisatin* (Sandoz). These tablets containing Allium Sati-vum fixed on highly activated charcoal are odourless and tasteless and act as oxyuricide when swallowed whole with water *before meals* in doses of 2 tablets t.d.s. for three weeks (adults) or 1 tablet t.d.s. (in children) and repeated after an interval of one month. Purgatives should be given once a week during the treatment.

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Procaine Penicillin: Effect of Single Daily Injections

Jones and Shooter of St. Bartholomew's Hospital, London, report on the value of Procaine Penicillin in prolonging the presence of Penicillin in the blood, and being relatively free from undesirable complications. The study relates to the treatment of 45 ambulant and four ward patients of both sexes, their ages ranging from 17 to 83. The diseases for which treatment was given (300,000 units daily) included carbuncles, furunculosis, breast abscesses, cellulitis, pulp infections, and infected abrasions with lymphangitis and adenitis. Detectable amounts of Penicillin were found between 18 and 26 hours in 37 out of 45 cases after their 1st injection of 300,000 units of Procaine Penicillin. After the 2nd injection detectable amount after 18 to 26 hours were found in 93 out of 109 samples.

It has proved possible with one daily out-patient attendance and rest at home to manage some patients with this drug, who would otherwise have required admission into the hospital wards. (*Br. Med. Jour.*, 27.11.48)

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The Treatment of Acute Dysenteries in Outlying Rural Dispensaries*

BRIJ-MOHAN BHARADWAJ,

Medical Officer, Padam Hospital, Jubbal, (Himachal Pradesh).

THE hair splitting specialist may feel greatly annoyed at the term 'Acute Dysenteries' used in the title of this paper for he is not satisfied even with a closer term as bacillary or amœbic dysentery; he is exact and nothing short of a more accurate description as bacillary dysentery of type Shiga etc. will satisfy him. But then he can avail of the help of an able pathologist who in turn has a well-equipped laboratory at his disposal, fitted for all bacteriological work. His patients are usually the few rich who can afford to consult him and pay for the bacteriological examinations, or those residing in places close to one of the few well-equipped hospitals in the larger cities of India. The majority of the people who are poor and living in the outlying villages have to seek the advice of a Medical Officer incharge of the nearest dispensary, who, more often than not, is seated in an ill-equipped dispensary with hardly enough medicines, leave alone a laboratory. It is for such a practitioner that this small paper is primarily intended. The heavy toll of human lives that is taken by this amenable disease is really a disgrace to our Medical Relief Department. In this small Province, where no regular statistics were maintained, I have found that acute dysenteries are responsible for at least 20% of the total deaths. The people of Himachal Pradesh, particularly of Jubbal, are very backward in all respects, and as a result are equally poor. They are of a very unhygienic temperament and this fact coupled with their emaciated physique make them an easy prey to this condition. The stools of a dysenteric patient are invariably exposed to flies; and as the patient is kept in the kitchen, because it is the warmest room in the house, these flies have an easy access to the food. This results in nearly all the members of the family, particularly the small children, getting the infection. The rate of mortality amongst children is the highest, the ratio being children: adult :: 3:1. A detailed account of the ætiology and pathology of the dysenteries will be found in any good textbook of medicine, and is therefore here omitted, it being beyond the purview of this paper. But for the purpose of this article the term acute dysentery may be defined as a syndrome presenting the following: any case suddenly developing colicky abdominal pain, with or without vomiting, urgent diarrhœa with flakes of mucus or naked eye blood and mucus in the stools and fever may be regarded as a case of acute dysentery. The well advanced cases show in addition marked dehydration, a cold clammy skin, dry tongue and anxious facies.

* Specially contributed to THE ANTISEPTIC

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Cramps in the calf-muscles and extreme exhaustion are also complained of. In spite of the fact that this condition takes a heavy toll every year the people in this part of the country do not yet realise the seriousness of this disease. For the first few days local remedies such as raw opium etc. are given and when the case is growing hopeless the Medical Officer is consulted. If intravenous injections are advised to combat the severe dehydration that is nearly always present in these advanced cases, treatment is stopped, and if unluckily, the child or the adult, as the case may be, is taken home is reported dead within a few days.

The Medical Officer in these outlying dispensaries has to make good use of his clinical sense in such cases. As far as is possible he should never fail to make a naked eye examination of the stools at least once daily. Though this will not help him in distinguishing with any amount of certainty whether the dysentery is of bacillary or the amoebic type, the consistency of the stools and the quantity

TABLE

Number of cases	Age group	Signs and symptoms	At commencement of treatment	After first 24 hours of treatment		2nd Day	
				A	B	A	B
50 control		(a) Temperature	103— 104.5°F	103— 104.5°F	101— 101.5°F	103— 104°F	99— 100°F
50 cases	18-35 yrs	(b) Blood and mucus	+++	+++	++	+++	+
		(c) No. of stools	15-30	15-30	7-15	16-30	5-0
		(d) Abd. discomfort	+++	+++	++	+++	+
		(e) Urinary output	5-10 oz.	5-10	15-20 oz.	5-10	15-20 oz.
		(f) Fluid intake	Sips only	20 oz.	60 oz.	20 oz.	60 oz.
		(g) Vomiting	++	++	+	++	—
		(h) General condition	Poor	Poor	+	Poor	++
		(i) Toxæmia	+++	+++	++	+++	+

Notes:—I (1) It was found too risky to keep children as controls, hence only adults were considered. (2) People of the same age and sex and condition only were paired. (3) As far as was possible only those cases were paired as were coming from the same village and taking identical diet. (4) Control cases were put on to routine treatment with the Cibazol and Entero-Vioform after the fourth day, in some cases earlier too, as they may have succumbed to the disease while under observation on imitation powders. (5) The fluid intake has been considered including the amount of fluids given parenterally. (6) With the intake of semi-solid diet the fluid intake was decreased. (7) The urinary output which was not significantly increased rose to normal after the fifth day.

II. The symbol A and B at the head of the column denotes the control group of cases on imitation powders and those Cibazol and Entero-Vioform respectively.

of blood and mucus, whether this is decreasing or not, will give some help in the treatment.

Adjuncts to actual treatment.—Dehydration must be combated, if possible, by fluids by mouth, if not then parenterally.

In cases that complain of very severe abdominal discomfort an injection of Morphine should not be withheld. The old methods of starvation and treating with Magnesium Sulphate etc., which are still being practised by some of my colleagues are to be condemned. On the other hand, I would strongly emphasise that semi-solid food such as mashed potatoes with curds and sugar or salt to taste, curds, and 'Dallia' and boiled rice water should not be withheld but rather be given *ad lib.* Fluids like warmed barley water, weak tea and Potus Imperialis should be given two hourly. The above procedure regarding diet may regularly be followed. I don't think that the strict dietary regimes advised in the textbooks would help

I

3rd day		4th day		5th day		6th day		7th day		Remarks (Observations)
A	B	A	B	A	B	A	B	A	B	
103— 105°F	98— 99°F	103— 105°F	97— 98°F		97— 98°F		97— 98°F		97— 98°F	(1) A worse B Marked improvement.
++	+	+++	—		—		—		—	(2) A— „ B „
16-30	2-6	16-30	1-3		1-2		—		—	(3) A— „ B „
+++	—	+++	—		—		—		—	(4) A— „ B „
4-10	25-30 oz.	4-10	30 oz.		35 oz.		40— 45 oz.		40 oz.— 45 oz.	(5) A— „ B „
15 oz.	50-60 oz.	15 oz.	40-60 oz.		40-60 oz.		40 oz.		40 oz.	(6) A— „ B „
++	—	+++	—		—		—		—	(7) A— „ B „
Worse	+++	Bad	+++		+++		+++		+++	(8) A— „ B „
++++	—	++++	—		—		—		—	(9) A— „ B „

III. Adult dose of the Sulphathiazole and Iodochlorhydroxyquinoline (brand of Cibazol and Entero-Vioform) was Cibazol 26 tablets, Entero-Vioform 14 tablets divided as stated elsewhere, over a period of 7 days.

IV. Doses for children and the aged were adjusted accordingly.

V. Any case that had a relapse was given another course. Relapses were negligible.

VI. After the fifth day the patients were constipated. No purgatives were given and the bowels moved after the third day. Fluid intake was increased.

VII. Powders containing Sod. Chloride etc. were administered to both the control and the other groups.

VIII. Most of the cases, particularly the young men and all women resumed their work after the fifth day.

more. Moreover, those predigested foods and the like recommended therein are not within the reach of the poor peasants.

Actual treatment with drugs.—I have not met with any appreciable number of cases out of the total of three hundred odd cases treated by me over a period of three years, who did not respond to the treatment recommended. Moreover, I found my patients made a quick recovery and relapses were few. *Cibazol brand of Sulphathiazole and Entero-Vioform brand of Iodochlorhydroxyquinoline were the main drugs used.*

A powder containing Sod. Bicarb gr. 20, Sod. Chloride gr. 5, Pulv. Ginger gr. 5 and Glucose gr. 30 was given with each dose of the Cibazol and Entero-Vioform tablets. Immediately on presentation the adult case is given 4 tab. Cibazol and 2 tabs. Entero-Vioform. Thereafter he is given 2 tab. Cibazol and one of Entero-Vioform two doses at six hourly intervals.

On the second, third and fourth day, the patient receives 4 tablets of Cibazol and two of Entero-Vioform divided over four

TABLE						
Number of Cases	Age group	Sex	Signs and Symptoms	Condition at commencement of treatment	After the first 24 hours	Second day
50	1—5 yrs.	M. 30	Temperature	102—103.5°F	101—102°F	100—100.5°F
			Blood and Mucus	+	+	+
		F. 20	No. of stools	12—25	10—15	6—8
			Abd. discomfort	+	+	—
30	6—10 yrs.	M. 18	Temperature	103—104.5°F	102—102.5°F	100—105.5°F
			Blood and Mucus	+	+	+
		F. 12	No. of stools	25—30	7—15	5—8
			Abd. discomfort	+	+	+
30	11—17 yrs.	M. 17	Temperature	103—104°F	102—102.5°F	99—100°F
			blood and mucus	+	+	+
		F. 13	No. of stools	15—30	7—15	5—9
			Abd. Discomfort	+	+	+
60	18—35 yrs.	M. 35	Temperature	103—104°F	102—102.5°F	99—100°F
			blood and mucus	+	+	+
		F. 25	No. of stools	15—30	7—15	5—8
			Abd. discomfort	+	+	+
30	36 and above	M. 16	Temperature	102—103°F	100—101°F	99—100°F
			blood and mucus	+	+	+
		F. 4	No. of stools	12—30	10—15	5—10
			Abd. discomfort	+	+	+

doses. On the fifth and sixth days he is given 3 tablets of Cibazol and 2 tablets of Entero-Vioform divided over three doses each day. After the sixth day treatment is discontinued as by this time all cases are cured.

Thus one adult receives for a complete cure, a total of 26 tablets of Cibazol and 14 tablets of Entero-Vioform.

The dosage for children was adjusted according to the age and condition of the patient at the time of attendance.

The results with this method of treatment have been very promising at my hands and I am sure that if this routine treatment is carried out more extensively it will greatly help to alleviate suffering of the poor dysenteric patient which vitally affects the economic life of this country. Although reports have appeared in the Medical Press from abroad regarding the newer Sulphonamide compounds said to be more efficacious for this disease, yet the prices of some of these products are prohibitive, to allow of their use in all

II

Third day	Fourth day	Fifth day	Sixth day	Seventh day	
99—100°F	98—99°F	97—98°F	97—98°F	97—98°F	Parenteral fluids used for only 6 small children. The rest took fluids by mouth.
2—7	1—4	1	—	1	
99—99.8°F	98—99°F	97—98°F	97—98°F	97—98°F	
3—4	1—2	—	—	One	
92—99°F	97—98°F	97—98°F	97—98°F	97—98°F	Bowels moved themselves after the seventh day.
2—6	1—3	—	—	—	
98—99°F	97—98°F	97—98°F	97—98°F	97—98°F	
3—4	1—2	—	—	One	
98—99°F	98—99°F	97—98°F	96—97°F	97—98°F	Only one fussy case was given a glycerine enema to move his bowels. Morphine used for only 5 old people for abdominal discomfort.
2—7	1—4	1	1	1	
—	—	—	—	—	
—	—	—	—	—	

dispensaries with the danger of accompanying side reactions on account of their toxicity.

The combination of a Sulphonamide with an amoebicide will effect a cure in nearly all cases, as it has been found by various workers on the subject that cases of mixed bacillary and amoebic dysentery are not infrequently met with.

In spite of the fact that more than 300 cases of all ages have been treated by this method, it is regretted that more detailed investigations could not be undertaken, specially with the help of control cases, as adequate facilities for hospitalization were not available. Anyhow even in these adverse circumstances, observations with control cases treated on imitation powders were made and the result, as tabulated elsewhere, will show that this combination is effective. The powders described above containing Sod. Chloride etc. I have found greatly help in repleting the Sod. Chloride loss from the tissues, and thus lessening the cramps so often complained of. Table II appended shows the reaction of this combination of Cibazol and Entero-Vioform in cases of acute dysentery.

Summary.—An investigation was undertaken to find the ideal treatment or one nearest to it for the treatment of acute dysenteries in outlying dispensaries, where adequate facilities for detailed bacteriological investigations were not available. It was found that Cibazol and Entero-Vioform were the drugs which approached the ideal. This method has been found to be cheap, rapid and efficacious. The full treatment costs hardly Rs. 3 per head for an adult. The patient is well and can attend to his normal work within a week. Relapses are negligible. Stress has been laid on the free administration of semi-solid foods and liquids *ad lib*.

Details of results with and without controls in Tables I and II have been appended (*vide page 358 to 361*).

Results have been based on data patiently collected since 1946.

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P. O. Jubbal, (Simla Hills).

Application of Infra-red Rays in the Treatment of Certain Diseases*

HARENDRA NATH BAGCHI, M.B.B.M.S. (B.),
Calcutta.

WE learn from Newton's discovery how a beam of sun-light coming through a narrow vertical slit and allowed to pass through a glass prism placed with its refractive edge parallel to the slit the emergent beam when received on a vertical screen is seen drawn out into a long coloured band which shows different colours of the rainbow, "VIBGYOR". We also learn how light is always partly accompanied by heat and can bring out certain chemical reactions and that the solar light possesses: (1) Luminous effect; (2) Heating effect; and (3) Chemical effect.

The brightness of the solar spectrum is not uniform throughout. It is maximum in the yellow and decreases gradually on both sides. The heat radiation from different parts of the solar spectrum is not the same. There is a little rise of temperature towards the blue region but the effect increases as we move towards the red and attains the maximum value at a little distance beyond red and is known as the infra-red part of the spectrum. The infra-red portion has a very feeble chemical effect but the heating effect is prominent being maximum a little beyond the visible red. The infra-red region covers a range of wave length from 7,800 A.U. to about 0.04 cm. (1 AU=10⁻⁸ cm. = $\frac{1}{10,000,000,000}$ c.m.)

The infra-red rays occupy a position below the red end of the visible spectrum. They are invisible but their presence can be detected by a thermometer.

The source of these for therapeutic purposes consists of a high candle power glass electric light bulb partially filled with Nitrogen and mounted on a suitable stand with a reflector. The radiations consist of a mixture of visible luminous rays and of the invisible infra-red heat rays.

Action:—Infra-red radiation is absorbed in the sub-epidermal capillaries, the superficial muscles and fascial planes producing heat which causes local vaso-dilatation and erythema within about 10 minutes, relieving congestion of the deeper parts and thereby exerts a marked sedative effect. The temperature of the skin is raised with stimulation of metabolism, moderate increase of red blood corpuscles and diminution of white blood corpuscles.

Therapeutics:—Locally applied in fibrositis, neuralgia, neuritis, myositis, muscular wasting, rheumatoid and gouty arthritis, abscesses, whitlows, dry pleurisy, gingivitis, early pyorrhoea alveolaris, leukæmias.

*Specially contributed to THE ANTISEPTIC.

Actinotherapy denotes treatment by rays. These rays are forms of electromagnetic energy transmitted at uniform speed (186,000 miles per second).

Units of wave-length:—1 AU (Angstrom unit) $\frac{1 \text{ m.m.}}{10,000,000}$ (10⁻⁷ millimeter Angstrom unit used in Britain).

Associated with the wave length is the energy quantum of radiation by the equation $q = hn$, in which q is the unit of radiation, h is constant (6.55×10^{-27} erg sec), n is the frequency of vibration. This equation means that diminishing wave length means increasing energy quanta i.e., shorter ultra-violet rays have greater photochemical activity. Radiant energy can be measured quantitatively in physical units such as ergs or watts. For practical use in actinotherapy is to estimate dosage in terms of biological reactions to certain short wave lengths. To estimate the radiation received from a source either by physical methods (spectroscopy etc.) by biological estimation (erythema reaction, bactericidal effects) or by a combination of the two methods.

Filters.—In certain therapeutic applications the need arises for filters which will transmit certain portions of spectrum.

Filter	Uses	Spectrum limits (A.U.) Short Long	Filter	Uses	Spectrum limits (A.U.)
Sollux Red 2 m.m.	Red and infra-red radiation	6000	Sollux blue	Blue and infra-red radiation	3010

Radiations used in actinotherapy:—Wave lengths (A.U.), frequency; Notes:

Infra-red (near region)	Frequency	15000	2×10^{14} /Secs.	Rays of deep penetration.
Do. (long rays)	Do.	40000	75×10^{13} /Sec.	Little penetrating effect.

Physiological effects:—Infra-red rays produce no notable changes in the blood chemistry, they produce and maintain powerful penetrating hyperaemia. The aim of applying infra-red rays is to produce hyperaemia and local rise of temperature deeply within the subcutaneous tissues of the irradiated area.

The tissues are penetrated by rays of 11000 A.U. The visible rays increase in penetration as they increase in wave-length. The visible red rays and the short infra-red upto 11,000 A.U. have the greatest penetration.

The most effective sources to produce deep penetrating hyperaemia are therefore those of infra-red rays shorter than 15,000 A.U.

Effects on the skin:—The total reaction of the skin to actinic rays is the latent erythema which develops after a few hours. The latent period varies inversely with the exposure and may range from 1 to 8 hours. The erythema is associated with increase of temperature and emission of heat by the skin.

Actinic irradiation takes effect primarily on the skin functions which in time sets up secondary reactions in the organisms as a

whole. The treatment of the whole body surface with infra-red rays is administered to promote circulation and excretion by the skin. It is valuable in rheumatism.

Records:—The time, distance and area of each irradiation should be noted and the resulting reaction entered.

The skin should be quite clean. A hot bath will cleanse the skin and improve its circulation thus promoting the actinic reaction. Contra-indications of infra-red bath in patients with low leucocyte counts or with markedly low blood pressure.

13, Baghbazar Street,
Calcutta.

Cases and Comments

Streptomycin

M. P. RANJAN, L.M.P.,

Formerly House Physician and Surgeon, D. M. S. Hospital,
Hospital Road, Laheria-Sarai.

Definition.—It is a basic anti-bacterial substance obtained as the surface salt from actinomyces griseus culture fluid.

Historical.—Anti-biotic substance announced in January '44 by Schatz, Bugie and Waksman. Experiments are still being carried.

Pharmacology.—1. *After hypodermic or intramuscular injection:*—Readily absorbed by tissues and reaches general circulation. In blood serum, the peak of concentration is reached within 1 to 2 hours and gradually falls with about 2/3 of the total dose being excreted during the first 24 hours. The level of the drug in serum is proportionate to the amount administered.

The excretion is less rapid than Penicillin. 40 to 80% of the drug is excreted in the urine, hence a daily dose of 2 million units of the drug maintains a concentration of 1330 units per c.c.

The drug does not diffuse into C. S. fluid, diffuse into peritoneal fluid but not into empyæmic cavities.

2. *After intrathecal administration:*—To ensure adequate C. S. fluid concentration, supplementary I. T. injection at 24 hours intervals is necessary. Repeated I. M. injection in cases of meningitis (to inhibit bacterial growth) may cause it to appear in C. S. fluid when given in sufficient amount.

3. *After oral administration:*—Owing to slight absorption from the gastro-intestinal tract, oral administration of the drug is not suitable for treating systemic infection. As the drug is not destroyed by intestinal contents, it may be administered orally to

eliminate certain Streptomycin susceptible organisms. Further, although it appears in bile, little is found in faeces after parenteral injection.

1. *By inhalation*:—No effective absorption into blood stream.

Manufacture.—1. *Culture*:—It is derived from culture of an actinomyceae of the soil (*Streptomyces griseus*) by a process of extraction and purification which yields a highly soluble, hygroscopic powder, usually cream coloured or light tan in appearance. A basic substance prepared either as Streptomycin Hydrochloride or Sulphate. Although more stable than Penicillin, it should be refrigerated especially when in solution form which should always be prepared fresh.

2. *Units of potency*:—One microgram of pure Streptomycin base is approximately equivalent to the original unit of Waksman. Hence, 1 mg. of Streptomycin is equal to 1,000 of 'S' units and 1 gm. of Streptomycin equal to 1,000,000 'S' units.

3. *Organic base*:—Chemical constituents of the drug is unknown—soluble in water and dilute acids—more stable in dry state and in aqueous solution than Penicillin—the solution can be easily sterilised by heat without any loss of activity.

Storage.—To prevent deterioration, it should be stored below 15°C (59°F). The prepared solution can be kept in refrigerator for one week without any loss of potency.

Indications.—1. Highly recommended: Tularæmia.

2. Effective in (a) *B. coli*; (b) tuberculosis; and (c) dysentery.

3. Experimental stage:—(a) Miliary tuberculosis; (b) tuberculous meningitis; (c) urinary infections; and (d) typhoid fever (when given both orally and parenterally 1 to 4 million units daily).

Mode of preparation.—1. *Subcutaneous*: 1 G. dissolved in 10 c.c. of Pyrogen free re-distilled water.

2. *Intramuscular*: 1 Gram. (1,000 mg.) dissolved in 8 c.c. of sterile Normal Saline or Pyrogen free water—the solution containing 125 mg. per c.c.

3. *Intravenous*: 1 G. dissolved in 10 to 20 c.c. of Saline with 5% solution of Dextrose.

4. *Intrathecal*: 25 to 50 mg. dissolved in 5 to 10 c.c. Saline.

Method of administration.—1. *Subcutaneously*: 125 to 250 mg. every 3 to 4 hours.

2. *Intramuscularly*: 125 to 250 mg. every 3 to 4 hours or six hourly, provided a daily dose of 1 to 3 gm. (1,000 to 3,000 mg.) is supplied.

For continuous injection: The total daily dose of 1 to 3 G. may be dissolved in 500 to 1,000 c.c. of Normal Saline with the rate of flow so regulated as to deliver the entire amount in 24 hours.

2 or 3 c.c. of a solution containing 100 mg. per c.c. every 4 hourly making a total dose 1.2 to 1.8 gm. for each 24 hours.

3. *Intravenously*:—No advantage over subcutaneous or intramuscular injection, hence not recommended. Concentrated solution must not be used on account of the potent impurities. The drug is excreted less rapidly than Penicillin but when given I.V. the dose should be 4 to 6 hourly.

May be given by continuous drip or I.V. infusion in very dilute solution—the daily dose being dissolved in 1 or 2 litres of Isotonic Saline and 5% Glucose solution: Addition of 100 mg. of Heparin to each litre of solution to be used minimises the possibilities of venous thrombosis.

4. *Intrathecaly*:—In meningitis caused by Streptomycin susceptible organisms, I. T. injection at 24 hours intervals should supplement the systemic injection. 25 to 50 mg. in solution injected directly into the spinal canal. In children, total amount should not exceed 10 mg. per day.

5. *Orally*:—When not necessary to obtain a systemic effect.

6. *Intrapleurally or intraperitoneally* may be used but the concentration should not exceed 50 mg. per c.c.

7. *Topically*:—To wounds and conjunctiva—the concentration should be 2.5 to 5 mg. per c.c.

Dosage.—1 G. at 4 hourly intervals I. M. course—100 days. Minimum daily dose—1 or 2 gms. (1,000,000 to 2,000,000) 'S' units.

Toxicity.—(a) Nausea, vomiting. (b) Vertigo: faintness, headache. (c) Flushing of skin (face). (d) Ataxic gait. (e) Abrupt fall in B. P. (f) Irritation to the kidney. (g) Anaphylactic reaction. (h) Local irritation at the site of injection. (i) Transient deafness: Reduced labyrinthine response to caloric test.

Clinical uses.—1. *Tularæmia*:—Quick response, therapeutic use confirms diagnosis: I. M. injection of 30,000 units every 4 hours for 5 days brings down fever, helps to subside the buboes and the disappearance of mental symptoms within 48 hours.

2. *Tuberculosis*:—Most sensitive organism: Effective both *in vitro* and *vivo*: Exerts a suppressive effect in some forms of clinical types: Inhibits the activity but does not kill the bacilli.

3. *Bacteraemia*:—When the infective organism is susceptible to the drug: Due to gram negative bacilli: Severe infection of genito-urinary tract.

4. *Infection of genito-urinary tract*:—Before instituting the therapy, every effort should be made to ensure adequate drainage of the urinary tract, disposal of indwelling catheter, close supra-pubic cystotomy, incisions or nephrostomy. Give 1 to 2 mg. or more daily.

5. *Tuberculosis*: (a) Miliary no improvement. (b) Renal or cutaneous encouraging. (c) Laryngeal considerable benefit. 1 million units daily for 6 days.

6. *Influenzal meningitis*

7. *Brucellosis*.

8. *Encouraging results in*—(a) *B. coli*. (b) *B. pyocyaneus*. (c) *B. proteus morganii*.

9. *Temporary benefit in*—(a) Pertussis. (b) Bronchiectasis. (c) Ozaena.

10. *Experimental stage*:—(a) Typhoid fever. (b) Bacillary dysentery. (c) Plague. (d) Cholera. (e) Liver abscess.

Drawback.—It creates drug resistance to T.B. does not kill the tubercle bacilli in lungs but only inhibits the bacilli and thus allows time to healing by fibrosis and hyalinisation.

Conclusion.—Great difficulty is experienced in the use of the drug on account of its manufacture only in U.S.A. and that too in small quantities. The Mayo Clinic computes the American product at about 500 mg. each month. The drug is still in experimental stage. Still more positive evidence is forthcoming as to its good effects even in miliary tuberculosis or tuberculous meningitis. On account of low toxicity, no doubt, it will one day become a very useful chemo-therapeutic agent when available commercially.

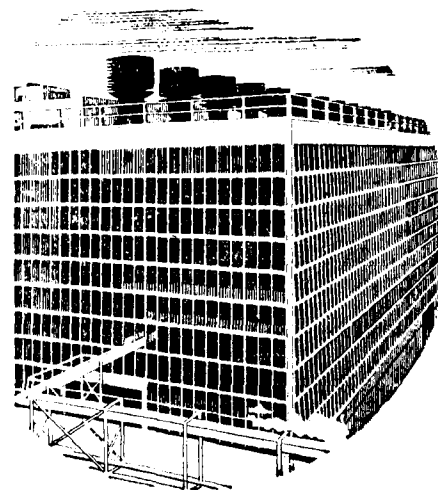
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2. Editorial—I. M. Journal, Vol. 41, No. 10, page 193.
3. Wilson, A.—Medical Annual, 1947, pp. 331-34.
4. Literature by Upjohn & Co., U.S.A.

STREPTOMYCIN is effective in the treatment of urinary tract infections, bacteremia and meningitis due to susceptible strains of *E. coli*, *B. proteus*, *A. aerogenes*, *Ps. aeruginosa* and *K. pneumoniae*; also effective in tularemia and in all *H. influenzae* infections.

STREPTOMYCIN is helpful in the treatment of the following diseases, but its exact position must still be determined: Peritonitis; chronic pulmonary infections, empyema, liver abscesses, and cholangitis, when caused by susceptible gram-negative organisms; *K. pneumoniae* pneumonia; endocarditis caused by penicillin-resistant but streptomycin-susceptible organisms; tuberculosis.

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STREPTOMYCIN HYDROCHLORIDE SQUIBB

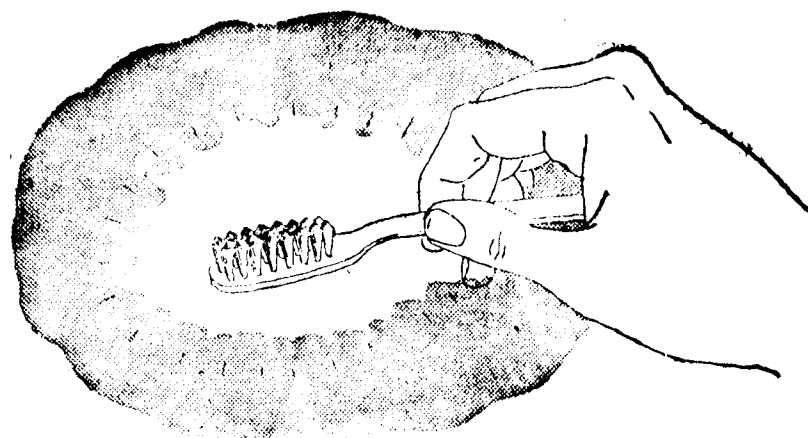
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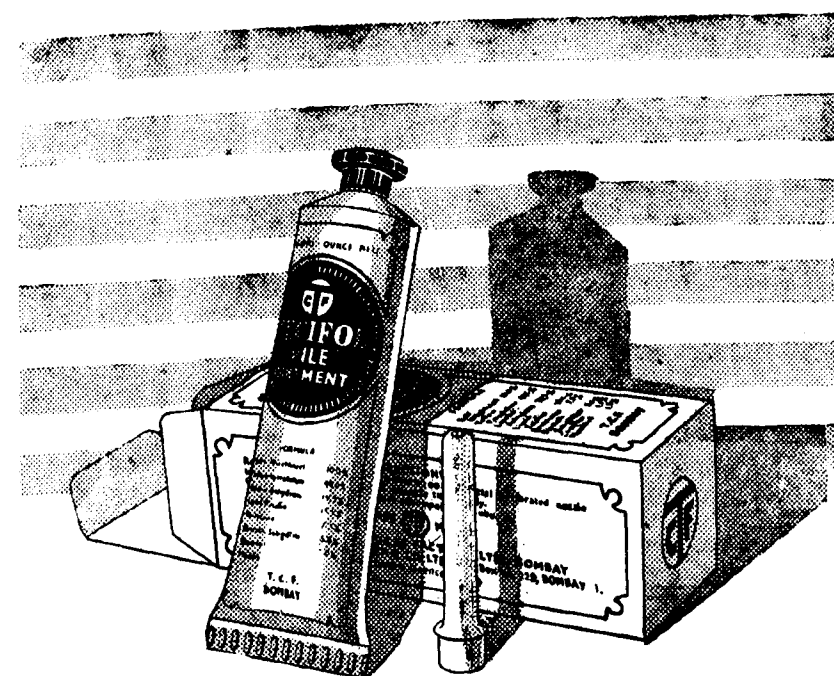
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Carbolic Acid in Eye Diseases

NEM BAHADUR, I.D.S.C., D.O.N.,
Eye Specialist and Dental Surgeon, Nepal.

CARBOLIC Acid is a crystalline compound, obtained from distillation of a thick, black, semi-liquid, known by the name of Coal-tar. It is a very powerful antiseptic and disinfectant. It is a very poisonous and oily colourless liquid with burning taste.

Pure carbolic acid has been used with good results in eye diseases such as corneal ulcer, hypopyon ulcer, pannus, phlyctenular conjunctivitis, iris prolapse etc.

Carbolic acid cannot be instilled into the eyes under any circumstances as it will burn cornea as well conjunctiva. It is however applied with great precaution to circumscribed area with the help of a small pointed match stick in the form of cauterization. But the Cocain solution is to be instilled into the eyes before it is applied.

During the last 12 years I am using this acid in my eye clinic and find favourable results. I shall now give records of a few severe cases which I have come across:

Corneal ulcer.—A girl of 14 years was suffering from corneal ulcer in her right eye. She had been treated properly but there was no improvement and the ulcer became bigger and perforating. The patient, being of afraid of losing her eye sight, came to me for consultation. On examination I found a deep and infected ulcer in the centre of cornea. There was redness, photophobia, lacrimation and œdema of the lids. She complained of severe pain at night. Her vision was very poor with perception of light only.

Treatment.—On the very first day I cauterized the ulcer with pure Carbolic Acid with the help of a small pointed cotton roll and applied bandage. After 48 hours she had no pain at all and the redness became pinkish and less watering. The same treatment was continued for a few days along with Atropine and the antiseptic drops. After 15 days the patient became free from all troubles except some opacities in the cornea.

Hypopyon ulcer.—An elderly man of about 50 years was badly suffering from hypopyon ulcer in both the eyes. He has been treated with all available medicines including Penicillin injection. There was no improvement, but on the contrary the condition became worse and the case was left as hopeless. The patient was brought to me for consultation. On examination I found deep corneal ulcer covering the whole cornea and pus in the anterior chamber in both the eyes. There was severe pain and the patient could not open the eyes properly. The vision was very poor, not even finger movement, but only perception of light was present.

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Treatment.—As he had already been treated with all available medicines I could not find any other drug except Acid Carbolie, so I cauterized the ulcer with this acid in the form of cross mark from one end to the other of cornea and applied bandage. I also gave enema to clear his bowel. On the next day there was no improvement but there was a check to the process of deterioration. The same treatment alternately along with Atropine drops was continued and on the fourth day the patient had no pain and could open the eyes for a short while. On the sixth day I could see the pupil and the iris in bright illumination through some portion of cornea, and after ten days the patient could see the window bars of his room. There was no pus in the anterior chamber and hence I stopped cauterization with Carbolie Acid. Atropine drops were continued and the patient was discharged after a month with a big opacity in the cornea.

Iris prolapse.—A girl of 16 years was suffering from iris prolapse in her right eye. She had corneal ulcer but due to negligence the ulcer became perforated and some portion of iris came out of the wound. She was advised cutting of the prolapsed iris in some local hospital but the patient, being afraid of operation, came to me. I applied pure Carbolie Acid on the prolapsed iris with the help of a small pointed cotton roll and bandaged. The same treatment was continued for three weeks and the prolapse subsided and the wound healed up.

Similarly other eye diseases such as pannus, phleetenular conjunctivitis etc. are cured by the pure carbolie acid.

47, Joodha Road, (Near Fire Brigade),
Katmandu, Nepal.

High Blood Pressure and Pulse Rate as Manifestation of Hyperglycemia

Coachman asserts that patients with hyperglycemia do not always show glycosuria because of a high renal threshold, which prevents the sugar from being passed from the blood into the urine. This repeated retention produces headaches, photophobia, lacrimation, dizziness, increased post-nasal discharge, paresthesias, generalized fatigue, transient scotomas and diplopia, which may progress to nausea and vomiting. Erroneous diagnosis of Meniere's syndrome, "sinus trouble," "loss of equilibrium" and allergic manifestations are often made. Many of these patients have no sugar in the urine and have normal fasting blood sugars, and so the existence of hyperglycemia is often missed. The two hour dextrose tolerance test, however, will reveal hyperglycemia. Elevated blood pressure and fast pulse rate in the presence of a normal warrant doing two hour dextrose tolerance tests routinely in these cases. Overweight, short neck, stout persons predominate in this group, and cataracts are so frequently seen that hyperglycemia is suspected of being a contributing cause. Latent vertical phoria is found in persons with elevated blood pressure and fast pulse. Latent vertical sion of the vertical correction in their lenses, the dizziness, photophobia, lacrimation and asthenopic symptoms are further relieved, even after control of the blood sugar. Latent vertical phoria should enter in the differential diagnosis in suspected cases of Meniere's syndrome and in "sinus" or "sick" headaches. — *Oklahoma State Medical Assn. Jour.*, Oklahoma City, 41: 369-406 (Sept.) 1948. — J.A.M.A.

Two Interesting Cases

M. R. BAIL, M.B., B.S.,

Ex Hony. Asst. Surgeon, Ear, Nose & Throat Dept.,
Government General Hospital, Madras.

CASE I.—A middle aged villager came with a history of acute nasal obstruction of a few days' duration. On anterior rhinoscopy, nothing could be detected to explain for his distress. By posterior rhinoscopy, a huge growth was found in the nasopharynx. It was so high, that one could not get a glimpse of it, even by pressing the soft palate upwards with a probe. On palpating with the tip of the right forefinger, behind and above the soft palate, the mass was cheesy at first, and hard inside, with a very foul smell. A tonsil volsellum was gently passed upwards, behind and above the soft palate, guided by the left forefinger. After obtaining a good grip with some difficulty, gentle traction was applied downwards and forwards, just to have an idea of the nature of the tumour—the soft palate being held above by a probe all the while. A few minutes later, a huge dirty mass of the size of an average bael fruit, slowly began to make its appearance downwards. Slight further traction brought the anterior one third of a middle sized river fish, with sudden relief to the operator as well as the operated. It was a great surprise for the patient, who happened to belong to an orthodox South Indian class.

Evidently, the gentleman must have swam or dived with his mouth wide open, or would have gulped some bouts of water, when the foolish fish would have got in. Finding itself helpless, and the two orifices downwards being too small for that size of the fish, the desperate creature would have found the line of least resistance, and got impacted finally in the choanal orifice. What happened to the posterior end of this strange foreign body, is a mystery to me, as the party was not in a position to believe his own story. It must have disintegrated and a part of it at least must have polluted his holy stomach. However, there was neither necessity for any further treatment, nor the patient appeared subsequently for any follow up, as is usual with our people.

CASE II.—At about 10 p.m. a Brahmin gentleman came to me with a sudden severe pain in his ear. On examination with an aural speculum, nothing could be found out, except a waxy looking mass, close to the drum. It was softened with Peroxide of Hydrogen and syringed several times. But, the mass would not budge. Then an ear scoop was employed. That too was of no avail. Though the use of forceps to remove foreign bodies from the ear is rather harmful, I could not withhold the temptation, as I saw a few long bristle-like structures in the external meatus, converging towards the main mass, deep inside. On gentle traction, I got a deeper and better grip of the mass, till the whole body of a pretty middle-sized brown

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cocroach came out with a jerk, to our utter astonishment. The meatus was dried and no injury could be seen anywhere. It was touched with Glycerine Carbolic 5%. The patient was immensely pleased, and he did not complain of any subsequent trouble.

"Srinivas," 32, Mukhernallamuthu Chetty St.,
G.T., Madras.

A Case of Neuromyelitis Optica (Devic's Disease) with Recovery of Useful Vision

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Ophthalmic Surgeon,

AND

B. SAHAI, L.M.P.,
I c, Medical Ward, Victoria Hospital, Bharatpur, (Rajputana).

DURING recent years increased attention has been directed to the large and important group of diseases of the nervous system which possess as a common pathological feature foci in which the myelin sheaths of the nerve fibres are destroyed and hence these affections are labelled as demyelinating diseases of the nervous system. These diseases are of both interest and importance to the ophthalmologist as the demyelinating process which predominantly attacks the white matter has a predilection for the visual fibres and the visual cortex. Below is recorded a case which is probably the first of its kind noted by us in this hospital to be a typical case of neuromyelitis optica with recovery of useful vision which the patient retained up to his last.

Case report.—Uttam Chand, aged 32, Hindu male, was admitted in the medical ward on 12-6-'48 with history of loss of power in both lower limbs with creeping numbness extending from feet upwards. The left leg was more involved than the right. On the next day of admission, i.e., on 13-6-'48, the patient complained of blindness in both eyes. He could hardly recognize any body although hand movements could be perceived when moved quite close to his eyes. By the evening of 13-6-'48 the vision was totally gone in both the eyes. Side by side with these changes the anaesthesia and loss of movements were spreading up speedily and by the morning of 14th June, 1949 had almost reached the epigastrium with retention of urine and faeces. The patient when examined on 14th June, 1948 morning was lying prostrate in bed quite still. He could hardly whisper but was able to take the milk feeds.

Examination.—We made a thorough examination of the patient on the morning of 14th June, 1948. There was complete loss of sensation both superficial and deep from feet right up to the

umbilicus on both sides. All superficial and deep reflexes were absent in the involved area with complete loss of motor power in the lower limbs. There was retention of both urine and faeces. The patient retained his consciousness and average intelligence.

Eyes.—Both pupils were widely dilated, did not react to light at all. Media of both eyes clear, vessels of normal calibre. The patient could not even perceive the light of a hand torch.

Investigations.—(1) Blood tested for Khan's on 15-6-'48 was found negative.

(2) Urine examination showed pale yellow. Specific gravity 1019. No albumin, no sugar, a few epithelial cells present.

(3) Lumbar puncture on 16-6-'48. About 5 c.c. was drawn not under pressure. Laboratory report revealed slightly turbid fluid, few pus cells, no organisms found.

(4) Blood picture :—Hb 75%. Total red cell count 3,25,000 per cmm. No abnormality in blood cells. Leucocytes 8,000 per cmm; Differential count :—Poly 75%; Mono 4%; Lympho 19%; and Eosinophil 2%.

Progress.—His eyes were examined on 17-6-'48. Left eye upper lid swollen, marked oedema of bulbar conjunctiva.

Ophthalmoscopically :—Right eye :—Disc edges hazy with exudation on it hiding the course of vessels. Veins tortuous and congested. Pupil widely dilated does not react to light or accommodation. Left eye :—Condition same as of right eye but less marked. No vision in both eyes.

Examination of eye on 21-6-'48 :—Clearing of exudate on both optic discs with beginning pallor.

11-7-'48. Patient could perceive light and dark. Both pupils did react sluggishly in bright oblique illumination. Fundus picture same as on 21-6-'48.

28-7-'48. Both eyes :—Pupils active, react briskly to light both directly and consensually.

Vision R.E.—Counting fingers 1 1/2 metre.

L.E.—Counting fingers at 2 metres.

Ophthalmoscopically :—Both discs were found pale now, vessels appear of normal calibre, disc edges clearly seen. Pallor of disc marked on temporal side.

10-8-'48. Optic discs both eyes pale white with clean edges. Right optic disc was more white than the left vessels of normal calibre.

Pupils Size R.E.—3.5 mm. L.E.—3.5 mm.

Pupils reacted to light and accommodation quite briskly.

Vision R.E.—Counts fingers at 3 metres.

L.E.— " " " 6 metres.

2-1-49. Revealed almost white optic discs, arteries and veins of normal appearance. Vision was still improving.

R.E.—Counts fingers at 6 metres. L.E.—6/60.

Termination.—The patient was being treated on the lines of any other case of paraplegia. Urine had to be drawn regularly by catheterisation; bowels had to be cleared by enemias every alternate day.

The patient gradually went downhill, developed extensive bed sores spreading from buttocks down to calves on both sides. He developed septic cystitis, pyelonephritis and died on 15-2-49.

He retained useful vision upto his last. The patient being of below average intelligence, the field of vision in each could not be tested accurately. It appeared restricted when tested roughly.

Summary.—This connection between a bilateral optic neuritis and myelitis as in the above case was first noted by Clifford Allbert (1870) and Erb (1879) and was described as a clinical entity by Devic (1894) but the disease is rare. Goulden (1914) was able to find only 52 cases in the literature, a number brought upto 70 by Beck (1927) and since then, according to Duke Elder, about 50 cases have been reported.

In the above case myelitis appeared before optic neuritis and both eyes were affected almost simultaneously. After an interval of about two weeks the vision gradually returned and the patient retained useful vision upto his last.

The above case is reported due to its extreme rarity. We shall be glad to know further about its occurrence in other hospitals, and the result of the cases from any of your readers.

We are highly indebted to late Dr. Kamal Nain, L.T.M. and Dr. S. N. Chopra, D.P.H., Clinical Pathologist, Victoria Hospital, for carrying out the various laboratory tests for us in the above case.

Nitrogen Mustards

Besancon *et al* describe their findings in the treatment of 40 cases of different blood diseases with Nitrogen Mustard. The toxicity of this group of drugs for the epithelium of the digestive tract, the gonads and the bone marrow and the small margin between the effective and toxic dose make it quite essential to treat only the right type of case and to proceed with the greatest caution. In their opinion it should not be tried in anything but malignant conditions like Hodgkin's disease.

This drug can be regarded only as a palliative and never a cure. Severe anaemia, generalized bone pain and the terminal stages are contra-indications to the use of the drugs.

They insist that the drug should not be given unless 2 months have elapsed since the last session of X-ray therapy and that X-ray therapy should not be given immediately after treatment with Nitrogen Mustards.—(*Bull. Soc. Med. Hosp. Paris, Abst. World. Med.*, 1948, No. 2154-2155.)

T.N.S.R.

A Case of Hypotension

DR. D. R. PRABHAKER,

M.O., I/c., Civil Dispensary, Paraspur, Dist. Gonda, (U.P.)

A patient, G. L., 56, H., M., visited the outpatient department for following complaints on 6-8-'48:

1. Constriction sort of pain in the chest and back, duration 2 months.
2. Weakness and inability to work, duration 6 months.
3. Loss of appetite, duration 2 months.

History of present illness:—For the last 6 months patient is feeling weak and unable to do any hard work, on which account remains mentally worried. For the last 2 months, he is feeling a sort of constricting pain in the chest and back, as also loss of appetite and disinclination towards work.

Past history:—Nothing particular. He is not an addict to intoxicants.

General examination:—Good build, but is moderately pale. Teeth all absent (extracted due to pyorrhœa). Bowels regular, with occasional passing of thread worms. Pulse 52 p.m. He is a labourer by profession.

Direct cross questions, however, revealed valuable information. He admitted attacks of palpitation off and on, especially noticeable at night, otherwise during day, only after some sort of exertion.

Circulatory system:—Pulse 52 p.m., not exactly regular, volume and tension low. No displacement of the apex beat. Both heart sounds appeared to be normal except that these sounds were not exactly regular, as was the pulse beat.

Blood pressure was remarkably low, i.e., 80/55 mm. of Mercury N.A.D. in respiratory and digestive system, only there was a little fullness of abdomen, for which the patient used to take a little 'Hing' (Asafoetida) to expel the flatus, which had a favourable effect on the frequency of palpitation attacks. After a review of the above he was labelled as a case of 'hypertension' or low blood pressure.

By blood pressure is meant, the pressure that the blood exerts against the walls of the vessels in which it is contained, and this depends upon:—

- (a) The force of the contraction of the left ventricle.
- (b) The peripheral resistance.
- (c) The elasticity of the walls of the arteries.
- (d) The volume of the blood.
- (e) The viscosity of the blood.

The arterial pressure that synchronises with the systole of the ventricle is called the systolic, and that which synchronises with the diastole is called the diastolic blood pressure.

Causes of low blood pressure, especially systolic.

1. Lowered myocardial efficiency as in acute febrile conditions like diphtheria, influenza and coronary thrombosis.

2. Loss of peripheral resistance from relaxation of the arterioles as in Kala-azar, T.B. cancer, acute febrile conditions as pneumonia, typhoid or diphtheria.

3. Lowered viscosity or volume of the blood as in anaemias from any cause or loss of fluids as in severe vomiting and purging.

4. Paralysis of the vasomotor system as in—

(a) Bowel diseases as in cholera, bacillary dysentery, ptomaine poisoning, also algid malaria.

(b) Addison's disease.

(c) Vagal overaction as in fainting attacks. Splanchnic stasis is frequently associated with lowered vasomotor tone.

Causes of low diastolic pressure:—(1) High grade vasodilatation as in aortic regurgitation, arterio-venous aneurysm, patent ductus arteriosus, thyrotoxicosis, neuro-circulatory asthenia including effort syndrome. In spite of all this, in some individuals, low B. P. may be normal or say habitual. In this particular case, anaemia, and general malnutrition coupled with old focal dental sepsis may be the probable cause. To this may be added his mental worry and anxiety to support his family.

*Treatment and progress report:—*Mist. Iron Tonic 3i thrice daily and Cal. Lactate gr. xv thrice daily advised plenty of nourishing food intake. Subsequently "Bellergeral" tablets (Sandoz) were started to stimulate his nervous system, one tablet thrice daily from 20-8-'48, when again reading was 80/55. On 31-8-'48 it was 85/60. On 11-9-'48 it was 100/70. At this stage he stopped the tablets treatment, as he could not afford it, and on 3-10-'48 it again went down to 90/60. He was, however, now persuaded to get Belladonal tab. (Sandoz), one tablet b.d. which he did, and B.P. on 9-10-'48 was again noticed at 100/70. On 16-10-'48 it was 110/80. Pulse rate upto the time of writing (7, Feb. 1949). The blood pressure is stationary at 105/75, and he is keeping fit.

Splenectomy in Kala-azar

Air Vice-Marshal Dr. Norton and Dr. Cooke of the R.A.F. Institute of Pathology and Tropical Medicine, record their successful results obtained in 3 drug resistant cases of kala-azar by combining splenectomy with specific therapy (pentavalent antimony preparations). Splenectomy was first performed in 1912 by Makkas in Athens, and subsequently was in vogue for some years; but it has come to be regarded and entirely contraindicated in the treatment of Kala-azar.

After 16 months of intensive antimony treatment, without any benefit, splenectomy was performed with success and the patient is well and on full duty. Splenectomy is, therefore, indicated in selected cases, resistant to drugs and the operation should be followed by a course of chemotherapy, under strict medical control.—(*The Lancet*, 2, 1948, pp. 920-923.)

T.N.S.R.

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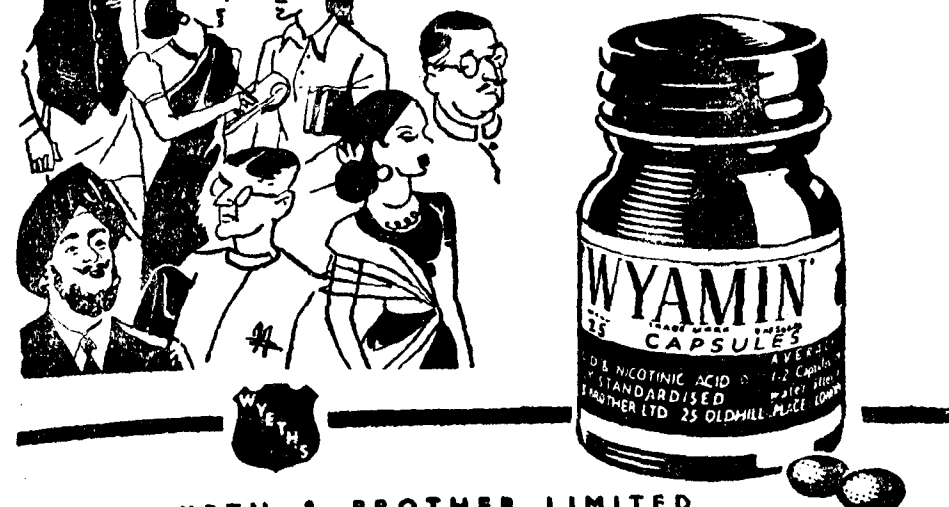
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B. C. G. Vaccination

IN India the incidence of tuberculosis is very large, the mortality rate very high, and the prospect of the spread of the disease very great. The causes for this most unsatisfactory state of things are many, viz., poor housing conditions, the low standard of living, lack of proper nutrition, etc., etc. We have therefore all along been emphasising that if positive health which is the birthright of every citizen in this country is to be assured, the Government should give top priority to schemes for the improvement of public health, sanitation and the like. But in a country where these essentials have been neglected for far too long a period, it is not possible to work out miracles and bring about changes overnight. Even with the best will in the world, it is not possible to bring about rapid reformation; the huge cost involved being a stumbling block in the way. As stated by Dr. P. V. BENJAMIN, Tuberculosis Adviser to the Government of India, at the inauguration of B.C.G. Vaccination in Madras, a sum of 500 crores will be required to provide the minimum standards of anti-tuberculosis measures, such as clinics, hospitals and sanatoria. Again with regard to the improvement of environmental conditions, the money needed for housing in Madras City alone according to the estimate of the Provincial Committee, is 100 crores. This has to be multiplied hundredfold if provision is to be made throughout India. Besides, the provision of good water supply and other essential amenities will need hundreds of crores. With the legacy of top-heavy and ever-growing administrative structure left to India by her erstwhile rulers, with various unexpected, complicated and extremely difficult problems affecting the safety and freedom of the country cropping up, entailing a great financial burden, it is not possible to expect the Government to do everything that has to be done.

We have therefore to think of measures, of palliatives, which will go some way at least to stop the growing menace of tuberculosis. For a long series of years, a large number of research workers have laboured to find out some remedy that would arrest or retard the progress of tuberculous infection; yet, in spite of the notable advances made in recent years in the treatment of other infections, the problem of tuberculosis remains unsolved. The reason is obvious. While the number of known cases is limited, the number of undetected cases is enormous, and the fell disease is not only eating into their vitals, but also spreads itself far and wide. That is why mass radiography has been suggested and is adopted in many countries. While this will help the detection of advanced cases, it may not be of much help in the detection of cases which are in the incipient stage. Therefore some other method had to be found out which will not only help detection but also induce specific resistance to tuberculosis.

Bacillus-Calmette-Guerin (B.C.G.) was developed by CALMETTE in the Pasteur Institute in Paris. A bovine strain of tubercle bacilli was isolated by CALMETTE in 1906 and this was subcultured on an ox-bile potato medium, and as a result of considerable research extending over a period of 13 years, a strain of a virulent bacilli was secured. It is this strain that is called B.C.G. After testing it on animals with a view to be assured of safety and security it was tried on human beings for the first time in 1921. But the proper technique and doses had to be worked out, and these were carefully worked out during the years 1921-'35. In the beginning, in most countries, B.C.G. Vaccination was given only for new-born infants following the practice adopted by CALMETTE, but in the Scandinavian countries it was given to older children and young adults also. By 1948, according to Dr. P. V. BENJAMIN, Tuberculosis Adviser to the Government of India, more than one million people were vaccinated in Denmark, Norway and Sweden alone. This B.C.G. Vaccination has been in vogue in other countries like the United States, Canada, S. America, Russia and, more recently, in most of the other European Countries.

This vaccination, like that against small-pox, is one for preventing tuberculosis. It is considered to be one of the effective methods not only for reducing the incidence of tuberculosis, but also the mortality from it, and for the present holds the field as the only known practical method of inducing specific resistance to tuberculosis. It is not also to be administered indiscriminately, for it can be of benefit only to those persons who have not been infected by the tubercle bacillus. Also, it has no value in the treatment of tuberculosis disease. The test known as the tuberculin test is a simple one. Tuberculin is an extract prepared by special methods from killed tubercle bacilli. It has the property of causing a local reaction when it comes into contact with a person who has been

infected with tuberculosis, but not with one who has had no such infection. This test is made by injecting a drop of a suitable dilution of purified tuberculin into the skin of the forearm of a person. If, on the third day, when the reading is made, a small reddish thickening at the site of injection is seen, it is considered a positive reaction. If there is nothing seen at the site of inoculation, or the thickening is below the standard size, it is considered that the reaction is negative. For children under 12 years of age a plaster containing tuberculin ointment is applied on the front of the chest and it is allowed to act for 24 hours, when the plaster is removed. The reading is taken after 72 hours. In positive cases small pin head nodules will be present surrounded by some redness. At least four such nodules are considered necessary to constitute a positive reaction. Such a positive reaction indicates that the person has had a natural infection with tuberculosis which has resulted in the development of anti-bodies which offer a considerable amount of protection (immunity) against a tuberculous infection from outside. B.C.G. Vaccine has also the power to convert the non-reactors into reactors to tuberculin in a harmless way. It is stated from the experience gained that it confers almost the same immunity as natural infection with tuberculosis does but without the risk that some may develop tuberculosis as a result of it. By vaccinating non-reactors, a very high percentage of them (98-99%) may be converted into reactors. This means they will have, as long as they continue to be tuberculin positive, the same immunity as that obtained through a natural (but dangerous) infection. From extensive experience in the Scandinavian countries, U.S.A., Canada and other countries, it has been determined that B.C.G. Vaccination can cut down the number of new cases and deaths from tuberculosis to about 1/5th of what it would be without vaccination. This is not only of great value by itself, but by reducing the sources from which the disease can spread, vaccination will indirectly further control the incidence of new cases.

We are aware of the controversy that has raged in the Press about the suitability of B.C.G. Vaccination to this country, the economic and environmental conditions being what they are at present in India. The hesitation of Great Britain for a considerable length of time to adopt this method has, also, we are aware, been cited in support thereof. And the Lubeck disaster has been called to aid to support it. So far as the last is concerned, it falls to the ground as a most thorough legal and scientific investigation by scientists of world renown has established that the deaths were caused because of an unfortunate mistake by which a virulent strain of Tuberculin was substituted for B.C.G., both of them being cultured in the same laboratory and in the same incubator. In regard to the second, the memorandum on B.C.G. submitted by PROF. TYTLER in 1946 to the Joint Committee of the British

Tuberculosis Association and British National Association for the Prevention of Tuberculosis for the consideration of the Minister of Health has produced a favourable reaction and a National B.C.G. Laboratory is already under construction near London. So far as the first objection is concerned, the Red Indians in America live under conditions no more favourable than those obtaining in India at present, and that which has helped to minimise the incidence of the disease there is certainly bound to produce the same results in this country as well. We have besides the statement by eminent medical men headed by Dr. BIDAN CHANDRA ROY, Premier of West Bengal, that B.C.G. Vaccination is bound to be of great help in India in reducing the incidence of, and mortality from, tuberculosis.

There are, of course, certain precautions that have to be taken to ensure the safety of the vaccine as laid down by the B.C.G. Conference held in Washington in 1946. In accordance therewith the Government of India have restricted the production of B.C.G. to a single centre to ensure a uniform standard of potency. The chief of the Laboratory and his assistant have had the necessary training at the State Serum Institute, Copenhagen, and they have also visited the leading B.C.G. laboratories in Europe to gain a complete working knowledge. The laboratory is housed in a separate building and no other bacteriological work is done in this laboratory. The cultures, vaccine etc., are always kept under lock and key and only the staff, who have all been X-rayed to rule out tuberculosis in them, are allowed to have access to the laboratory. Every batch of vaccine produced here is put through the most stringent controls in the laboratory and on animals for purity, potency and non-virulence. The high standard of technique, competence of the staff and suitability of the building have been certified by Major General SIR SAMIR SINGH SOKHEY, Director, Haffkine Institute, Bombay, who had been requested by the World Health Organisation to visit the laboratory and report on it. For the present, the Vaccine will be supplied only to the B.C.G. Vaccination teams for use in specially selected centres by personnel who have had adequate training in the technique of tuberculin testing and vaccination. These are sufficient safeguards to ensure the purity and potency of the vaccine and the proper application thereof.

The question remains whether the vaccinated individuals who are living in infectious surroundings should be segregated. Dr. JOHANNES HOLM, Chief of the Tuberculosis Division, State Serum Institute, Copenhagen, who is a recognised authority on the subject and who is also the expert adviser to the W.H.O. says:

“It should be stressed that when isolation is used at all it is only to protect the reputation of the vaccine, and not because it is considered dangerous to give vaccine to a person in the so-called pre-allergic stage of a virulent tuberculous

infection, nor especially dangerous for a person who is vaccinated to be exposed to a virulent tuberculous infection before the vaccination has made him a reactor to tuberculin. If vaccine is given to persons in the pre-allergic stages of a virulent tuberculous infection, the vaccination will, of course, have little or no value and some cases of tuberculosis will develop. These cases do not develop because of the vaccination but would develop anyway.”

At its meeting in Paris on 15th June, 1948 the W. H. O. Sub-Committee on Tuberculin Testing and B.C.G. Vaccination recommended that “for the mass vaccination programme, isolation of vaccinated persons is not necessary and that segregation of open cases even though desirable for other reasons is considered not to be an essential measure for a mass B.C.G. Vaccination programme.” This expert opinion sets at rest all doubts in this behalf.

When vaccination was decided upon as a protection against the attack of small-pox, many were the objections raised against it. Even now there are those who hold the view—and we include among them the Naturopaths—that the inoculation of vaccine into the human body is productive of the greatest harm. But every one will admit that vaccination has afforded considerable protection and immunity. B.C.G. Vaccination belongs also to the same category and is certainly capable of assuring immunity. While therefore we accord our support to the B.C.G. Vaccination programme adopted by the Government, we hasten to warn them against being lulled into a fall sense of security against the disease and giving up measures to ensure satisfactory housing, environmental and other conditions. Both should be taken on hand together and worked out. Then and then only can real and lasting improvement be brought about. We would therefore urge upon the Government the imperative necessity of carrying on both the measures side by side to ensure that the results expected from B.C.G. Vaccination are assured.

Playing Upon Patriotism

[T] is very necessary that patriotism and the sense of service to humanity must permeate the fibres of every human being. It is one thing to appeal to the inner soul of the people to be patriotic and devote themselves to the service of suffering humanity, but it is quite another thing to ask them to be satisfied with a pittance which will not be enough to keep even their own bodies and souls together. This is what is exactly done in regard to the rural medical practitioners. “Go To The Village” has now become the most fashionable slogan of the high and the mighty. The reasons are obvious. It is the rural population that is the backbone of the Government, but in no country in the world have they been so

badly neglected as in this unfortunate land. They lack the elementary facilities for a healthy living. Protected or proper water supply they know not; many villages have no road whatever with the result that in the rainy season they have to wade through knee-deep mud to reach their villages; the less said about the medical facilities the better. It is accepted by every one in authority that it is the absence of these and other essential facilities that are very largely responsible for ill-health, diseases and the high rate of mortality among them. In the circumstances the provision of adequate medical facilities in rural areas has become a major problem now. Appeals are made to medicos and to students coming out of the medical colleges that they should settle down in villages and help relieve the sufferings of ailing humanity. But what are the emoluments given to these people who are asked to carry out one of the most essential and important duties of the State? A peon in a Government office in the metropolis who has only an elementary knowledge of the 3R's gets more than a rural medical practitioner who has, after sufficient education, taken to the study of either the allopathic or indigenous system of medicine and undergone a course of at least 4 or 5 years. Is this fair? We ask. Is this the proper way to attract medical men and women for service in rural areas which are badly lacking in facilities now? An appeal to patriotism and a sense of public service has its own use; but it is subject to certain restrictions and limitations. The appeal must be associated with acts consistent with common sense, fair play and justice. It is because very little attention is paid to these latter that service in rural areas has become less and less attractive and even those who responded to the Government's appeal and settled down for practice in rural areas are now giving them up with the result that many dispensaries had to be closed down for want of the necessary personnel.

The danger inherent in sending raw medical graduates to settle down in rural areas in-charge of dispensaries has not been given the attention which it certainly deserves. Apart from the fact that the young medical graduate who is scientifically minded and trained and who is used to the amenities of city life finds himself a misfit in the new environment, his inexperience forebodes real danger. The diseases prevalent in rural areas are many and varied, and the doctor who settles down in a village, if he is to justify his existence and be of real service to the people, must be efficient in all branches of the science to be able to deal with any kind of disease. It cannot be said that a graduate who comes out of the medical college and is appointed to a rural dispensary will be quite efficient in every branch of medical science to discharge his duties and responsibilities properly. And it is because such raw people are posted to the rural dispensaries that the question of their emoluments also does not receive that much attention which it deserves. The remedy

for this state of things, it is suggested, lies in bringing District Headquarters Hospitals up-to-date with specialists and transporting the patients in the villages to the hospitals for proper treatment, if need be. While we certainly support the idea that the District Headquarters Hospital should be fully equipped and maintained at the maximum state of efficiency to meet any kind of emergency, that will not, by itself, we think, help to solve the problem. A District covers a very wide area and it will neither be possible nor feasible to transport all emergency cases from distant corners to the headquarter hospital. The real and most effective remedy is the posting of able, experienced and efficient medicos who have put in years of service to rural dispensaries from among the members of the service to rural areas so that they can deal with every kind of disease at any moment. The District or Taluq Headquarter Hospital will always have a number of senior men, able and efficient, and the raw graduates can be posted to these institutions to undergo training for a reasonable period so as to equip themselves for undertaking sole charge of institutions. The adoption of such a plan will not only be a real boon to the rural population but also necessitate the giving up by the Government of their present niggardly policy in regard to the emoluments of rural practitioners and the elimination of the present distinctions altogether.

Gleanings From Medical Press

MEDICINE

Sulphadiazine and streptomycin in bubonic plague.—(*Br. Med. Jour.*, 29.5.48.)

Haddad and Valero treated 17 patients in Iraq with Sulphadiazine 2 g. followed by 1 g. 4 hourly as a routine. All but one recovered. Three others who were seriously ill on admission and got worse in spite of Sulphonamide were given Streptomycin. Before admission to hospital they had been treated by local practitioners for 3 to 6 days with Penicillin and/or Sulphathiazole. *B. Pestis* was isolated in each case from buboes. Streptomycin in doses of 200 mg. 3 hourly produced marked improvement in 12 hours in 2 cases while the third responded to 300 mg. doses within 24 hours. All three recovered: Buboes did not resolve, however, but suppurated and had to be incised and drained.

T.N.S.P.

Treatment of idiopathic pernicious anæmia.—(*Jour. Am. Med. Assoc.*, 138, 12, pp. 870-873; 1948).

Addisonian anæmia is a complex disease resulting from a lack of specific factors necessary for (1) the normal growth and development of the erythrocytes; (2) the nutrition of the nerves and spinal cord; and (3) the correct functioning of the gastro-intestinal tract. The clinical features of the disease are:—(1) macrocytic anæmia; (2) subacute combined sclerosis of the spinal cord and peripheral neuritis; and (3) atrophy and soreness of the tongue, often diarrhoea and a pronounced achlorhydria. The patient is completely relieved of active symptoms by adequate liver therapy.

The most desirable liver extract should contain 15 anti-anæmic units per cc. Drs. Haden and Borty have followed the principle of giving 15 units daily for

2 weeks, then twice a week for 3 months, and then weekly for a further 3 months. Two injections of 15 units are then given every month for six months. After this intensive treatment with a *potent* extract for a year, monthly injections are given for the rest of the patient's life. Usually 15 units suffice; 30 units will supply any need. Once the correct diagnosis is made of pernicious anaemia, we know that the patient will always have it for life. There is no record of a recovery from true pernicious anaemia of the Addisonian idiopathic type. The disease is not cured. Only compensation is provided for the deficiency by the administration of a *potent liver extract*. Treatment must, therefore, be constant, regular and continuous throughout the life of the patient. Rarely does a patient remain disabled with proper therapy properly administered.

Folic acid is only partial therapy and does not relieve the deficiency responsible for the neurologic lesion—the most serious aspect of the disease. T.N.S.R.

Reactions to penicillin therapy for syphilis.—(*Jour. Invest. Derm.*, 10, Feb. 1948).

Thomas *et al* have classified the complications (minor) met with in their experience in the treatment of over 10,000 cases of syphilis with penicillin.

1. *Urticaria*, in 2.5 per cent of the cases, appeared in 6 to 12 days after starting treatment. The reaction varied from mild itching to severe pruritus and angioneurotic oedema, with fever in some cases. The reaction lasted for 4 to 6 days and then passed off with or without specific treatment.

2. *Exacerbation of secondary syphilitic lesions* occurred only in secondary syphilis and lasted for 5 to 10 days, whether treatment was continued or not; local oedema, sore throat, and high fever, together with relapse in secondary lesions were the usual symptoms. No spirochaetes were however found on dark ground examination of the serous fluid from such lesions.

3. Papular reactions (erythema) developed in the first 2 days and lasted for about 3 days; this was seen in 25

cases only of this large series. This reaction was very mild and the treatment suffered no interruption on this score. The authors consider that such reactions were probably associated with fungus sensitivity.

4. *Localised dermatitis at site of injection*, symptomless and unimportant, on rare occasions (in a dozen cases) a marked papular or follicular eruption was seen, which, however, disappeared in 2 or 3 days. 5. *Bullous dermatitis* occurred in only 2 cases in which the bullae were noticed on the exposed surfaces of the upper arm. T.N.S.R.

Oral penicillin in the treatment of various bacterial infections.—(J.A. Robinson *et al*, *Am. Jour. of Med.*, 4, pp. 716-23, 1948.)

Penicillin tablets buffered with Cal. Carb. were given orally in various bacterial infections (pneumococcal pneumonia, scarlet fever, tonsillitis, pharyngitis, erysipelas, otitis, Vincent's angina, skin infections). The results were very satisfactory and compared favourably with those obtained with parenteral penicillin. The total oral dose was five times higher than the total intramuscular dose for the same infection. In pneumonia 75,000 units every 3 hours for 48 to 72 hours; scarlet fever, 125,000 units every 3 hours for 5 days; tonsillitis and pharyngitis, 100,000 units every 3 hours for 5 days; erysipelas same as for tonsillitis; otitis, 100,000 units every 3 hours for 48 hours after all evidence of acute infection has disappeared. Vincent's angina 50 to 75,000 units every 2 to 3 hours for 3 to 4 days; skin infections 125,000 units every 3 hours for 5 days. Oral penicillin is useless in bacterial endocarditis. T.N.S.R.

Treatment of obesity.—(M. M. Kunde, *Annals of Int. Med.*, 28, 971-988, 1948).

Fifty patients with obesity of various types were instructed by the author to eat anything they liked, within a high-protein and low-fat and low-carbohydrate range, and not to worry about their caloric intakes. Eggs, creamless cheese, gelatin, vegetables, fish and fowl formed

the bulk of their food. No other treatment was given. Supplements of vitamins and minerals were given when indicated. The results were strikingly successful. The high protein diet resulted in a reduction of blood-pressure in patients with previous hypertension. Thyroid was found to be of limited value by the author, in treating cases of obesity. The sweeping statement is made by the author that no hormones or glandular preparations are necessary adjuncts in the treatment of any type of obesity, except that associated with *hypothyroidism*. Some very remarkable and striking photographs are given and the clinical details are elaborately described with the results of frequent controlled observations during the entire period of treatment. T.N.S.R.

Pharmacology of the failing human heart.—John McMichael, *Br. Med. Jour.*, 27-11-48.

In his Strickland Goodall lecture delivered on 3rd June 1948 in London, Prof. McMichael gives an account of the action of "*Digoxin*" and "*Ouabain*" (g. strophanthin) in patients with heart failure using as his method of observation Forssmann's technique of right heart catheterization. In this procedure a catheter is passed into a superficial vein of the left arm and pushed upwards until the tip comes to lie at the junction of the superior and inferior venae cavae where the mixed venous blood enters the right heart. A sample of blood drawn through this catheter is examined for its oxygen saturation. The same test is done on a sample of arterial blood and the difference in oxygen content calculated. The oxygen consumption per minute is measured and the cardiac output in c.c. per minute is obtained by dividing this figure by the difference in oxygen content above mentioned. The pressure on the right auricle is also determined at the same time. By a continuation of these two assays McMichael assesses the effect of a drug like *Digoxin* on the performance of the heart.

There has been difference of opinion hitherto, about the way in which digitalis and its glycosides act. McMichael divides cardiac failure with congestion

and oedema into 2 groups (1) *failure with a low output* due to hypertensive, ischaemic and valvular heart disease; and (2) *failure with a high output* due to conditions demanding an increase in the output, such as emphysema, anaemia, arterio-venous aneurysms, Paget's disease of the bone and metabolic disturbances such as beri-beri. McMichael found that in the first group of heart failure (low output) *Digoxin* acts primarily on the venous pressure—an argument which may not convince all. Describing the results obtained with *Ouabain* he states that they afford clear evidence of a direct stimulating action on the myocardium. He found in 50% the cases of heart failure a definite increase in cardiac output with only a very slight reduction in venous pressure when strophanthin is given. McMichael found that *theophylline* caused a transient rise in the output of the failing heart and thereby reduces venous pressure. He describes also some interesting observations on *Mersalyl*. His colleagues have found that the intravenous injection of this substance into an oedematous patient caused a fall in right auricular pressure and a rise in cardiac output which reached its maximum 7 hours after injection. This is a clear demonstration of the circulatory action of *Mersalyl*. By this time the patient had passed 2 litres of urine. This is usually considered the reason for the lowering of the right auricular pressure. McMichael's exposition and arguments challenge the orthodox views of many pharmacologists. T.N.S.R.

Bone and joint lesions in leprosy.—*Radiology*, 50, 619-30., 1948.

Gomez and Acosta analysed the radiographic and clinical findings in about 500 lepers. In nearly 60 per cent of these, varying degrees of digit-mutilation were noted. The phalanges showed some degree of decalcification in the radiograms. Varying degrees of absorption were noticed in the terminal phalanges. Joint destruction with deformities due to softening and pressure was also manifest. In over 30 per cent of cases, skin lesions with no clinical evidence of skeletal destruction were found, but on radiographic examination in

nearly one half of these cases (87 cases) phalanges were found to be decalcified. In 68 per cent, there were bone changes suggestive of leprosy.

T.N.S.R.

Quinidine therapy.—Howard B. Burchell, M.D., Rochester Minn., Jan. 1949 issue.

It has been about a quarter of a century since quinidine therapy for cardiac arrhythmia became widely accepted in America. If one reviews the American publications of the early twenties which discuss the results of quinidine administration, he finds that the clinical analysis of the benefits and dangers were accurate. Despite 25 years of further clinical study, the administration of quinidine is still associated with disputed issues, particularly in regard to its use in established auricular fibrillation.

Quinidine is useful in the cure or prevention of many instances of paroxysmal tachycardia and extrasystolic arrhythmia, definitely mandatory in the treatment of ventricular tachycardia of any duration, and of some lesser value in auricular flutter. It has its most frequent application in the restoration of normal rhythm in cases of auricular fibrillation where discrimination in selection of patients should be most important.

The questions which are asked by most investigators have been the same since 1920:

1. What type of case will most likely revert to normal rhythm?
2. What type of case will maintain normal rhythm?
3. Is the circulation of patients improved by restoring normal rhythm as compared with their status with auricular fibrillation under digitalis therapy?
4. What are the dangers of quinidine therapy?
 - (a) Sudden death.
 - (b) Peripheral embolism.
 - (c) Severe reactions.
5. Can one outline definite contraindications to quinidine therapy?
6. Are any new methods available to regulate quinidine dosage, favourably modify its effect or minimize its dangers?

1. What type of case will most likely revert to normal rhythm?

My own impression has been that hearts which are the most anatomically and functionally normal are the easiest to convert to normal rhythm—as in cases of:

- (a) Persistent auricular fibrillation following relief of hyperthyroidism.
- (b) Persistence of auricular fibrillation in an apparently normal heart previously subject to paroxysmal auricular fibrillation.
- (c) Auricular fibrillation occurring as the only sign of probable arteriosclerotic heart disease.

Despite recent studies to the contrary, I have felt that cases of long-standing valvular disease associated with heart failure were more difficult to convert to normal rhythm, a view supported by the earlier studies in the literature. However, my personal experience is small as I have held the belief that in such cases quinidine is only occasionally indicated.

2. What type of case will maintain normal rhythm?

In general, the case of auricular fibrillation that will revert most easily to normal rhythm will be the one most likely to maintain it. However, exceptions are sufficiently frequent that one is not justified in giving up quinidine therapy of small dosage and of short duration on the assumption that if normal rhythm were restored it would not persist for a significant length of time. In the presence of extreme or aneurysmal dilation of the atria, however, or when heart failure is persistent, auricular fibrillation is apt to recur even after having been successfully abolished.

3. Is the circulatory status improved by having normal rhythm restored, particularly when compared to the status with auricular fibrillation under digitalis medication?

This question is not simple to answer, and one must avoid committing himself to a single possibility. From the standpoint of improvement, the clinical opinion, based on close study of the patient's exercise tolerance, is as important as more objective tests such as

vital capacities or even cardiac outputs. Patients may fall into three categories:

(a) The largest group, those in whom the re-establishment of normal rhythm causes no definite increase in cardiac efficiency, as compared to the status when under adequate digitalis therapy.

(b) Patients showing marked improvement in exercise tolerance and general well being after the restoration of normal rhythm. This second group may be subdivided into three types: (i) patients with arteriosclerotic heart disease in whom the rapid irregular ventricular rate with exercise seems to limit the cardiac work, even when under digitalis therapy, and in whom the restoration of normal rhythm is associated with decreased effort dyspnea and decreased effort angina; (ii) patients whose circulatory status is constantly jeopardized by pulmonary emboli; (iii) a small group of patients in whom, in the presence of valvular heart disease, heart failure was intractable to therapy until a phenomenal return to cardiac compensation took place following restoration of normal rhythm.

(c) Patients whose heart failure was initiated or aggravated by restoration of normal rhythm.

4. What are the dangers of quinidine therapy?

(a) Sudden death: I believe that anyone who treats patients with severe valvular disease with quinidine to restore normal rhythm will have sudden deaths, perhaps as high as one case in 30. I do not believe that these deaths can be ascribed simply to the fact that many patients with heart disease do die suddenly. The incidence of death in the "quinidine periods" is higher than the incidence of death in an untreated group in a similar short period. Deaths have most frequently occurred in the presence of cardiac enlargement and heart failure, and are not related to the amount of quinidine taken.

(b) Peripheral embolism: That the occurrence of peripheral embolism is associated with restoration of normal rhythm is, to my mind, an established fact. This does not mean that embolism might not have occurred anyway, but rather that, in a person who has had or

is about to have embolism from an auricular thrombus, this complication is likely to be initiated by restoration of normal rhythm. As will be later mentioned, I believe that systemic or pulmonary emboli associated with atrial fibrillation are indications for quinidine therapy to restore normal rhythm. The various factors that play a role in the cessation of embolic accidents with the restoration of normal rhythm are not entirely clear. The usual explanation, that the synchronously contracting atria is a poorer place for thrombus formation, is not entirely satisfactory. The additional factor of increased circulation rates, or even the cessation of digitalis therapy and its thrombophilic propensities, may have importance.

(c) Severe reactions, such as circulatory collapse with or without marked tachycardia or asystole, occasionally occur.

5. Can one outline contraindications to therapy?

In general: (a) Patients with valvular heart disease with long-standing atrial fibrillation who are well compensated on maintenance doses of digitalis.

(b) Patients with hyperthyroidism.

(c) Patients with greatly enlarged hearts and congestive heart failure, particularly when associated with slow ventricular rates.

(d) Patients with complete heart block. (I have not considered bundle branch block a necessary contraindication).

(e) Patients whose anginal pain has been relieved by the onset of auricular fibrillation.

The general indications for a quinidine trial to restore normal rhythm are:

(a) Auricular fibrillation in the absence of valvular disease and contraindications as listed above.

(b) A few cases of progressive heart failure associated with rapid ventricular rates.

(c) Occasional cases in which heart consciousness is a serious problem.

(d) Cases with the history of repeated emboli, if possible, with none of the contraindications listed above.

6. *Are there any new methods available pertaining quinidine therapy?*

Method of dosage: intermittent or continuous, both of which are effective, have long been used and may be combined. During the period the quinidine dosage is being increased, I like to have it accomplished in the early part of the day so that if a reaction should occur, the house physician will be available to observe the patient and to carry out appropriate treatment.

Premedication with digitalis is definitely indicated with the history of failure, in my opinion. If there is any doubt about maintenance dosage, I usually stop the digitalis during the immediate quinidine administration so that there will be no chance of having minor toxic reactions of digitalis to confuse the clinical picture during the time of high quinidine dosage.

From reports in the literature, quinidine therapy based on blood levels of quinidine does not yet seem to hold much promise. Considering the marked variability in dosage required for different patients, perhaps such a method of dosage calculation could not be expected.

With the availability of anticoagulant therapy and the possibility of embolic accidents with restoration of regular rhythm with quinidine, it would seem proper treatment to make every effort toward prevention of recent thrombus in the atrium prior to quinidine therapy. While heparin might be used, I have been employing dicumarol for a week to 10 days before quinidine in all patients who have a history of embolic phenomenon or of any suggestion of cardiac decompensation.—*Journal of Iowa State Medical Society.*

The management of peptic ulcer.

—A. On patients without gross hæmorrhage.

1. Diagnostic procedures.

(a) History and physical examination to include a rectal examination and a proctoscopic examination on the ward.

(b) Omit gastric analysis and gastro-intestinal series on patients who are hæmorrhaging.

(c) Gastro-intestinal X-ray series to be ordered immediately on admission.

(d) Gastric analysis to be ordered immediately.

(e) Three stools to be examined for ova and parasites and for occult blood. Begin on third day after admission.

(f) Red blood count, white blood count, differential, hæmoglobin and packed red blood count volume (hæmatocrit) on admission.

(g) Urine analysis.

(h) Gallbladder dye series when indicated.

(i) Obtain date for re-check gastro-intestinal series for the fifth week on patients with duodenal ulcer.

(j) Obtain date for re-check gastro-intestinal series for the end of the second week on patients with gastric ulcer.

2. Treatment.

(a) Bed rest for 21 days. Head privileges only. Progressive dietary regimen as described under No. 4.

(b) Begin the dietary regimen immediately on admission.

(c) If the diagnosis of peptic ulcer or one of its related conditions, such as hypertrophic gastritis, diverticulum, malignancy, etc., is not verified, transfer to the proper service.

(d) The milk and cream mixture is to be given every hour on the hour.

(e) The alkali every hour on the half hour.

(f) The gastric sedative three to four times daily, as ordered by the ward medical officer.

(g) Constipation and faecal impactions are a common sequela of the alkali used. Be watchful for its symptoms. Mineral oil, 15.0 cc. to be given each morning, and the cascara mineral of mixture, 15.00 cc. each evening.

(h) Percorten (desoxycorticosterone acetate—D.C.A.) 10 m. every other day, or 5 mg. every day intramuscularly until the first re-check X-ray of five weeks. On order of the ward medical officer only.

3. Alkalies.

(a) Amphojel or cremalin (aluminium hydroxide preparations) drams 1 (4.0 cc.) diluted in half a glass of water.

(b) Sippy powder number 1 (laxative) or Sippy powder number 2, 1 level teaspoonful, only on order of ward medical officer.

(c) Milk of magnesia may be used occasionally to combat constipation, on order of ward medical officer.

4. The progressive milk-cream dietary regimen.

First and second days: 1½ ounces milk and cream, equal parts, every hour on the hour from 0600 to 1830, and every half hour from 1830 to 2130 during the first three weeks. Whole milk, or whole milk boiled may be substituted on order of ward medical officer.

Third and fourth days: Add one soft-cooked egg at 0700.

Fifth and six days: One soft-cooked egg at 0700. Cooked cereal 3 ounces at 1200.

Seventh and eighth days: One soft-cooked egg at 0700. Cooked cereal 3 ounces at 1200. Jello, 3 ounces at 1700.

Ninth and tenth days: Cooked cereal 3 ounces at 0700. One soft-cooked egg at 1200. Jello 3 ounces at 1500. Custard 3 ounces at 1700.

Eleventh to twenty-first days: Cooked cereal 3 ounces at 0700. Jello 3 ounces at 1000. One soft-cooked egg at 1200. Custard 3 ounces at 1500. One soft-cooked egg at 1700.

Twenty-first day: Patient may be up and out of bed around the ward.

Twenty-second to twenty-eighth days: Five feedings as above. Continue milk and cream every hour on the hour. Continue alkali every hour on the half hour, the last dose at 2100. The half-hourly feedings of alkali after 1830 can be omitted.

Fifth week: Bland, meat-free diet, three meals daily on the ward. Milk, one glass at 1000, 1500 and 1700. Alkali every hour on the hour between meals until 2100. Re-check gastro-intestinal series on duodenal ulcer patients.

5. Sixth week until discharged.

(a) Bland diet with broiled or roasted meat at noon in mess hall, unless modified by order of the ward medical officer. (Patients who have not shown complete healing at the fifth week re-check, will

remain on the bland meat-free diet.) All vegetables are pureed. Continue mid-meal feedings and alkali every hour. Citrus fruits not allowed.

6. Smoking is not allowed.

7. Coffee is not allowed.

8. After the fifth week, the gastro-intestinal series is to be re-checked every month until healing is obtained.

9. Re-check the gastric analysis in the fifth week and again when X-ray evidence of healing has been reported.

10. Night pains:

If at morning sick call, the patient complains of distress at a specific hour after the lights are out, order additional feeding of milk and alkali, one hour before the anticipated symptoms. It may be necessary to awaken a given patient several times each night for such feedings.

Unless the patient is hæmorrhaging. *Do not order morphine for night pains.* An additional dose of the gastric sedative plus the milk and alkali will suffice in most instances. Atropine sulphate 1-100 gr. (0.006) by mouth or by hypo may be used with success.

B. On patients with gastro-intestinal hæmorrhage.

1. Diagnostic procedures.

(a) Red blood count, white blood count, hæmoglobin, differential and packed red cells volume (hæmatocrit), on admission as an "emergency."

(b) Blood type, Rh factor, and cross match for immediate transfusion of blood, at least 500 cc. initial. Additional amounts of blood or of washed red cells.

(c) Re-check the hæmatocrit every morning.

(d) Non-protein nitrogen immediately on admission as an "emergency."

(e) Re-check the non-protein nitrogen each morning if bleeding continues.

(f) Urine analysis immediately on admission for specific gravity, albumin, sugar, acetone; chemical for hæmoglobin; sediment for red blood cells, white blood cells, casts.

(g) Daily urinalysis as above until discontinued by order.

2. Treatment.

(a) Absolute bed rest.

(b) Morphine sulphate grains $\frac{1}{4}$ (0.015) every four hours for six doses, until physiologically tolerated. Respirations may drop to 12; discontinue morphine if respirations go to 12. If you have overshot the physiologic dosage and Cheyne-Stokes appear, give 4 grains caffeine-sodibenzoate every three hours. The respirations will become regular. Do not give too often, or the patient will become very restless. Repeat the order for morphine with stop at six doses each day as above. Hemorrhaging patients stand large amounts of morphine.

(c) Nothing by mouth except cracked ice until the patient stops bleeding.

(d) Ice bag to abdomen.

(e) Immediately after admission to the ward, while awaiting the blood reports and blood for transfusion, begin an intravenous infusion of saline. Keep the fluid going until the blood is available.

(f) If the patient presents signs of shock, begin an infusion of plasma in the other arm. He will usually take 500 cc. before the blood is available. If the shock is severe, clamp off the tubing and inject 3 to 5 cc. of coramine (nikethamides) directly into the tubing on the vein side, open the clamp and permit the plasma to flow.

(g) Use the value of 45 hematocrit as the average normal. Order blood for transfusion in multiples of 500 cc. (100 cc. for each per cent. decrease in the hematocrit). For example: If the hematocrit value is 35, order 1,000 cc. of whole blood or its equivalent in washed red cells, for the initial transfusion. If the hematocrit is down to 30, order 1,500 cc. of whole blood for the initial transfusion.

(h) The clinical condition of the patient, the presence or absence and recovery from shock must be the guide for additional amounts of blood during each 24 hours. A re-check on the hematocrit within the first 12 hours after transfusion when aid as a rough guide as to the net gain and the rate of bleeding. For example, if all bleeding had stopped, you give 1,000 cc. whole blood transfusion when the hematocrit

was at 30 per cent., you cannot expect over five points increased in the hematocrit even if all the transfused blood is retained. Be satisfied if the next hematocrit is at the same level. This at least indicates that you have kept pace with the rate of bleeding.

(i) When the hemorrhage ceases, the hematocrit will begin to rise.

(j) Continue the daily transfusion until the hematocrit reaches 40 per cent.

(k) Between the second and fourth day, usually about the third day, a patient receiving multiple transfusions of whole blood, is apt to have a chill and a rise in temperature during a transfusion. This usually occurs late, after most of the transfusion has run in. This may or may not be true transfusion reaction. Stop the particular bottle of blood. Give the patient an enema. The result will be a foul and putrid stool of decomposed blood. Check all urine for six hours. If dark urine appears, test for hemoglobin and notify the chief of medicine that the patient has a delayed transfusion reaction. If the urine remains negative for hemoglobin, proceed with the next 500 cc. bottle if the patient still needs blood.

(l) A reaction that appears within a few minutes after the transfusion has begun to flow is a danger signal. Look for dark urine containing hemoglobin. This is a true transfusion reaction, and signifies a possible renal shut-down. Stop immediately. When the first urine appears, watch the blood chloride level. Saline in large amounts is imperative.

(m) Each morning and evening administer 1,000 cc. of 10 per cent. dextrose in distilled water and saline respectively. This is in addition to the initial fluids given on admission and any blood that has been given. The basal caloric needs are covered by these infusions.

(n) When the hematocrit begins to increase, start the dietary regimen. Omit the gastric sedative for one week.

(o) In the third week after bleeding has ceased, order gastro-intestinal series, X-ray examination, and gastric analysis.—*Aphorism on Peptic Ulcer by Lewis Gunther*—Abstracted from the *General Practitioner*.

The toxicology of the newer metals—Lawrence T. Fairhall. U. S. P. H. S., *Brit. J. Indust. Med.*, 3:207-12 October 1946.

The toxicology of metals and metal compounds which have recently become of industrial importance is discussed on the basis of their specific uses in industry and a comprehensive review of cases reported in the literature. Beryllium, apparently not a protoplasmic poison, has caused dermatoses and chemical pneumonitis, this latter also attributed to beryllium sulfate. This compound entering skin cracks caused ulcerations. Beryllium oxide is suspect in sarcoidosis.

Cadmium is dangerous. The mortality rate of industrial cadmium poisoning is 15 per cent. Cadmium oxide, liberated on heating cadmium-coated metal, is avoidable by testing metals for the yellow cadmium sulfide stain or by compulsory labeling of tools, etc. Cadmium plated drinking vessels, ice cube trays or even refrigerating units have caused poisoning. Powdered cobalt has produced dermatitis and in the presence of sensitivity hyperemic edematous vesicular lesions.

There seems to be no experimental work to indicate that columbium and tantalum produce any such untoward physiologic effects as might be expected because they belong to the vanadium branch of Group V. Indium toxicity is not considered sufficiently high to be of industrial importance. Osmium tetroxide has proved dangerous, resulting in ocular disturbances, an asthmatic condition on inhalation (1 fatality reported) and dermatitis and ulceration on skin contact. Platinum salts, as dust or mist, are an industrial hazard, resulting in respiratory disorders and dermatitis.

Selenium exposure may cause pallor, gastro-intestinal disturbances, nasal and throat irritation, garlicky breath and metallic taste, perspiration and nervousness. Selenium oxides or salts may cause dermatitis. The symptoms of tellurium poisoning are garlicky odor of the breath and urine, sweat suppression, dry mouth and metallic taste, with some cases of dry itching skin, anorexia, nausea and vomiting and somnolence.

Owing to their radioactive properties, continued contact with thorium compounds is dangerous. Thorium dioxide and mesothorium have shown untoward effects. Uranium and uranium hexafluoride are considered extremely toxic, with nephritis, necrotizing arterial lesions and chemical pneumonitis as possibilities. Vanadium compounds are toxic, causing lung damage different from ordinary pneumoconiosis.—*Quarterly Review of Medicine*.

Hemolytic transfusion reactions.

—M. J. Nicholson, M.D., Boston, condensed from *Anesthesiology*.

The purpose of this paper is to discuss the etiology, signs, symptoms and treatment of hemolytic transfusion reactions.

ETIOLOGY.—Many errors may cause the patient to receive incompatible blood. These are listed in Table I.

TABLE I.—Source of error in blood transfusions.

I. Errors in grouping and cross matching.

A. False negative reactions—weak typing sera.

1. Low titer from outset.
2. Deterioration from improper storage.
3. Deterioration from bacterial contamination.

B. False positive reactions.

- (1) Pseudo-agglutination (rouleaux formation)
- (2) Auto-agglutination.
 - (a) Cold agglutinins act on all bloods.
 - (b) Warm agglutinins.
- (3) Iso-agglutination—cold iso-agglutinins act only on certain blood.

II. Clerical errors in handling of blood at Bank.

- A. Incorrect labeling of pilot tube.
- B. Wrong blood released from bank.
- C. Mix-up in administering blood to patients.

Too much cannot be said about the importance of clerical errors which have in our experience resulted in the greatest number of transfusion reactions. It is

questionable whether this group of errors can ever be entirely eliminated, but they can be materially reduced by placing the responsibility for the collection of blood from the recipient and donor on the technical staff of the blood bank.

Elimination of the aforementioned errors along with the dangerous universal donor (Group O whose anti-A or anti-B titer is above 1 to 32) and the routine use of Rh compatible blood should go far toward preventing hemolytic reactions.

SIGNS AND SYMPTOMS.—A conscious patient who receives incompatible blood may show subjective signs and symptoms almost immediately, or the reaction may be delayed and the initial manifestations develop several hours after the completion of the transfusion. When incompatible blood is given to a patient under general anesthesia, however, there are no signs and symptoms to indicate what has happened unless by chance one should examine the urine and discover it to contain precipitated hemoglobin. Usually, in a conscious patient, 10 cc. of incompatible blood will cause a reaction and this is the basis for the "biologic" test (that is, carefully observing the patient during the administration of the first 50 to 100 cc. of blood). The earliest complaints registered by the person receiving incompatible blood are generalized tingling sensations, discomfort, anxiety, fullness of the head, precordial pressure and a sense of constriction in the chest or difficulty in breathing. The next most characteristic features are pain in the lumbar and chest regions and a chill which is generally followed by a high fever. In addition, signs of shock become evident, such as rapid pulse, cyanosis, nausea and vomiting, and a fall in blood pressure.

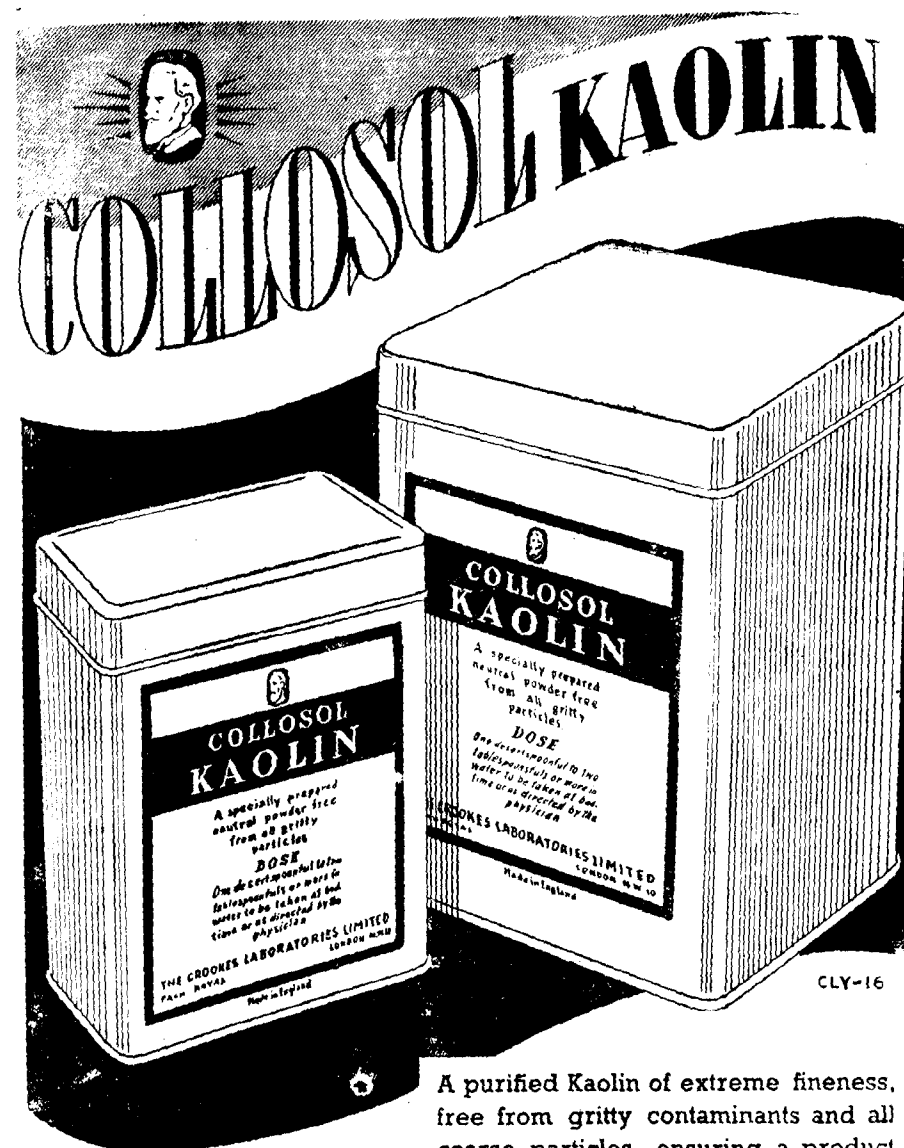
DIAGNOSIS.—The reactions associated with blood transfusions may be divided into three groups: (1) chemical and physical, (2) allergic and (3) hemolytic. In addition, the possibility of transmitting malaria, syphilis and infectious hepatitis in transfused blood must be constantly guarded against. Most of the chemical reactions can be prevented by using pyrogen-free distilled water, eliminating

foreign material from the transfusion apparatus and preventing bacterial contamination when the blood is collected. Physical reactions can be prevented by allowing banked blood to reach room temperature, administering it slowly to patients with organic heart disease and being ever on guard against air embolism. Allergic reactions can be held to a minimum by paying strict attention to the allergic history of the donor and recipient and establishing a routine of collecting blood from donors at least six hours postprandial.

No matter how insignificant a transfusion reaction may appear, a sample of the patient's blood must be obtained at once, preferably during or shortly after the chill, and the serum examined for hemoglobinemia. If a specimen of the blood being administered can be obtained, its grouping and cross matching along with the Rh factor should immediately be determined. If the entire transfusion has been administered, special studies of the recipient's blood should be made for evidence of incompatible cells.

The urine must be examined carefully for any evidence of hemoglobinuria. While these observations are being made, a complete check of the original grouping, cross matching and clerical work pertaining to the handling of the blood should be undertaken. It goes without saying, of course, that on even the slightest suspicion of a transfusion reaction, the administration of the blood should be stopped immediately. Once a transfusion has been discontinued because of a suspected reaction it should not, under any consideration, be started again until absolute certainty of its compatibility has been established.

TREATMENT.—Although urinary alkalization has not proved a cure-all, enough evidence exists in its favour to demand its immediate institution. The urine can be made alkaline by the administration of sodium bicarbonate or potassium citrate orally, or sodium bicarbonate, sodium racemic lactate or sodium citrate intravenously. If the urine is kept at a pH of 7.5 to 8, hemoglobin is precipitated. The urine should be kept alkaline as long as hemoglobinuria is



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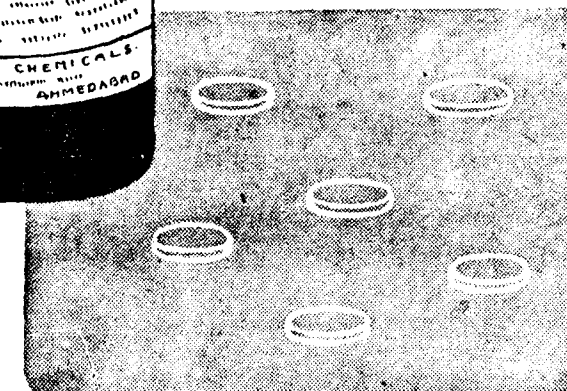
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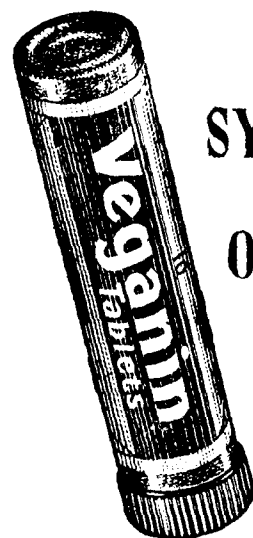
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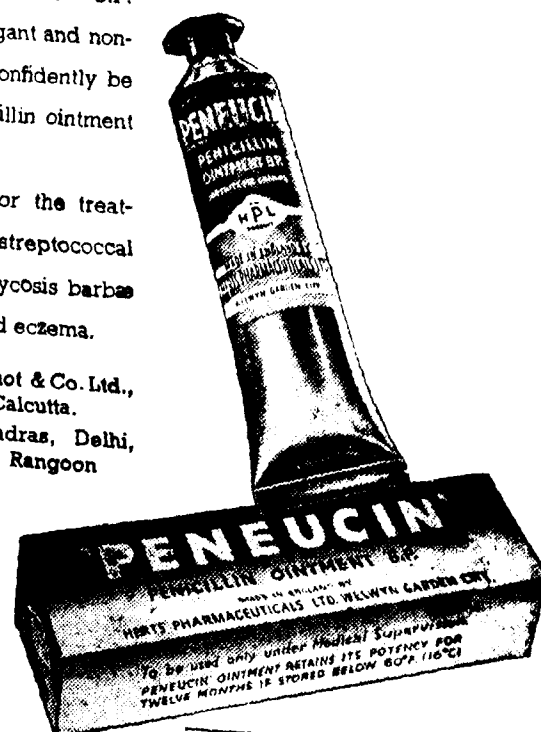
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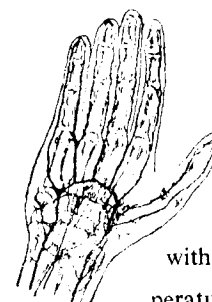
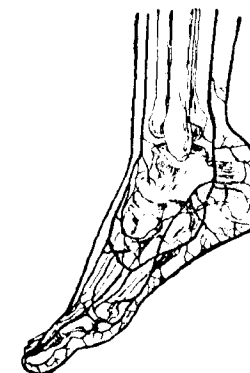


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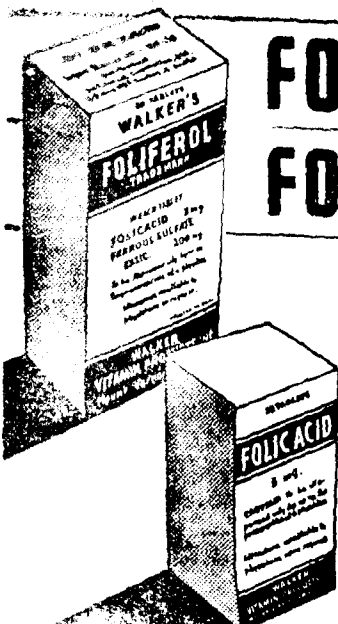
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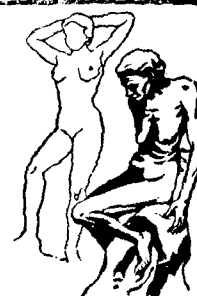
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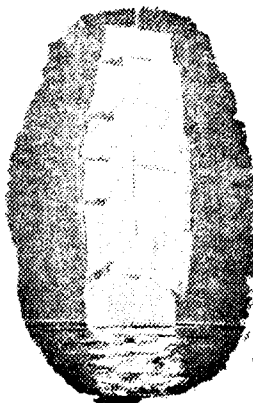
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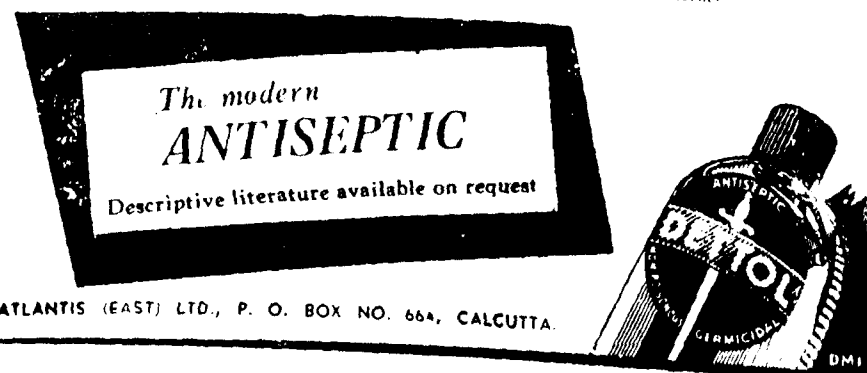
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Special Number on "Food and Nutrition."

The Antiseptic

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No. 6.

Nutritional Disease in India

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Indian Research Fund Association, Coonoor, South India.

It has been said that India harbours a veritable museum of nutritional diseases of all kinds and varieties. Unfortunately it is only too true. India is a large country with marked differences in climate, soil, water, crops, dietary habits and economic status of its inhabitants. Over all these supervenes the general poverty of the masses. It is no wonder therefore that one should encounter undernutrition and malnutrition in all its forms, mild, marked and severe. Whereas the clinically recognisable conditions of malnutrition can be fairly easily distinguished, the subclinical states of malnutrition escape recognition unless special efforts are made to study them. Almost all the known diseases of nutritional origin can be found in India. Several of them are more common while others less so although not completely absent.

There have been numerous nutritional surveys in various parts of India and they have brought to light the occurrence of nutritional disease on a widespread scale. I do not wish to stress the fact which has been evident for some time that the low state of health and the poor physical development of an average Indian can be ascribed to his poor nutrition. Mentioning it is merely stressing the obvious. In this article I propose to restrict myself to the geographical distribution of those manifestations which result from nutritional disorders. Not a single system of the human body has escaped the ravages of malnutrition and those who have made nutrition as their special study can very easily satisfy themselves

by closer scrutiny of the available information in India. It will be instructive to study the characteristic distribution of nutritional diseases within the country. For the sake of convenience these are described below under three general headings of diseases attributable to:

- I. Mineral element in nutrition.
- II. Vitamins
- III. Unknown aetiology.

I. Diseases attributable to minerals.—In this category one finds diseases which are due to (a) deficiency of a mineral element, primary or secondary or (b) excess of a certain element, e.g., fluorine.

In the submountainous regions of the Himalayan range right across the northern border of India one meets with the occurrence of simple goitre in which iodine deficiency has been known to play the leading part. An attempt to control this disease was made by the Punjab Government in Kangra Valley a few years before the partition. The author is not fully aware of the results of that experiment although reports about the ameliorative effects were available in 1946. It is also not known what steps have been taken by the other provincial governments to combat this disease in the goitre regions lying within their respective jurisdiction.

Surveys in the Kangra Valley of the undivided Punjab had shown the prevalence of disease due to deficiency of calcium and phosphorus accompanied by that of vitamin D deficiency. The phosphorus content of the blood serum and the wide occurrence of rickets and osteomalacia. A reference to these will be made at a later stage in this article. The diet of the poor South Indian has also been found to be deficient in calcium. Apart from the occurrence of mild rickets sometimes and low skeleton weight, however, no evidence of specific calcium deficiency has been forthcoming from South India.

Although direct evidence has still to be produced the classical researches of McCarrison have provided sufficient information to indicate that in the production of urinary calculi an imbalance between calcium and phosphorus and the deficiency of vitamin A may be responsible. In a survey carried out two decades ago it was found that in Sind, North West Frontier and the Punjab the incidence of urinary calculi was very much greater than in other parts of India. Owing to the partition of the country the "predominant stone areas" are no more with us, although the aetiology of the disease is still an interesting problem requiring further work.

Anæmias of the microcytic hypochromic type arise as the result of the deficiency of iron which may be a primary or secondary deficiency. Diet surveys, however, have provided ample evidence

to show that in Indian diets iron is present in sufficient amounts. It might be said that as all the iron present in food may not be physiologically available a condition of iron deficiency might exist in spite of there being enough iron in the diet. This could be true and the results of work on this problem at present in progress are awaited with interest. There is reason to believe, however, that iron deficiency may be secondary due either to increased demands by the body on the available iron or due to increased losses of iron from the body. In the former category can be placed microcytic anæmias in pregnancy, and in the latter, anæmias due to excessive menstruation and hookworm infestation. The condition is particularly common in young women. Anæmias such as above must be fairly common in India although their widespread occurrence has attracted attention in certain large industrial cities and amongst labourers employed in the plantation industry in Bengal, Assam and South India.

The example of a nutritional disease wherein the excess of a certain element is the causative factor is best illustrated by fluorosis. During the last ten years considerable evidence has been collected in Madras Province about the incidence of this disease in human beings (and cattle) in relation to the fluorine content of water supplies. The worst affected parts are the Ceded Districts and certain other regions forming part of the Andhra Districts of the Province of Madras.

II. Diseases due to vitamin deficiency.—In this category diseases due to vitamin A deficiency hold the pride of place. If there is one deficiency which can be considered characteristic of poor Indian dietary common to all parts of India it is that of vitamin A and its precursors. These nutrients are lacking in the average diets in varying degrees and correspondingly varying degrees of severity of the deficiency states can be encountered. The manifestations include discoloration, thickening and folding of the scleral conjunctiva, xerosis, general and localised as in Bitots spots and advanced conditions such as keratomalacia. Particularly in Bengal and Madras cases of preventable blindness attributable to this cause occur fairly frequently. Information about the eye disease attributable to vitamin A deficiency is not available from some parts of India. Night blindness due to vitamin A deficiency is fairly common along the West Coast of the peninsula and in the South. Phrynodema which has been ascribed to a deficiency of vitamin A (about the aetiology of which there are now grave doubts) associated with a dry horny and hyperkeratotic skin is common in South India although its occurrence in the North is comparatively rare. These manifestations of vitamin A deficiency are particularly common during the growing period of life.

Deficiency states referable to vitamin B complex are widely prevalent in the South. Frank beri-beri in adults has been reported

from restricted areas only; a few years back infantile beri-beri had become a real problem in Northern Circars. The reason for this localisation would appear to be that in Northern Circars people parboiled rice is consumed to a large extent. Among the children objective evidence is however lacking. This does not mean, of course, that clinically recognisable cases do not occur outside the Northern Circars; they do, but their frequency is not great. On the other hand, angular stomatitis, glossitis, scrotal dermatitis and involvement of other mucocutaneous junctions referable mainly to riboflavin deficiency are fairly common. Here too much more information is available in the South than in the North of the country. "Burning feet syndrome" resulting probably from pantoic acid deficiency has been attracting the attention of investigators since Dr. Gopalan's first observation reported in 1945.

Most of the macrocytic anæmias encountered in India may be of nutritional origin. Recently favourable reports of treatment with folic acid have provided sufficient evidence in support of the above statement. Folic acid has also been shown to be useful in the treatment of sprue. It is yet difficult, however, to assess the actual role of folic acid in these disorders.

Frank scurvy has been met with comparatively rarely in spite of the reported low intake of ascorbic acid by Indians.

At one time it was commonly believed that vitamin D deficiency must be rare in India. The reason for this was supposed to be the abundance of sunlight throughout the year with which tropical and subtropical countries are usually favoured. This opinion has had to undergo change during the last few years. Reference to the incidence of rickets and osteomalacia in Kangra Valley has already been made earlier. The work of Dr. D. C. Wilson and others has shown that both rickets and osteomalacia are common in cities of the Punjab where there is overcrowding and where people live under slum-like conditions. Besides, this work has also demonstrated the ill-effects of Purdah system which denies sunlight to women. This effect superimposed on a poor dietary inevitably leads to osteomalacia in women of child-bearing age, the severity of which goes on increasing with each successive pregnancy. Comparatively, osteomalacia is rare in Bombay Province and in the South. Skeletal deformities due to past rickets are marked in Kangra Valley. Although they are not so common in other parts of India, there is reason to believe that a fair proportion of infants and children suffer from rickets, probably of mild degree, particularly in the large towns and cities.

III. Lathyrism.—This has been classified under nutritional diseases for there is little doubt that its origin lies in the consumption

of *Lathyrus sativus* for prolonged periods. The disease is endemic in certain regions such as Behar, Eastern United Provinces, Central Provinces and certain regions of Central India. It comes in the wake of prolonged drought and famine. *Lathyrus sativus*, being a hardy crop, survives where others fail, and villagers from such regions have to subsist on this grain as the most important article of their diet. When consumed for a few months cases start occurring, the earliest cases have been known to occur within ten to eleven weeks of the commencement of the lathyrus diet. Unfortunately little is known about the treatment of this crippling disease.

General malnutrition.—Cases of general malnutrition resulting from multiple deficiencies are not uncommon. Nonspecific diarrhoeas, œdema and ascites for which no organic lesions are responsible, a malignant type of malnutrition affecting particularly the infants and children and macrocytic anæmias of pregnancy are only a few manifestations which arise from a chronic state of malnutrition in which more than one factor is involved.

The purpose of this article is not to describe the nutritional disease with respect to its ætiology, symptomatology, diagnosis and treatment. The intention is to draw attention to the fact that clinical conditions attributable to faulty and deficient dietaries are widespread in the country. It will be clear from the above, however, that accurate information even on this aspect of the nutrition problem is far from complete. The information given in the preceding pages has been collected from the reports of diet and nutrition surveys and studies on particular diseases such as, goitre, lathyrism, fluorosis etc. A fairly correct picture of nutritional disease in India would not only be of scientific interest, but if put together and presented in a proper form may provoke the authorities to take determined action for their eradication. Apart from the specific nutritional diseases above described, the prevalence of the background of malnutrition among the population increases its susceptibility to various infectious diseases like tuberculosis. It also decidedly worsens the prognosis in these latter diseases. Malnutrition is, thus, the major public health and clinical problem in the country today.

Facts and Fads about Food and Nutrition

RAO SAHIB T. N. S. RAGHAVACHARI,
Retired Public Health Bacteriologist,
(Formerly of the King Institute).

It was in the middle of the nineteenth century that for the first time the dietary requirements of men were stated in terms of nutrients. Voit¹ of Munich laid down 118 grams of protein, 56 of fat and 500 of carbohydrate (which together supplied 3000 calories) as adequate for the correct nutrition of a man engaged in moderate work (1870). American dietitians in 1903 considered this inadequate, and stated that standards should vary with conditions of activity and environment. In 1919 the Royal Society Food Committee, accepted the standards of Lusk of the Cornell University, based on calorimetric studies of the energy output of individuals of varying age and weight. The British Medical Association's Nutrition Committee, in 1933, issued a comprehensive report² on nutrition in which the energy requirements of man in relation to age, health, weight and working capacity were carefully assessed. The Committee suggested that protein requirements were a very important consideration and should provide about 15 per cent of the total calories. In the same year, the United States Department of Agriculture published the standards laid down by Stiebeling³ for calories, protein, minerals, (Ca, P, Fe) and Vitamins A and C and these were correlated to food materials. The latest is that of the National Research Council, Washington⁴ (1941) which has amplified Stiebeling's standards by the results of more recent researches. The recommended daily allowances for particular nutrients published by the N.R.C. have been designed as a guide to the planning of food dietaries, required for the maintenance of optimal health. The total calories, per head have been put down as 2775, the protein as 66 grams, Ca 0.91 g, Iron 12 mg. Vitamin A 4696 (I.U.), Vitamin B₁ 516 (I.U.), Vitamin C (Ascorbic Acid) 71 mg., Riboflavin 2.5 mg., Nicotinic Acid 15.5 mg. and Vitamin D 210 (I.U.). This is by no means an infallible or rigid guide. More recent dietary surveys have, however, revealed that excellent health can be maintained on diets supplying fewer calories, less protein and even fewer vitamins than the above N.R.C. standard.

It requires knowledge and circumspection to select the foods best suited for body-building and for the supply of energy. To obtain a diet that would ensure maintenance of health, the foods selected must include sugar, butter, fats and cereals for providing energy; milk, cheese, eggs, cereals, legumes and meat for providing building; mineral salts found in milk, fruits and meat for body-vitamins that occur in vegetables, fruits, milk, and vegetables; grain etc.; and finally roughage. The British Ministry of Food furnished the people of Great Britain during the war years, with

much good advice, from time to time, on food planning consistent with the prevailing difficult food position. In 1945 they published a *Manual of Nutrition*⁵ which contained detailed compositions of 'meals' to suit different classes of people (income-groups). These and similar manuals and standards are no doubt valuable guides but can never be considered the last word or the universal and rigid standards for human nutrition. From our knowledge of the living conditions of men, women and children it is becoming increasingly manifest that instruction in the essential elementary principles of healthful living should be an integral part of our educational system and that the solution of the problem of national nutrition depends on an adequate production and a fair distribution of food.

Fads and false ideas about food constituents.—Superstitious beliefs, ignorance and the absence of discerning and critical spirit, are mainly responsible for several queer and some absurd statements that are often made on the subject of foods. There are hundreds of people who indulge in fanciful experiments and with a persistence worthy of a better cause, start preaching the results of their experiments for the benefit and practice of others!! People who have very little experience and no special training in this field, are the most prone to dogmatize. In the commercial field, the art of advertisement has been deluging the world with a most surprising array of fanciful facts, based on imperfect evidence concerning the function of the human subject as a socio-physiological and aesthetic person. This type of propaganda has done immense harm and should be controlled early by legal enactments, particularly in those spheres where there has been a decided abuse of the art of advertising. There is, then, the insidious propaganda by some ill-informed and self-opinionated food faddists—and these are legion—which should be effectively countered. Amongst several false suggestions, one finds the statement that starches should not be eaten with nitrogenous foods because protein stimulates the gastric secretion, which these faddists assert, stops the digestion of starch. "The acid of the stomach only stops the enzymatic breakdown of starch (in the stomach), which is started in the mouth by the ptyalin of the saliva. To stop enzymatic digestion of starch in the stomach is merely to prepare the way for its further digestion in the intestine by means of the starch splitting enzyme, amylase. There is no incompatibility between any two starches and the delay in starch digestion in the stomach is 'quite physiological'." ⁶ There is one fallacy in this, namely, to digest starches the stomach juices should be alkaline. The salivary digestion of starch is continued in the stomach until the hydrochloric acid penetrates the saliva *cum* starch mass and destroys the ptyalin. This usually takes about half an hour after the food enters the stomach and by this time "80 per cent of the starch would have been converted to maltose,

Alkalinity of the gastric juice is not necessary, for no further digestion of the maltose takes place until the first part of the intestine is reached where the enzyme *maltase* is waiting to receive and convert the maltose into glucose. The pancreatic secretion does the rest and enables the (ingested carbohydrates) converted glucose to enter the blood stream in an assimilable state.

There are any number of myths and fads, concerning food. For instance, fish is stated to afford 'food for the brain' because of the rich phosphorus contained in it. Fish flesh has phosphorus no doubt but much more than phosphorus is needed to build brain tissue. Again, other foods we take, milk, cheese, whole cereals, eggs also supply phosphorus. Again, meat is considered essential for building muscle. There are millions of people who have never touched meat in all their lives and who enjoy robust health and maintain muscular vigour in abundance. And amongst habitual meat eaters there are a large number who are not in perfect health and vigour. And we find numerous publications, which condemn animal protein outright, and extol the virtues of vegetarianism to the skies! Many of these are typical of the food faddists. The lay public is ever ready to believe everything they read, without ever pausing to consider if they may not be half-truths at best. The advocates of meat-diet (as being essential for muscle building), emphasise the superiority of meat protein to vegetable protein. What is a protein? The best example is egg-white which is the only pure protein in solution in water. The egg-yolk is a mixture of proteins with fat and salts, as are also cheese and meat. The proteins are made up of numerous nitrogenous compounds or units closely knitted together, which are called amino-acids. Some 15 or 16 such units are usually found in a protein. These amino-acids get separated in the process of digestion and enter the blood stream through which they pass on to the various tissues of the body. The tissues have the power of selecting the amino-acid suitable for them, as different tissues require different amino-acids. The functions of the tissues determine the type of amino-acid needed. No single protein in our food contains all the amino-acids necessary for the building or repair of the body. The elements comprising the amino-acids are nitrogen, carbon, hydrogen, oxygen and sulphur. Our bodies have the power to synthesize from simple combinations of these elements certain of the required amino-acids. Such, as cannot be thus synthesized or built up have to be supplied by the protein-intake in our food supply. Proteins which contain all or nearly all the amino-acids required for body growth and repair are called 'complete' proteins, and these afford full 'biological value'. Recent researches have shown that the "proteins of maize and wheat" are well balanced with regard to amino-acids, required for growth and compare favourably with amino-acids of animal origin. "The nutritive value of proteins depends upon (a) the amount; (b) the digestibility; and (c) the biological value of the protein." ⁷ The

complete proteins are found in eggs, milk, cheese, nuts, and meats. In vegetable and cereal foods, incomplete proteins are found associated with a complete one. Safety lies, therefore, in using mixtures in our food.

The newer discoveries of specific nutrients and their metabolic consequence and the justifiable enthusiasm in such discoveries have to some extent distracted attention from the oldest problem in nutrition *viz.*, simple caloric inadequacy, with its attendant general malnutrition which is now widespread all over the globe. The tragedies of famine, pestilence and mortality that we have witnessed in recent years in Bengal and other places afford strong evidence of the exceedingly slender margin of safety. Caloric inadequacy generally involves a parallel protein deficiency in most cases. Any food shortage—and this has been fairly common and frequent in our country during the last five years and more—rapidly reduces such sources of protein as are usually available and subsistence depends almost entirely on rice, tapioca and other foods containing little or no protein.

It has been stated in the foregoing paragraphs that the protein moiety is made up of many different proteins and so deficiencies are sure to occur under food-shortage conditions. An example of unsuspected amino-acid deficiency and an inter-relationship with a vitamin has recently been investigated and brought to light in pellagra.⁸ It now appears that pellagra may result from a deficiency in either tryptophane or nicotinic acid and can be controlled successfully by either the amino-acid or the vitamin.⁹ This means that the protein requirement may be altered by either its tryptophane content or by the amount of the nicotinic acid in the diet. It is, therefore, quite possible that other similar relationships between the amino-acids and vitamins may yet be discovered! The protein requirement will, therefore, not be an independent entity. General protein deficiency may result from lack of a single amino-acid; adequate knowledge and careful scrutiny may result in hitherto undiscovered methods of supplying the missing factor at small cost!! Another very interesting and quite recent finding by Dr. Paul Cannon and his collaborators¹⁰ has stressed the importance of nutrition, particularly protein nutrition, in antibody formation and resistance to infection. This particular finding has, however, been made on the results of animal experiments and the exact significance and relevance of the observations to human caloric deficiency will have to be and are being studied in greater detail. Clinical experience in hospitals associated with prison camps in the Second World War shows that surgical procedures were often performed on persons in a deplorably bad nutritional state without unusual complications.¹¹ The evidence, though not conclusive either way, cannot be ignored particularly in India, where several environmental factors operate adversely to bring about a lowered vitality and tone in the major

portion of our population. Also be it noted that the absence of complications during surgical procedures on undernourished persons relate to soldiers living under controlled and necessarily better environmental conditions.

Fallacies relating to vitamins and the real facts about them. It is unnecessary at this stage to go into the history of the discovery of these accessory food factors. Sir Frederick G. Hopkins of Cambridge, McCollum and Davis of U.S.A., are the outstanding names in this field of nutrition. To date, some 17 vitamins have been distinguished and classified according to their different properties and modes of action.

Vitamin A is found in abundance in fish liver oil, and in fairly large quantities in milk, cream, butter, egg-yolk and liver; vegetable leaves that become green are good sources of this vitamin. Green vegetables and carrots do *not* contain the vitamin A, but contain its precursor, carotene. It has been computed that only 40 per cent of the carotene ingested (as vegetables and carrots) can be converted into vitamin A by our bodies. According to the most recent authorities a diet can be regarded as adequate if it contains 60 International Units of vitamin A for every 2.2 lbs. of body weight (or say about 4000 I.U. daily for an average sized adult and about 6000 I.U. for nursing mothers.)

The vitamin A content of milk, butter and cream is liable to vary with seasons and changes in the feeding of the cattle. It is liable to be destroyed by the application of *excessive heat*, such as we apply in our homes in boiling milk to give a rich caramelised taste, and melting the butter (already deprived of some of the vitamins in over-boiling the milk) into ghee. Our ghee is prepared at high temperatures usually from butter, derived from boiled and soured milk (curds) and further stored for several days till rancidity develops. So it would be idle to imagine that we are deriving plenty of vitamin A from our ghee and superboiled milk. Pasteurization is the ideal method for retaining the full biological and vitamin values of milk and its products. The raw flavour of such milk may not readily appeal to the taste of people who are used to well boiled milk, but the sooner all of us learn to shed our prejudice and dislike for it, the better will it be for national health; in open pans, nearly 25 to 30 per cent of the vitamin content initially present in the butter is liable to be lost. The vitamin A content of different samples of butter in South India has been estimated to range from 800 to 6000 I. Units per 100 grammes. It is also estimated that the vitamin A value of pure cow's ghee is between 1000 and 2500 I. Units while that of buffalo's ghee is considerably lower.¹² And we get in the market only buffalo butter and ghee of a very poor quality and often adulterated. "Vegetable

ghee or Vanaspathi made from various vegetable fats does not contain vitamin A¹³ and its wholesomeness to health has been a matter of controversy amongst scientists for sometime now and in spite of the Government of India's directives for speedy research no final decision appears to have yet been reached. This and similar products have been and are being, however, extensively used, without compunction in our hotels. As vitamin A deficiency is rather common in our country, great care is needed to ensure an adequate supply of this factor in our diets. Hotels and other eating houses should, therefore, also cooperate by using such food materials as would be wholesome and contain the essential primary and accessory food factors in abundance.

The term vitamin B complex came to be used subsequent to 1936, to denote a number of factors included in this group. For several years B₁ and B₂, the anti-neuritic and growth-promoting factors, were considered to be the only two covered by the term. When some six factors had been isolated, the alphabetical terminology was found to be no longer suitable and all vitamins with the exception of A and D have come to be designated by their official names. Sir Robert McCarrison¹⁴ found that deficiency of vitamin B₁ (Aneurin or Thiamin) was responsible for many of the common diseases of the intestinal tract, "and for the retardation of growth in infants and young children." Foodstuffs rich in this factor are wheat germ, rice germ, egg yolk, yeast, liver, beans, lentils, peas, and soya bean. Milk is a good source of this factor.

Vitamin B₂ or G (Riboflavin) which promotes growth and maintains a healthy condition of the skin in man and the coat in animals is found in fairly large amounts in raw liver, egg yolk, wheat germ, spinach, peas, cauliflower, mangoes and pulses. Milk contains riboflavin in large amounts, but as it is readily destroyed by exposure to light¹⁵ milk should be stored in a dark place. Our traditional and age-long custom of boiling and storing milk in mud pots in secluded corners is thus an ideal procedure.

Vitamin B (P. P. factor) Nicotinic Acid, was found by Elvehjem to cure pellagra in 2 to 3 days even when the patient showed signs of mental derangement¹⁶. Foods rich in this vitamin are yeast, liver and peanuts; it is present in small amounts in meat, eggs, milks, cheese, curds and buttermilk.

The vitamins of the B group still afford an ever expanding field of research. There are (besides the above mentioned three factors) several other factors down to B₁₂ which are of scientific interest and whose functions have, however, not yet been fully studied. A considerable amount of experimental work followed by practical clinical trials has been in progress in England and America. With regard to the therapeutic and food values of these factors, chief among which are folic acid, pyridoxine, pantothenic acid,

biotin and p-aminobenzoic acid. With the exception of pyridoxine, the rest are synthesized by the intestinal flora of animals and possibly also of man. Kon *et al*¹⁷ have recently demonstrated that the intestinal flora of man do manufacture vitamin B₁ (Thiamin) from potato starch. Eijkman¹⁸ found in 1897 that, whereas polished rice produced polyneuritis in fowls, the addition of potato starch prevented this happening. He was not, however, then aware of the story of vitamins; nor were our ancestors in India probably in possession of knowledge of the scientific concepts underlying the vitamins. But they advocated and practised the use of hand pounded rice for food, of par-boiled rice for *iddalies* and cakes, of cold rice soaked in clean water for one or two days (rich in yeast) with curds for breakfast, of vegetable kosumbaris (salads) containing cucumber, green gram (uncooked) coriander leaves, green chillies and fresh lemon juice for side dishes, and sprouted grains (green and Bengal grams specially) for incorporating in the main dishes. Had we of the recent generations had the good sense and relish for these wholesome vitamin-rich foods, there would have been no need to buy and use expensive and comparatively less efficient, medicinal preparations of the various vitamin varieties. Better and more robust health would have been the rule amongst our young men. It is, however, never too late to mend and the new order *can* change yielding place to the old and be productive of real and lasting good.

Vitamin C (Ascorbic acid) is the scurvy preventing factor. It is found chiefly in certain vegetables and most citrous fruits. The fresh green leafy vegetables, (cabbage, Brussel's sprouts, tender greens, drum stick leaves, gonkoora leaves, spinach, lettuce, and green coriander leaves and germinated pulses, are particularly rich in this factor. Amla, or nellikai (Indian gooseberry) is a very rich source. Its botanical name is *Phyllanthus emblica*, Linn. "A single fruit of this tree is equivalent in vitamin C content to two oranges." Potatoes, because of the frequency and quantity in which they are used in diet, are a food source of vitamin C. Oranges, grapes and lemons are, however, the finest sources. Tomatoes, plantains, pineapples, pappaiyas, guavas, (country varieties), pomegranates, pomeloes, and strawberries also contain this vitamin in fair amounts. Heat quickly destroys this vitamin. The main purpose of germinating the pulses (red gram and mochai) with red earth and water is to increase the vitamin C content and perhaps also the iron content, before storing them for future daily use. Sir P. Hehir, I.M.S.²⁰ wrote in 1917, referring to the severe epidemic of scurvy in Kut-el Amara (1916) that the failure to use coarsely milled, germ-troops while the Indian troops who used coarse grain and germinated pulses escaped. "Had the Indian dhal been moistened and allowed to germinate in the sun it would have contained the

vitamin to protect them against scurvy".²¹ This possibility was discovered in 1916 by Dr. H. Chick of the London Lister Institute.

A good deal of controversy has centred round the human adult requirement of vitamin C. The generally accepted English standard is 50 mg. per day of ascorbic acid; the American (U.S. N.R.C's.) figure is 75 mg. per day. Quite recently we had the preliminary report²² of the Vitamin C Sub-Committee of the Accessory Food Factors Committee of the British Medical Research Council, based on carefully controlled experiment carried out for nearly 2 years at the Sheffield University. Twenty human volunteers took part in the research. This report states that there is no evidence to show that the American standard has any advantage over the 30 mg. which was the amount recommended by the Technical Committee of the League of Nations (Nutrition Section).²³ A daily intake of as little as 11 mg. per adult, was found adequate in the experimental subjects. The report concludes by saying that it would be almost impossible to provide 75 mg. per day to every adult in the whole population and that such a large amount was really unnecessary for health. Tests in the Toronto University Pædiatric Clinic have revealed that ascorbic acid given in the form of orange juice was much more effective than synthetic ascorbic acid tablets. As small an amount as 5 to 7 mg. of vitamin C will prevent the development of scurvy but 20 to 30 mg. are recommended for optimal growth and health.²⁴ The cheapest, easiest and absolutely dependable method of securing full vitamin value in our diets is to have recourse to sprouting of pulses, and the daily use of green leafy vegetables and salads.

Vitamin D controls the calcium and phosphorus metabolism in the tissues. A deficiency of this vitamin D produces rickets in children and osteomalacia in adults. Children who constantly play in the sun do not usually suffer from rickets to the same extent as those who live most of the time indoors. This is manifestly due to the former obtaining a good supply of vitamin D through the sun's rays. Certain rays in the ultra-violet end of the spectrum act on a substance in the fat of the human skin and produce the vitamin D. The *rationale* of this was discovered in 1922, when it was found that the effect of irradiation of the animal could be equally well produced by irradiating its diet deficient in vitamin D. It was also found that the capacity to become *activated* resided in those portions of the food which contained fats. Fats, such as gingelly oil, olive oil, cotton seed oil which are inactive in themselves become markedly antirachitic on irradiation. Rays of the sun with wave lengths between 280 and 310 millimicrons will alone activate the sterols present in these oils. The relationship between the mineral salts, the fatty acids in foods and in the skin and ultra-violet rays of the sun has now been scientifically established. In this connection again, the traditions and ancient customs of South India have to be mentioned here. The practice

of expressing the oil out of gingelly seeds (after letting the latter soak in water for some time and then dry in the sun) in country oil mills (chekku) driven by bullocks, and allowing the expressed oil in large mud pots to remain exposed to the sun's rays (in our court-yards) for 3 days before finally storing it in the house, has manifestly been designed to irradiate it in bulk. In modern life, there is hardly time for the expressed oil to be stored or exposed to the sun. It is used up in the culinary art within a few hours of its leaving mechanised or even country mills. Then again, our young brethren of this century have totally discarded oil baths—a most valuable institution designed by our well meaning and better informed ancestors, to provide comfort, vitamin D and also radiant health to the body. It was only after Sir Robert McCarrison pointedly drew the attention, some 20 years ago, of the readers of his simple treatise on *Food*²⁵ to the virtues of taking oil baths regularly that some of us reverted back to oil baths and derived benefit therefrom. The irony of it all! A Westerner to re-discover for us the great value of a healthy practice discarded by merely out of sheer cussedness!!

Vitamin E (Tocopherol).—Evans of California (1936) and his collaborators²⁶ isolated three different vitamins E—*alpha*, *beta* and *gamma* tocopherol. (Greek Tokos—childbirth, phero—to bear). This vitamin comes into play only when about a third of the gestation period has passed. Nutritional muscular dystrophy usually appears as a result of a deficiency of this vitamin E. The best form in which to supply the deficiency is whole wheat germ. Dry lettuce contains this vitamin in fair amounts—cocoanut oil, also contains it.

Vitamin K (coagulation factor). Dam and Schonheyder²⁷ discovered this in 1936. It is found in green leafy vegetables, tomatoes, egg yolk and liver. Synthesis is influenced to a great extent by sunlight. Cabbage leaves, cauliflower, spinach, carrots and potatoes contain this vitamin. It is believed to have a profound influence on the regulation of the processes of blood coagulation.

General.—From the foregoing account it will be seen that each vitamin has specific relations to certain structures of the body:—*A* to epithelial and nerve tissues; *B* to the gastro-intestinal tract, skin and nervous systems; *C* to the matrix which binds the cells of the body; *D* to bones and teeth; *E* to the reproductive system; and restricted to these; each may assist or collaborate with the others in maintaining the health of the skin, teeth, bones, digestive tract, or the nervous system and all are closely interlinked with the other food constituents.

The diet of the large majority of the Indian people is grossly deficient when judged by scientific standards. The main dietary

defects in India are a too high carbohydrate and too little protein intake. Ours is a land of poverty. The advent of rice mills and the consequent extensive use of polished milled rice, have deprived us of the much needed vitamin B₁. This is particularly marked in S. India. Home-pounding is still in vogue and practised by a large per cent of the people in Assam, Bihar, Orissa and Hyderabad. The Hindu population of S. India is largely vegetarian and the higher castes strictly so. Their consumption of milk and milk products is deplorably low. Vegetables and fruits are not eaten in such amounts as would provide the requisite quantities of the accessory food factors. Wholesome culinary practices have been abandoned in favour of vitamin destroying and mineral-salts-depleting methods. Healthful habits of personal hygiene and cleanliness have been relegated to the scrap heap. Parental control over the habits of young children and adolescents have considerably slackened and resulted in the younger generation following their own whims and tastes in the matter of food, oil baths and dress. The high incidence of tuberculosis is the direct result of these indiscretions. These facts point to the imperative need for immediate reform and improvement.

The most urgent needs for our country as a whole at present, are to increase dairy farming (milk production) and to place agriculture on a sound, rational and scientific basis. Careful and perhaps also skilful planning is needed for the large scale production and equitable distribution of the food so produced. JAI HIND.

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Clinical Signs and Symptoms of Early Malnutrition

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EVERYDAY we waste energy in our daily routine work. The wastage is according to the quality and quantity of work. We therefore need to recoup the wasted energy and maintain the body health. This is to be balanced in our daily diet which must have a caloric value sufficient to provide the requirements of the basal metabolism, the stimulating action of the food stuffs, loss of energy in the excreta and the needs of muscular work. So our diet must have adequate amounts of proteins, fats, carbohydrates, water, salts and ample vitamins. We need between 3000 to 4000 calories daily. Light work less calories (3000 C) and hard work more calories (4000 C) no work (2500 C). Our normal diet therefore must have at least 100 gms. of proteins, 100 gms. of fats and 500 gms. of carbohydrates, besides minerals and vitamins. Proteins for wear and tear and growth of the body and fats for energy. At least 50% of the fats should be animal fats which contain Vitamins A and D. Vegetable fats are deficient in vitamin A and D but do contain vitamin E.

Carbohydrates are the main parts of the diet and are necessary for the proper oxidation of fats.

Minerals—one ounce of common salt is essential in any form in our daily diet.

Vitamins are necessary for the life and normal functioning of an individual.

Water is essential as all the biochemical reactions take place in this fluid.

So whatsoever diet we take we must consider its nutritive value, otherwise we are liable to fall a prey to malnutrition at any stage. Malnutrition is not a disease itself but it is a stage arising from all or any of the following conditions:

(1) Insufficient caloric intake (causes under nutrition or inanition).

(2) Insufficient intake of one or more of the essential nutrients (causes minor or major deficiency disease).

(3) Inadequate absorption or utilization of nutrients (may be secondary to disease or may cause under or malnutrition in any form with special features superadded by the presence of the disease).

The diagnosis of malnutrition remains largely a matter of subjective impressions. A normal and well nourished man means that he has suffered no disease which has left any permanent disability.

In stature and build he has no postural defects and no skeletal disproportion or asymmetry. The spinal furrow is not completely flattened at any point, and when the arms are raised above the head there is no winging of the scapulæ. When he touches his toes the vertebral column forms a smooth curb without definite flattening at any point. His skin has the bloom of vigorous health and is smooth, soft, elastic and slightly moist to the touch. There is a moderate amount of subcutaneous tissue over firm and well-developed muscles. He has no potbelly, no palpable spleen or liver. His eyes show a clear cornea surrounded by a white smooth sclerotic with little visible vascularisation and during inspection there is no undue photophobia and watering. His vision by night and by day is excellent. The teeth are regular with firm and healthy gums. The tongue is neither too large nor too small, with no more than a slight fur, and there is no discolouration, fissuring, stripping of the epithelium or enlargement or flattening of papillæ, the mucous membrane well coloured and healthy. Blood examination shows normal hæmoglobin content and red cells of normal appearance and size. The appetite is good and bowel function regular. The normal, healthy and well-nourished man gives an impression of alertness and vitality which is the opposite of apathy and listlessness. On the other hand, a malnourished man shows impairment of endurance, vitality and resistance to disease, slow recovery from disease or even impaired morale. Diagnosis is one of considerable difficulty and can be made with assurance only after full investigation of previous history and dietary and full consideration of differential diagnosis. At a later stage depletion of nutritional reserves reaches a stage when more or less specific signs and symptoms appear. Again the picture is not necessarily clearcut. If caloric intake has been so low as to cause inanition specific deficiency lesions may be masked or residual signs from malnutrition in the past may persist or confuse the tissue. The presence of infectious disease may also modify the picture. The onset of malnutrition is generally very insidious, the lack of dramatic manifestations may lower standards of judgement so that an impaired level of health is taken as normal. In the final stage the picture becomes that of severe inanition or one of the classical deficiency diseases. We are here concerned only with the earlier clinical picture which may be met. All clinical impressions should be confirmed by finding out whether the diet has been such as to cause the type of malnutrition suspected and all causes of secondary malnutrition should be looked for.

Hereunder are some clinical signs and symptoms of early malnutrition described by bodily systems and region as covered by a clinical examination. Most individual signs and symptoms are themselves valueless for diagnosis. The whole picture must be assessed in relation to nutritional and other factors. Where no specific nutritional cause is mentioned the sign or symptom described should be considered as non-specific sign of malnutrition.

General signs. 1. *Loss of weight*: Muscle wasting and loss of subcutaneous tissue, undue looseness of skin.

2. *Loss of energy*: Lassitude and chronic fatigue, less output of work, failure to recuperate normally after hard work, loss of strength, poor muscle tone.

3. *Impaired morale*: Loss of normal high spirits, curiosity and contentment, lack of mental application, nervousness and irritability.

Skin.—1. Healthy skin is elastic, slightly moist and has a glossy texture. In malnutrition the skin may be dull and sand paper like or show crazy pavement changes.

2. *Phrynoderma* (follicular hyperkeratosis) is a noninfective papular hyperkeratinization of hair follicles, found on extensor surfaces of the extremities specially but may be generalized. It is a chronic, persistent condition said to be due to vitamin A deficiency.

3. *Desquamating dermatitis* of a fine branny nature found on scrotum or nasolabial region (in patches) suggestive of riboflavin deficiency.

4. *Ulcerative skin lesion* on account of lack of vitamin C.

Mouth and pharynx. 1. *Tongue changes*: In the early stages there are stripping of the fur and the epithelium of the edges and tip, leaving these areas smooth, dark red and magenta in colour and with flattened papillae. Centre of the tongue covered with thick white fur. In the second stage tongue completely stripped of fur, smooth and slightly swollen with lateral indentations caused by the teeth. The papillae flattened. There is fissuring also which may be longitudinal or transverse, fairly deep and raw. Soreness and burning on taking food is a complaint in some cases. Above signs in association with angular stomatitis, cheilosis, branny scrotal dermatitis, hoarseness, pain on swallowing and eye signs are suggestive of riboflavin deficiency. In nicotinic acid deficiency the tongue may be small, glazed and scarlet in colour with shallow longitudinal fissures.

2. *Angular stomatitis*. Consists of transverse fissures at the corners of the mouth with maceration and incrustated exudation but in early stages there may be simply a white sodden epithelium without fissuring—indication of riboflavin deficiency.

Cheilosis.—Redness, desquamation and finally ulceration along the mucus membrane of the lips. Soft palate also seen injected with a sharp demarcation line between the red soft palate and the white hard palate, indication of riboflavin deficiency.

3. *Scorbutic gingivitis*.—Hyperplastic condition of the gums, bleeding on pressure. The gum tends to project in the interdental

spaces forming characteristic red buds best seen on the buccal side of the gums. Later on marked swelling of the gums occurs due to the hemorrhagic exudation. It should not be mixed with pyorrhœic gingivitis which is not due to any vitamin deficiency. In early scurvy small petechial hemorrhages may be present on the palate. Anæmia, debility and subcutaneous or intramuscular hemorrhages are likely to be present.

4. *Sore throat*: Dysphagia, hoarseness and aphonia—symptoms of riboflavin deficiency.

Eyes.—1. *Xerosis conjunctivae*.—Vitamin A deficiency. Lack of lustre distributed irregularly as small patchy areas. This lack of lustre appearance is easily detected if the eye is kept exposed to air and light for a few seconds. There is wrinkling of the conjunctiva specially marked in the palpebral region, due to lack of elasticity of the conjunctiva. At a later stage Bitot's spots may appear. These are white patches resembling a flake of foam or of plaster on the surface of the conjunctiva due to hyperkeratinizing metaplasia of the epithelium and accumulation of seborrhœic excretion.

2. *Superficial keratitis*:—Curable by riboflavin. The patients complained of a burning sensation in the eyes with foggy vision, photophobia and watering. There may be circumcorneal injection and corneal opacities may appear.

3. *Corneal vascularization* (peripheral vascularization via the limbus, in centripetal fashion):—Riboflavin deficiency. This sign may arise even in the absence of any deficiency.

4. *Anæmia*:—The lids are retracted and examined for evidence of anæmia.

5. *Night blindness*:—Vitamin A deficiency. But night blindness can arise from many other causes, both physical and psychological.

Gastro-intestinal system.—1. *Anorexia*:—Vitamin B₁ deficiency.

2. *Diarrhoea*.—Nicotinic deficiency.

Blood and vascular system:—Nutritional anæmias—may arise as a result of iron deficiency (hypochromic anæmia) or of deficiency of the specific hæmopoietic principle or principles present in liver and in marmite (Macrocytic anæmia).

3. *Oedema* due to hypoproteinæmia may arise in any form of severe malnutrition.

4. *Cardiac vascular disturbances*.—Vitamin B₁ deficiency. Tachycardia, arrhythmia.

Nervous system.—1. *Peripheral neuritis* with stocking and glove anaesthesia, calf muscle tenderness, muscular weakness and depression of reflexes (sign of early beri beri).

2. *Mental changes*.—Diagnostic features of pellagra. In pellagra "men previously strong, courageous and enduring become shaky, weary, apprehensive before clinical pellagra can be diagnosed. Sub-clinical pellagra or beri-beri may be an important factor in the fighting morale of an army."

Mental and physical inefficiency succeeded by irritability, agitation, depression and restriction of activity are earliest signs of B₃ deficiency.

Respiratory system.—Bronchitis has been said to be a result of vitamin A deficiency.

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Aetiology of Early Vitamins-Deficiency States

DR. M. B. DAVER,
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VITAMINS are produced in human body either in almost negligible amounts or not at all. It is a known fact that they do not furnish energy and are not utilised for building organism. Vitamins are chemically heterogeneous organic compounds; and their functions differ immensely.

There has been great advance in research work on vitamins and it can be said that clinical medicine cannot keep pace with the advancements; thus we know a larger number of different vitamins, but few human deficiency syndromes. This is due to the fact that avitaminosis in human beings is due to not one single vitamin deficiency but multiple deficiencies.

Aetiology of deficiency states

The conditions of vitamin deficiency are either due to primary causes or due to some other secondary conditions.

Primary deficiency means, deficiencies arising solely by consuming inadequate diet.

Secondary or conditioned deficiency means, vitamin deficiency occurring in spite of taking adequate diet, due to some other conditions.

Primary deficiency.—The factors responsible for inadequate intake are poverty, ignorance in cooking and preparation of food due to certain fads, or eccentricity. From the nutrition and diet surveys carried out, it can be safely said this inadequacy is mostly due to first two causes, namely poverty, ignorance and improper cooking.

Inadequate food intake will supply inadequate nutritional factors needed for body requirements. This we will call as "dietary inadequacy", but there is failure to absorb and utilise nutritional factors in adequate amount to meet body requirements and this we will call "nutritional deficiency."

Nutritional deficiency occurs when essential factors of nutrition do not reach the tissues. At this stage, early symptoms of deficiency arise.

When in spite of taking adequate diet, "Nutritional deficiency" occurs, we call it secondary deficiency. These secondary factors are of greater importance in deficiency states and should not be ignored.

These secondary factors may be due to:—

- I. Increased body requirements.
- II. Increased excretion.

III. Interference with absorption.

IV. Increased destruction.

Increased requirements.

Vitamins are required in larger quantities in :

- (1) Increased body activities, strenuous physiological effort.
- (2) Pyrexia and delirium.
- (3) Bright sun-shine, excessive winds etc.
- (4) During therapeutical measures adopted, *e.g.* excessive fluid intake, high carbohydrate diet, giving of glucose parenterally.
- (5) Administration of single pure vitamin.

It is true that adequate diet and nutritional intake should supply increased body requirements. But under certain conditions, metabolic requirements are increased and these conditioning factors require increased amount of vitamins. Strenuous efforts, hard work, even strong emotion raises metabolism 5 to 10 times above the basal level. (Best and Taylor, 1943). The same is true in cases of pyrexia.

Deficiency symptoms can be provoked by sun shine, excessive wind and other environmental influences.

Sunshine precipitates the symptoms of pellagra and it is ascribed partly to the excretion of B vitamins in sweat (Cornbleet *et al.*, 1943) and partly to damaging effects of sun-shine.

Pregnancy and lactation provoke deficiency state and well known ailments are neuritis, toxæmic conditions and beriberi. Either this is due to increased requirements or damage to liver from pregnancy toxæmia, impairing liver functions and thus interfering with utilisation and storage of vitamins of B group.

Therapeutic measures—such as—fever-therapy, parenteral administration of large amount of glucose, high carbohydrate diet increase the demand for vitamins (Spies *et al.*, 1938).

Repeated infusions of glucose may wash out water soluble vitamins of B group out of tissues; and the resulting oedema or water logging is attributed to deficiency of vitamin B₁.

Administration of pure single vitamin. The question whether large doses of pure single synthesized vitamin of a group the same group, or other factors is not yet fully understood and recognised. It has been reported that if pellagrins are treated with Nicotinic Acid—the symptoms of vitamin B₁ and riboflavin deficiency become more manifest. Lehmann and Nielson (1934) have drawn attention to the fact that if a patient is treated with large doses of vitamin B₁, sign of pellagra may come up; so also signs of riboflavin deficiency may become more pronounced after large parenteral doses of vitamin B₁.

It is very often seen in practice that if large doses of one single vitamin is given, partly abortive cases of other factor of the vitamin become more manifest, in other words "sub-clinical latent deficiencies within the vitamin B group may often become manifest after energetic treatment of the obvious deficiency" (Leilver, 1947). Thus single pure vitamin should be used with care and caution and it is a better policy to make use of vitamin B complex. For this purpose liver extract may be advocated as it contains a factor which liberates members of the vitamin B complex.

Increased excretion

Increased excretion of vitamins are found in :—(1) Polyurea and (2) excessive sweating.

Polyurea may be due to *diabetes mellitus*, or due to excessive intake of fluids to cause polyurea for urinary infection or in sulphonamide therapy—such polyurea may cause increased excretion of vitamins.

Excessive sweating in hot countries is also a factor which causes deficiency condition of B vitamins. After heavy physical exercise, some of B vitamins is detected in sweat (Cornbleet 1942).

Such increased excretion of vitamins may lead to malnutrition though there are very few authentic observations on this.

Interference with ingestion

The factor arises in : (1) Anorexia ; (2) gastro-intestinal disorders ; (3) neurasthenic conditions ; (4) restrictions due to diseases ; (5) disproportion of food factors, protein, fat, carbohydrate.

Disease may interfere with adequate food intake. During convalescence also, food intake is very poor. Gastro-intestinal disorders may cause anorexia, nausea and vomiting. Gastritis or gastric ulcer may place restriction on food intake and consequently on intake of foods containing vitamins.

Slimming or reducing diets are usually very short of B vitamins.

Due to economic conditions, there is preponderance of carbohydrate in diet over proteins and fats, which are expensive articles of food. Such relatively low protein and fat and high carbohydrate proportion is also responsible for improper ingestion of vitamins.

Interference with absorption

Factors responsible for this may be summarised as : (1) Achlorhydria ; (2) gastro-intestinal disorders ; and (3) therapeutic measures.

Absorption may be interfered with due to anatomical and physiological changes in intestinal tract. The reduction in the

absorbing surface, due to tumours, intestinal tuberculosis, etc.; or hyper-motility of the tract or even fashionable lubricants used as purgative may interfere with absorption and may be instrumental in causing vitamin deficiency.

In diarrhoea the hyper-motility of intestinal tract and reduction of the absorbing surfaces, interfere with absorption. How diarrhoea brings about sprue, pellagra, riboflavin and vitamin A and D deficiencies is well-known. Achlorhydria also interferes with the absorption of ascorbic acid and vitamin B₁.

The habitual taking of liquid paraffin as purgative is a very bad habit, as it interferes with the absorption of fat soluble vitamins A and K (Swell 1939).

Biliary disorders also will interfere with absorption of fat soluble vitamins as bile salts play an important part in their absorption.

Destruction of vitamins

Factors responsible for these are: (1) Achlorhydria; (2) sulphonamides; and (3) toxic doses of minerals.

In the presence of achlorhydria, assimilation of ascorbic acid is interfered with. In the acid gastric juice vitamin B₁ is stable but if antacids or alkalies are added vitamin B₁ is completely destroyed.

It is a known fact that sulphonamides reduce the therapeutic action of nicotinic acid.

Najjar and Holt (1943) and others have shown that after giving sulphaguanadine or sulphasuxidine, the vitamin bio-synthesis of Vit. B₁, riboflavin and nicotinic acid in the intestine was substantially reduced or completely inhibited. Loss of this function of bio-synthesis should be considered as equivalent to destruction.

Thus deficiency state cannot be judged by mere calculations of food intake. A person may be taking adequate diet and if yet deficiency state arises—consideration of various factors which interfere with ingestion, absorption and utilisation, or conditions which increase the requirements of vitamins and which increase their excretion or destruction should be carefully considered. This is very essential from treatment point of view, and to understand the case.

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Evaluation of Nutritional Status

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It need hardly be stressed that the prosperity, if not the very existence, of a modern State depends to a large extent on its capacity to provide the agricultural services, industries and defence organizations with adequate and efficient manpower. Consequently existence of large groups of population in a sub-normal state of physical fitness constitutes a definite handicap in any country. Such were some of the considerations which broadened the outlook of the health workers in the more progressive countries beyond the long concept of three-fold public health programme: (a) Provision of pure and wholesome water supply; (b) safe disposal of sewage; and (c) prevention of epidemics and other crowd diseases.

In an individual free from any of the specific diseases or disabilities, the 'state of nutrition' is almost synonymous with 'state of physical fitness' or 'state of health'. It must be understood that the term 'health' in the sense it has been used implies a positive attribute. Every one would agree that it is easy to grade or classify the absence of health than to measure it as a positive factor. It has to be admitted that the medical profession in general has always acquiesced in the negative conception of health, in other words, if a man was not actually ill he was, and still is, regarded by the profession to be as healthy as he was ever likely to be. The fault does not lie with the profession alone because in the past too much stress has been laid on the study of disease and nothing has so far been done to study and analyse health. Our ignorance of the subject is still so deep that we can hardly claim to know or define scientifically what health is. Recently the World Health Organization has declared in its constitution that "health is a state of complete physical, mental and social well-being and not merely the absence of a disease or infirmity". The definition is no doubt a positive one but admittedly broad and vague.

In a well documented and thoughtful monograph on health published about 9 years ago by a member of the medical profession, a reference has been made to the paradox that whilst owners of automobiles, sewing machines and typewriters periodically send their machines to the workshop for overhaul and repairs with a view to prolong the life of these machines, the owners themselves rarely consult a doctor unless actually ill. But supposing the public was so health-minded that they visited their doctors every six months, what would happen? It is not difficult to imagine the embarrassing position of a medical man if he has to report every six months either on the state of positive health or nutritional status or physical fitness of the members of his clientele who possess no discernible signs or symptoms of disease. By the force of circumstances he would have to be vague in his report. A

repetition of such vague reports even when couched in technical terms, would act as a wet damper on the enthusiasm of any health-minded individual.

At one time states of mal- and under-nutrition were separately defined. Mal-nutrition was evidently implied to signify a condition caused by intake of the food not of the proper quality whereas under-nutrition was supposed to have been caused by quantitative deficiency in the diet. It was a distinction without a difference and has consequently been dropped. The evaluation of nutritional status of any population groups can be made indirectly by the study of dietary survey and by the comparative study of mortality figures and life tables. For indirect measurement of the nutritional status, three types of examinations have to be depended upon I. Anthropometric; II. Physiological including biochemical; and III. Clinical.

I. Anthropometric. From time to time certain anthropometric indices such as Quetelet's, Livi's Index Ponderalis, Pignet's Coefficient de Robusticite or Von Pirquet's Pelidisi, were devised. Lately came to the field the A.C.H. index of the American Child Health Association. These indices were used for a while and ultimately discarded as none of these physical standards were helpful in gauging the state of nutrition in any group of population within reasonably precise limits. The averages for height and weight figures are still in use. The ratio between height and weight may not often be a perfect yardstick to assess the state of nutrition or physical fitness, but its usefulness within certain limits cannot be denied. If a group of children of any particular age or a group of healthy adults recorded a lowered average for weight, the inference would be that their standard of nourishment or nutritional status is low. In the occupied zones of Germany and Austria the nutrition units of the Allied occupation forces drew their inferences about nutritional status of the people of different age groups by recording the average weight of respective groups.

II. Physiological dynamometers have been used from time to time to measure the capacity of strength in an individual. But these gadgets have also been proved to be unsatisfactory; an individual himself records different readings on different days and different months. Power of endurance has also been used as a yardstick of measure of physical fitness and nutritional status. Children have been asked to hang from horizontal bars and the endurance in terms of time determined by a stop watch. This index also has not been very helpful in scientific investigations. This index also has one time was also thought to be an approximate index of an individual's physical potentialities. It is not suggested that all or some of these methods which are still used, should be discarded and abandoned in nutritional surveys but a more intelligent approach may be made, and when interpreting the results of such records, the limitations inherent in each of them should not be lost sight of.

Estimation of the concentration of some of the ingredients of blood may be used for assessment of health or nutritional status. The determination of hæmoglobin in gms. per 100 ml. of blood is a very useful technique. The protein concentration of plasma has also been used as a fairly reliable index of nutrition. Serum phosphatase concentration is another measure, particularly applicable to subjects where rickets are suspected. With the gradual development of comparatively easier techniques, concentration of the different vitamins in the blood and their excretion in urine, greater attention is being paid to such biochemical yardstick. But the standardisation of such methods, establishment of norms and their comparative efficiency in the assessment of nutrition on a large scale have yet to be worked out.

III. Clinical.—For the estimation of nutritional status clinical examination still ranks as the method of choice. The texture of the hair, elasticity and appearance of the skin, lustre of the eyes, the thickness of the subcutaneous fat, the development of musculature, and above all, the general appearance, do give a clue as to the state of nutrition or physical fitness of the subjects examined. It is admitted that reliance on this method is unscientific, because even the physicians are said to differ in their opinion on what constitutes 'good', 'fair' or 'poor' grading of nutrition. Another flaw of the clinical rating is that during large scale surveys the same subject may be graded as 'fair' and 'poor' or as 'good' and 'fair' on successive examinations by the same medical examiner. This is not at all surprising in the examination of borderline cases. One often hears about deficiency diseases or stigmata caused by deprivation of one or more vitamins in human subjects. Some of the well-known stigmata of avitaminosis are xerophthalmia, angular stomatitis, phrynoderma, vascularisation of cornea, hemerolopia etc. But large scale investigations in Bengal and Bihar during the food shortage of 1943 and the experience in prisoner of war camps within recent times have proved that the percentage incidence of the stigmata of avitaminosis is no index of the state of nutrition in any group of population. Extreme degrees of poor nutrition may not be associated with the evidence of such stigmata.

So far no reliable index or group of indices have been evolved for evaluation of nutritional status in human subjects with any reasonable degree of precision. Scientists engaged in the researches on the physiology of man and his well-being have not paid enough attention to this aspect of the subject. But the task is by no means an easy one, because human organism is not a mere conglomeration of different kinds of tissues and the power of adaptation to environments is beyond all proportion to other potentials.

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Vitamin Deficiency Diseases in Infancy and Childhood

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IN a recent survey of school boys (10 to 20 years old) in a large High School in Madras, out of 506 boys examined by the writer 54 had marked conjunctival xerosis *i.e.*, 10% vitamin A deficiency. 1 had hemeralopia to crown it, 3 had dermal xerosis, 2 had keratosis pilaris, and 8 had angular stomatitis *i.e.*, 1½% Arabinoflavosis.

Mal nutrition is the main picture amongst the poorer children appearing in the hospitals and it is only advanced cases with serious intercurrent diseases like pneumonia, typhoid, or diarrhoea, that are admitted; and all this is necessitated by want of accommodation. Sixteen children were admitted into the Government Royapettah Hospital, Madras, under the writer's care during the last 27 months with markedly advanced nutritional disorders and the following is a statement about the same:—Cancerum oris 1; nutritional oedema 7; mal-nutrition 3; vitamin B deficiency 1; pellagra 1; beri-beri 1; I. These small figures are representative of the quality and not the quantity of the cases under mal-nutrition attended to in the Hospital. Signs of vitamin deficiency could be discerned at the out-patient department every day and in almost every patient.

There are today about 31 vitamins and provitamins. The student of medicine is concerned only with those valued in human nutrition.

Vitamin A is an unsaturated alcohol related to carotene (yellow pigment occurring in the body and colouring certain neutral fats and also in company with chlorophyll) and is not synthesised in the system. Present in milk fat, eggs, fish liver oil, and young green plants. Animals convert carotene into vitamin A. Daily requirement of infants is about 2,500 I.U. and 4,000–6,000 I.U. for older children. Vitamin A deficiency causes xerophthalmia, night-blindness, and keratosis. Hypervitaminosis A in rats causes tenderness of limbs and fractures.

Vitamin B complex is now valued on account of Aneurin, Riboflavin, Nicotinic Acid, Pyridoxin, Biotin, Folic Acid, Para-aminobenzoic Acid, and Pantothenic Acid.

Aneurin is extracted from rice polishings and also chemically synthesised. Found in cereals and yeast. The human body is unable to synthesise or store it in any amount. Daily requirement is probably 1–1.5 mgm. or 500 I.U. for children. Deficiency produces beri-beri. Children, pregnant and nursing mothers require a specially high vitamin B₁ intake as do patients suffering from diabetes

mellitus, increased thyroid activity and febrile illnesses, and individuals undergoing prolonged muscular activity, and lastly in the writer's opinion all rice eaters.

Alcoholic peripheral neuritis and cases of neuritis (as in cirrhosis liver, pellagra) may be due to B₁ deficiency. Aerodynia in rats and Pink disease in children deserve B₁ therapy. Wernicke's encephalopathy benefits from vitamin B₁.

Riboflavin (a pigment found in milk, egg, yeast and plants) causes by its deficiency oro-lingual ulceration (sprue, pernicious anaemia, pellagra and corneal vascularisation). Daily requirement 1 to 3 mgm.

Nicotinic Acid—daily requirement 20 mgm.—(constituent to carbohydrogenase systems). This PP vitamin rich in yeast and liver, can be synthesised outside the body. It is beneficial in Vincent's angina, intermittent claudication, cerebral thrombosis, Meniere's syndrome, mental confusion, typhoid fever (Sarma, A. V. S. 1943).

Pyridoxin is used in anæmias, muscular atrophy and dystrophy, and Parkinsonism.

Biotin deficiency in man includes a scaly dermatitis, a greyish pallor of the skin, atrophy of the lingual papillae and disturbed erythro-poiesis according to Sydenstricker.

Folic acid has glutamic acid and para-aminobenzoic acid residues in its molecule; and is found in green leaves, and is synthesised by intestinal flora. Folic acid is remarkable in the successful therapy of nutritional macrocytic anaemia of pregnancy, sprue and Addisonian anaemia. The writer has found it efficacious in coeliac disease and in the refractory anaemia of chronic interstitial nephritis.

Para-aminobenzoic acid is useful in the treatment of rickettsia infection, rheumatic fever, and also human achromotrichia.

Vitamin C occurs naturally in citrus fruits and tomatoes. The daily requirement for infants is 20 mgm. and for children 50–100 mgm. It is prophylactic and curative for scurvy, and lowered by pasteurisation, by standing, by further heating and by dilution. Hypervitaminosis C does not exist. Many chronic illnesses may be due to a degree of vitamin C subnutrition (latent scurvy). It is probably important in the etiology of pneumonia, rheumatic fever, and delayed wound healing (peptic ulcers included). The writer has a conviction that vitamin C deficiency is important in the genesis of hepatic cirrhosis and it has also been used by the writer (1939) in controlling the later vomiting of typhoid fever.

Vitamin D is anti-rachitic, occurs naturally in fish liver oils and can be prepared by irradiation of inactive sterols found in Nature, and is synthesised by human skin by action of sun's rays. D₃ is

suggested as the naturally occurring vitamin D. At least 10 pro-vitamins D are known. Daily allowance of 500 to 800 I.U. is sufficient to prevent rickets. This vitamin is prophylactic and curative of rickets, and is used in tetany to raise blood calcium, and in rheumatoid arthritis, hay fever, and lupus vulgaris. Heavy doses are known to be dangerous and produce renal calculosis and failure.

Vitamin E (prepared from wheat germ oil) is said to be useful in muscular dystrophies.

Vitamin K is the prosthetic group of enzyme thrombin (Lyons) or that it along with a protein carrier acts as an oxido-reductase system. Support to the former theory lies in the colour reactions characteristic of 1:4 naphthaquinones given by the colour reactions tryptin digestion. It is capable of biosynthesis by the products of in the bowel from after a few days after birth. The newborn requires 1 to 2 μ g. per day. Menaphthone is more used today. Indications are: (1) Spontaneous subcutaneous and internal hæmorrhages or the liability to bleeding during and after operation (due to lack of bile in the gut); (2) hæmorrhagic diseases of the newborn. Prophylaxis is in giving to pregnant mothers and thus raising the plasma prothrombin value in the infants. The hypoprothrombinaemia occurring in ieterus gravis neonatorum, anemia neonatorum and hydrops congenitus also responds to the administration of vitamin K; (3) urticaria; and (4) coronary heart disease, thrombosis. (Dicoumarol antagonises vitamin K).

Vitamin P (Citrin, Hesperidin) is said to be useful in allergic purpura.

Vitamin Deficiency Diseases

- I. Rickets (spasmophilia). Late rickets (bony changes in coeliac, renal and hepatic diseases.)
- II. Xerophthalmia. (Night blindness; dermal xerosis).
- III. Infantile scurvy.
- IV. Pellagra.
- V. Beri-beri.
- VI. Pink disease.
- VII. Nutritional œdema.
- VIII. Conditioned deficiencies as in fevers and protracted dietetic regimes.

I. Rickets.—Rickets is a general metabolic disease due to deficiency of vitamin D, characterised by skeletal changes especially near the epiphyses and tendency to catarrh of mucous membranes.

Etiology:—Rickets is worldwide in distribution and is said to be rare in the tropics and Iceland. Children of dark races in the temperate zones and premature babies are more prone to rickets. Poverty is an important factor.

Rickets may be caused by (1) Errors of diet, deficiency of vitamin D and excess of cereals (antagonising vitamin D). (2) Lack of sunlight (ultra-violet light). (3) Defective hygiene and confinement. The predisposing factors are: (a) Age 6 to 18 months. Rickets is never congenital and unknown before 4 months; (b) commoner in winter and spring in British Isles; (c) climate and locality (temperate zones and large cities); (d) artificial feeding; but rickets is known amongst the breastfed; (e) prolonged breastfeeding (later than 1 year). This is a factor prevailing in South India, and is to be attributed to poverty in the poorer classes, if not ignorance and custom. Late weaning with rice and pepper water is also a factor; and (f) rapidity of growth in babies.

Pathology:—The calcium phosphate content of bones is low, about 21% instead of the normal 63%, and owing to lack of calcium the bones bend. Changes are seen in the long bones near the epiphyses; the end is enlarged with widening and unevenness of the epiphyseal line (normally 2 mm.), due to irregular and excessive proliferation of the cartilage cells. Also sub-periosteal bone formation is interfered with. Normal ossification stops and marrow becomes vascular. The deficiency of calcium and phosphorus in the bone is probably due to failure of absorption from the intestine. Normal blood calcium and phosphorus are 9 to 10 mgm. and 2 to 5 mgm. per 100 c.c. respectively. In rickets either calcium becomes low (low calcium rickets with tendency to spasmophilia, i.e., laryngismus stridulus tetany, and convulsions) or phosphorus become low (the more common, low phosphorus type). Occasionally both may get reduced to below normal. When the product got by multiplying Ca and P values as mgm. per 100 cc. below 30 means the presence of rickets (Howland); while rickets is probably present when the figure is below 40. The normal figure is 40 to 60. Calcium phosphate is normally deposited in growing bone owing to the action of an enzyme phosphatase formed in the bone. Phosphatase is present in the intestinal mucosa, kidneys and blood. The normal plasma alkaline phosphatase figure is 3.5 to 13.5 units (King and Armstrong method). In rickets the plasma phosphatase is increased during the active stages of the disease. The liver may be enlarged and fatty, and the spleen fibrous. As rickets improves the calcium and phosphorus contents of the blood rise and phosphatase content falls.

Symptoms:—Rickets in England is fast getting to be a rare condition in recent times.

The onset is gradual; manifestations are rare before 4th month. The infant is plump, becoming restless and irritable with head sweating.

The skeletal changes are characteristic. The anterior fontanelle is large and closes later (normally at 1½ years.) Rachitic "Parrot's nodes" are seen as the bossing of frontal and parietal

bones and with the sagittal and coronal sutures the hot cross bun appearance becomes obvious. "Craniotabes" elicited early in rickets (particularly over lower parietals) is due to osseous rarefaction, and is appreciated also in hydrocephalus and osteogenesis imperfecta. Rickety head becomes square contrasting with the globular head of hydrocephalus.

Delayed eruption of milk teeth and poor calcification of the permanent ones are seen in rickets.

"Rickety rosary" (*vide Fig. 1-a*) due to beading of ribs at costo-chondral junctions, and "pigeon-breast" type of thorax due to side to side fattening of chest wall and "Harrison's sulcus," the horizontal groove on each side of the chest above the hepatic and gastric levels are typical appearances of the rachitic chest. Some children though not suffering from rickets have soft and springy chest wall.

Kyphosis and scoliosis of spine (dorsal) and pinched pelvis with forward projection of the sacrum are noteworthy; the latter being a dangerous risk in female children.

Enlargement of the epiphyses of the long bones (typified early in the lower end of ulna) is common finding. (*vide Fig. 1-b*) X-ray of the wrist is ideal for the study. "The X-ray changes consist of a splaying out and hollowing of the ends of the long bones, while the line of ossification is irregular and fluffy instead of being an even transverse line, and the bone behind the epiphyseal line is rarefied. In cases of some standing one or more transverse lines of more dense bone can often be seen lying parallel and close to the epiphyseal line as though the rachitic process had made previous attempts at healing." W. Sheldon.

Bending of long bones occurs giving bandy legs. Arm bones may bend in crawlers. Anæmia (hypochromic) and mild leucocytosis are seen. Loss of muscular tone and greater ligamentary laxity are responsible for delay in sitting and walking. Pot belly is due to high cereal diet and fermentation. Spleen is felt enlarged.

Spasmophilia includes a triad: Tetany, (*vide Fig. 1-c*) laryngismus stridulus, and convulsions seen in low calcium rickets. Tetany is seen in carpo-pedal spasm and when it is latent is recognised by Chvostek's sign, Trousseau's sign, and Erb's sign.

Laryngismus stridulus (child crowing) is due to spasmodic closure of the glottis. Convulsions resemble epilepsy.

Complications of rickets are respiratory; bronchopneumonia; and diarrhoea.

Diagnosis:—Early symptoms of rickets, i.e., restlessness, head-sweating, and irritability are common to many conditions. Differentiation from congenital syphilis (age under 3 months), and scurvy (9 months age-period) is easy. Early signs are craniotabes and ribbeading in rickets; these are earlier than the radiological signs.

Larger anterior fontanelle, bossing of the skull, delayed dentition, and bending of the bones are later findings. The "physiological" curvature of the legs seen in the 2nd year of infancy is different from the rachitic "bandy legs" which are prominent in the lower third of tibiae.

Anaemia and laxity of ligaments need not suggest blood dyscrasias and amyotonia congenita respectively.

Early radiological signs diagnostic of rickets are seen in the X-ray of the wrist, particularly at the lower ulnar end.

Prognosis:—Rickets by itself is not fatal. Bronchopneumonia or diarrhoea are dreaded complications. Prognosis of infectious fevers, particularly measles, pertussis, and influenza, is worse with rickets.

Bony deformities persist if treatment is delayed until the child is 3 or 4 years old, when stunting of growth results with kyphosis, deformed thorax and pelvis, and with bow legs or knock knees. Harrison's sulci and skull bossing persist long.

Treatment consists in administering vitamin D (Fish liver oil, concentrates like Adexolin, or Halibut liver oil, irradiated ergosterol or exposure to ultraviolet light.) Natural source of vitamin D, like fish liver oil, is preferred. Overdosage is avoided as calcification of kidneys and arteries is feared. If cod-liver oil is not tolerated (diarrhoea, vomiting, allergy), concentrates are administered. Cod-liver oil and malt are reserved for older babies. Calciferol m. 5 thrice a day will serve well. Ultraviolet light exposures are given graded and systematically, 1 minute to 15 minutes daily or on alternate days. Healing is checked by fortnightly X-raying of the wrists and improvement is shown by the denser and regular epiphyseal line.

Calcium and phosphorus administration may be unnecessary if adequate milk supply or use of butter, egg-yolk, green vegetables are ensured. Iron is given for anæmia. For tetany, parenteral calcium therapy is urgent in addition to vitamin D.

After a fortnight's treatment the child may be allowed up and about. Splinting prevents further bowing of legs. Natural correction of bony deformities occurs in the year following treatment for rickets and therefore orthopaedic surgery may be resorted to if necessary after sufficient interval.

Prevention of rickets lies in ensuring sound breastfeeding during the first nine months of child's life. Rickets is common in children reared on condensed and sweetened milk or a regime rich in cereals (rachitogenic) as after weaning in poorer children.

Two teaspoonfuls of cod-liver oil or ten drops of halibut-liver oil as also open air life serve a good prophylaxis.

The vulnerability of premature babies and convalescents to rickets will be remembered.

Vitamin-resistant rickets is a rare form of rickets and needs heavy vitamin D per day (500,000 I.U. daily) and hypervitaminosis D is a danger.

Late rickets.—Infantile rickets neglected and not treated is not late rickets which was appreciated after the 1914-'18 war in poor factory workers, and ascribed to low mineral and vitamin intake. Osteomalacia, the adult counterpart of rickets, is still prevalent in India in purdah women.

Late rickets is exemplified by coeliac and renal rickets.

"According to Parsons, coeliac rickets does not occur unless the coeliac disease has been severe, and recovery has been delayed until the child has reached seven years of age."—W. Sheldon. The bone changes consist in beading of ribs and enlargement of epiphysis, genu valgum, and even multiple fractures treatment consists in giving ultraviolet light therapy, and administration of vitamin concentrates and Ca and P.

In renal rickets bony deformities of infantile rickets are present, and particularly progressive knock-knee and dwarfism. Signs and symptoms of renal fibrosis (thirst, polyuria, sallow and dry conjunctures, pale urine of low specific gravity with occasional casts and traces of albumin) are all present.

Osseous rarefaction in renal rickets has been ascribed by Langmead and Orr to hyperparathyroidism and consequent osteitis fibrosa, as a result of low serum calcium and nephritis. Serum phosphates are raised (even 14 mgm. per 100 cc.) as the kidneys are unable to excrete them. Parsons attributes this disturbance in Ca:P ratio as the cause for failure of osseous calcification. Tetany is unknown because of co-existent acidosis. Prognosis is hopeless. Uraemia ends the picture, Graham and Oakley improved bone condition by administration of large doses of alkalis along with vitamins D and A. Light orthopaedic apparatus may be needed for bony deformities. (*vide Fig. 2-b*).

Hepatic rickets.—Children with infantile hepatic cirrhosis recovering from the disease by the 3rd year also develop rachitic changes in the skeleton and this has been elaborated by the writer (1939). The cause is in the cirrhotic liver not functioning in the storage and supply of fat soluble vitamins as also in the lack of food rich in vitamins and minerals for the growing convalescents. (*vide Figs. 2-a & 2-b*).

II. Xerophthalmia.—Xerophthalmia is due to vitamin A deficiency. The lesion lies in increasing epithelial hypertrophy and opacity of the cornea, progressing sometimes to ulceration and destruction. White patches appear on the palpebral and corneal

conjunctiva and with conjunctivitis. Night blindness may also be associated.

Treatment:—5 minims of Halibut liver oil twice a day. Good diet with milk and fruit. Anti-rachitic treatment given, if necessary. Prepalin is vitamin A injectable and used for urgent cases, but is very painful and threatens to suppurate.

III. Infantile scurvy.—Barlow described the morbid anatomy of this condition. (*vide Figs. 3-a & 3-b*).

Etiology:—Diet: Lack of vitamin C which is contained in juice of fresh oranges, lemons, grapes, swedes, tomatoes and also in potato. Orange juice prevents scurvy. Breast milk also is a source of vitamin C. Milk loses its vitamin C on boiling or pasteurisation.

Vitamin C is isolated and synthesised now.

Age: Between 6th and 18th months, and mostly at the 9th month cases of scurvy are detected.

Symptoms:—These are mainly of hæmorrhages, (*vide Figs. 4-a & 4-b*) under the periosteum of the long bones; into the gums; and occasionally into the viscera. The attitude of the child is frog-like and limbs appear motionless.

The hæmorrhage under the periosteum is large enough to cause a swelling above or below the knee. The muscles may be hæmorrhagic. The lower end of the femur (X-ray picture typical) or tibia and the distal end of the radius are common sites for the hæmorrhages. Epiphyseal separation may occur.

Hæmorrhages into the gums (turn purple) may be seen in children with teeth which then loosen and even fall out. Gums bleed. Bleeding may be on the palate or inside the orbit.

The chest shows scorbutic beading of the ribs due probably to hæmorrhage at the ribs with cartilages; and the sternum appears pushed backwards.

Hæmaturia is early and characteristic. Cutaneous and visceral hæmorrhages are rare except occasional blood in the stools.

Hypochromic anæmia is present with pallor.

Growth ceases in scurvy and is typified by the radiological dense white line of scurvy at the edges of long bones due to calcification with no corresponding growth. Separation of periosteum due to subperiosteal hæmorrhage (shell-like bone formation after 3 weeks) may be seen. Eventually with cure normal appearance is restored.

Diagnosis:—Scorbutic beading, hæmaturia, and bleeding into gums mean only scurvy. Differentiation from anterior poliomyelitis, sarcoma, syphilitic epiphysitis, acute rheumatism, and teething is easy enough not to cause doubt.

Prognosis:—Good. Associated pyelitis, diarrhoea or rickets add gravity. Frank scurvy clears with vitamin C administration in a couple of days; and large subperiosteal hæmorrhage clears in 2 or 3 months.

Treatment: Warm soft bed and gentle nursing. Weight of bed clothes is removed by a cradle. Baths are omitted till all tenderness clears.

Patent and dried foods are stopped. Fresh and unboiled milk is advised in England. Fresh fruit juice (orange, lemon, tomato, grape) is to be given daily; at least potato pulp, or potato cream.

50 mgm. of vitamin C is necessary per day. Initially intravenously 50 mgm. may be given and later orally, the same 50 mgm. daily. There is no risk of overdosage with C.

Sedatives (Chloral Hydras gr. $\frac{1}{4}$ to 1 at bed time) may be used for the first few days.

Prevention:—Breastfeeding is ideal. Vitamin C is added to the dietary of the artificially fed babies as orange juice; that of one fruit daily is sufficient.

The need of vitamin C is greater in rheumatic fever. The writer has shown that vitamin C is useful in stopping the vomiting of typhoid fever and again as a prophylactic against infantile hepatic cirrhosis. U. S. Rao supports the writer. In the Jain diet on religious grounds, frank and latent scurvy at every age can be seen; and this is particularly so in nursing mothers and children, both sharing purdah very unfortunately.

IV. Pellagra.—Pellagra is caused by deficiency of nicotinic acid in diet, and is characterised by dermatitis (erythema and thickening of exposed areas of skin), digestive disturbances, and progressive degeneration of the nervous system. Oedema may be present in children.

Onset is insidious. Irritability, headache, and giddiness, unsteady gait, tremor of the extremities, paræsthesia or anaesthesia of areas of skin appear. Digestive disturbances (sore tongue, gingivitis, vomiting, and diarrhoea) may arise.

The skin lesions affect the parts exposed to the sun: face, neck, hands and legs. Later desquamation and thickening of the skin set in with brown staining. (Vide Figs. 5-a, 5-b & 5-c). Course is punctuated with relapses, particularly in spring. Emaciation, dementia, and paralysis lead to death.

Treatment.—Early stages respond to feeding on high protein diet (meat, eggs, wheat and casein) and yeast (15 gm. daily). Exposed skin must be protected from sun. Nicotinic Acid (Nicotinic Acid Amide preferred) has been used with excellent results (daily 100 to 200 mgm).



FIG. 1-a.
Rickets
(Rickety Rosary).
[Vide page 432.]



FIG. 1-b.

The clinical photograph on the left top shows the typical syndrome of the "rickety rosary," pigeon breast and Harrison's sulcus. The X-ray photograph on the right top shows the lower ends of the Ulna and Radius which are fluffy with osteoporosis. The X-ray photograph of leg bones below shows (1) increase in width and translucency in the medullary cavity of both bones, (2) irregular growth and ossification of epiphysis and diaphysis, and (3) an ill-defined and widened proliferative zone.

[Vide page 432.]



FIG. 1-c.
Carpo-pedal spasm.
[Vide page 432.]



FIG. 3-a.
Cirrhosis Liver.



FIG. 2-a.
Renal Rickets.
(Note knock-knee).
[Vide page 434.]



FIG. 2-b.
Renal Rickets. X-rays wrists.
[Vide page 434.]

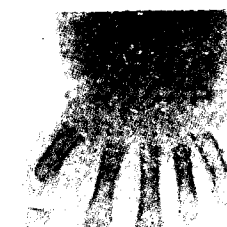


FIG. 3-b.
X-ray wrist of same child.
[Vide page 435.]

Vitamin Deficiency Diseases in Infancy and Childhood

L. J. S. SARGENT



FIG. 1.

FIG. 1. (Sargent). A child with rickets.

FIG. 2. (Sargent). A child with rickets. The separation of the epiphyses of the femur is visible.



FIG. 2.

(Sargent). Rickets.



FIG. 3.
Pollagra.



FIG. 4.
Pollagra (Pollagra) with
pollagra.



FIG. 5.
Pollagra (Pollagra) with
pollagra.

Vitamin Deficiency Diseases in Infancy and Childhood

L. J. S. SARGENT



FIG. 6a.
Pink disease. (Shining light).
[Vide page 137.]



FIG. 6b.
Pink disease.
(A miserable baby).
[Vide page 137.]



FIG. 6c.
Pink disease. (Biopsy of skin. Low power).



FIG. 6d.
Pink disease. (Biopsy of skin. High power).
[Vide page 137.]

Vitamin Deficiency Diseases in Infancy and Childhood
A. V. S. SARMA



FIG. 1.
Pink disease (Trophic
ulcer on right foot).

(Vide page 417.)

FIG. 2.

FIG. 8.
Nutritional oedema.

(Vide page 438.)



FIG. 8.

Nicotinic acid administered by the writer to typhoid patients controlled the alimentary and mental symptoms and particularly prevented mumps and diarrhoea and delirium.

V. Beri-beri.—The disease occurs in children (particularly in those breast-fed by beri-beri mothers) under 2 years, and is characterised by polyneuritis. It is due to deficiency of aneurin in the diet. The virulent form is in babies 2 or 3 months old who are seized with convulsions terminating fatally. This experience is common amongst the very poor, and the beri-beri infants come into the hospital only to die there. In the protracted cases restlessness, breathlessness, and aphonia are present. Generalised oedema may appear and death may be sudden due to cardiac dilatation and failure.

Treatment is dietetic and in incorporating milk, eggs, wheat flour, or barley. White rice must be abandoned. Mother's diet must be balanced and rich in vitamins. Yeast, a teaspoonful thrice a day, is allowed to mothers. Aneurin could be given by injection in emergency or orally ordinarily.

VI. Pink disease.—This is called also erythroedema polyneuritica or aerodynia; occurs in young children and is characterised by extreme irritability and misery, pinkness of limbs, and a variety of sensory, motor, and trophic nervous disturbances. It is either a deficiency disease or infection; the question is unsettled. The writer feels that streptococcal infection of the throat (allergy as in active rheumatic carditis or rheumatic chorea) can precipitate in a child, particularly deficient in the diet for some time with regard to vitamins of B complex, a disease; and this is really discussed by writer as pink disease.

Pink disease occurs between 3 months and 7 years, sometimes in epidemic form. Symptoms are characteristic. An upper respiratory catarrh starts and the child is hypotonic, miserable and unable to see the light. The extremities look swollen and pink, but feel cold and bear sweating and rashes and trophic lesions. Tendon reflexes are lost or diminished. Marked tachycardia is present. The writer has found that the pulse rate is the same rapidity whether the patient is alert or asleep, as it is in inactive rheumatic carditis.

Mortality is about 5 per cent. Recovery is slow and may be benefited by balanced diet and administration of vitamins of B group and liver.

Examples:—Two cases of pink disease were studied by the writer. A biopsy of the skin was possible in one and in the other the trophic ulceration of the foot is noteworthy. (vide Figs. 6-a, 6-b, 6-c, 6-d & Fig. 7).

VII. Nutritional oedema.—This is the condition denoting a diffuse pale oedema occurring in illnourished children particularly

apparent with intestinal helminthiasis and after a diarrhoea. (*vide Fig. 8*). Serum proteins are low. Treatment consists in giving high protein, high carbohydrate, and low fat diet. Injections of crude liver extract or vitamin B complex are beneficial. Orally concentrates of vitamins, and minerals, particularly calcium and phosphorus, are given.

Recovery is slow. Death is common from a silent bronchopneumonia or recurrent diarrhoea and exhaustion.

VIII. Conditioned deficiencies.

Vitamins.

(1) Vitamin A.

(2) Vitamin D.

(3) Vitamin B₁.

(4) Vitamin C.

(5) Nicotinic Acid.

(6) Folic Acid.

(7) Riboflavin.

Conditioned deficiencies.

(1) Protracted fevers exemplified in typhoid fever.

(2) Bronchopneumonia complicating rickets.

(3) Heart failure, postdiphtheric paralysis, acute toxic polyn neuritis.

(4) Rheumatic fever, fevers and toxic conditions, early hepatic dysfunction and vomiting.

(5) Typhoid fever and cancrum oris.

(6) Coeliac disease, diarrhoeas and anaemia in chronic nephritis.

(7) Nonspecific orolingual ulceration in fevers and diarrhoeas.

The writer is indebted to Dr. P. R. Kalyanasundaram, M.B., B.S., Superintendent, Government Royapettah Hospital, Madras, for all the help and encouragement received in the preparation of this communication.

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Nutritional Background in Psychological Inadequacies in Children

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Introduction.—Much attention has been directed in recent years to the part played by food and nutrition in the production of various types of deficiency diseases and possibilities of prevention, but no reliable studies are available, at any rate in India, either as regards its incidence or outcome in psychological inadequacies. Psychological inadequacies in children in India are often the result of poor nutrition, though in a number of cases it is found that the psychological inadequacy has no direct relation to poor nutrition. When problems of food and nutrition are being discussed throughout the length and breadth of India, it is of prime importance to determine with some degree of accuracy the part played by poor nutrition in the production of psychological inadequacies.

Incidence.—It is common to come across large variations in the health of children in the different socio-economic groups. In a few of the socio-economic groups the incidence of ill-health is assuming large proportions. Remedial measures ought to be taken on a nation-wide scale in order to lower to a low level the decidedly evil effects particular socio-economic conditions exert on human life and health. It is too true that health is affected to a greater or less degree by all social conditions.

During the last five years 670 children (of school and pre-school ages) were brought to the psychological laboratory, Mental Hospital, Bangalore, considered either mentally or psychologically inadequate. The behaviour of such children was considered inadequate as they were characterised by mental inferiority specially observed in their inability to adjust to environmental conditions. The problems for which these children had been referred included instances of delinquent conduct, undesirable habits, premature fatigue intellectual difficulties, poor school adjustments and other anti-social tendencies. Out of 670 children examined and treated, in 270 of them, i.e., 40%, there was a history of poor nutrition.

The subject matter of this paper is drawn from a study of these cases. But it is unscientific to deduce from this figure the actual incidence of psychological inadequacy due to poor nutrition in any single community. (*vide Tables I and II, page 440.*)

Unless it is impossible to manage with such children they are not usually referred either by the parents or the teachers. Secondly not every case of inadequacy though unmanageable, is referred due to the stigma generally associated with such a reference to a clinic in a Mental Hospital. Hence not even a rough approximation of its incidence in relation to the general juvenile population might be

attempted. However the incidence of psychological inadequacies in children is of such magnitude that it merits serious consideration.

TABLE I.

Number of psychologically inadequate children examined: all cases.

Year	COMMUNITY					Total
	Hindus	Muslims	Indian Christians	Anglo Indians	Others	
1944	70	20	18	6	6	120
1945	74	23	22	8	8	135
1946	72	28	20	11	10	141
1947	82	30	33	10	7	162
1948	63	15	18	9	7	112
Total	361	116	111	44	38	670

TABLE II.

Number and percentage of psychologically inadequate children associated with poor nutritional status.

Year	COMMUNITY					Total
	Hindus	Muslims	Indian Christians	Anglo Indians	Others	
1944	41	2	4	—	—	47
1945	46	3	6	1	—	56
1946	32	4	10	1	1	48
1947	52	6	6	2	1	67
1948	39	4	6	2	1	52
Percentage	58%	16%	29%	13%	18%	

In the practice of child psychiatry, it is common to come across three groups of children:

- (1) Children who are afflicted with organic disorders.
- (2) Children who exhibit definite signs and symptoms of psychotic behaviour.
- (3) Children who are neither organically afflicted nor are considered insane. These children who suffer from psychological difficulties constitute about 50 to 60% of all those children who throng the clinics.

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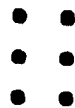
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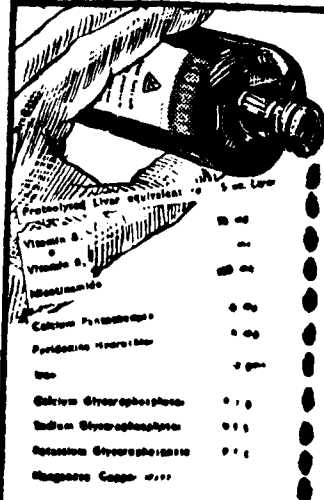
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General Principles of Nutrition

1. Basal energy requirement.—We human beings to which every class of society belongs cannot work without a supply of energy, which is got in the form of food. Such a supply of energy is required to serve two essential purposes in man. (1) To maintain the body heat; and (2) to sustain the action of the vital organs even when a man is asleep.

The amount of energy necessary for these vital processes when a man is resting is generally known as the basal energy. This basal energy requirement is calculated on the basis of the height and weight of a man. For example, a man five feet ten inches in height, and weighing 12 stones has a basal energy requirement of about 1·80 thousands of calories a day. It is customary to employ the term calorie when talking about the basal energy requirements. A calorie is the amount of heat energy required to raise the temperature of thousand grams of water through one degree centigrade.

It is very fair to assume that when a working man or a child is allowed only just about enough food to meet the basal energy requirements, he usually loses weight. Food which is just sufficient to keep him alive in a resting condition, is never enough for him to execute his professional duties. According to some observers (unpublished works) the majority of our countrymen in the middle and low income families, live and work on a diet, the energy value of which is less than two thousand calories a head per day. Naturally there is a tendency for their weights to go down. This has the undesirable effect on the physical standards of the nation. For example, a man originally weighing 13 stones and 5 feet 3 inches height would require a basal energy of about 1·82 thousands of calories. Due to poor diet and low vitamin content, his weight may go down say to 8 stones, with no alteration in his height. His basal energy requirement would then be about 1·48 thousands of calories. This again may go down in the course of a few months. Thus it is possible for the man to get thinner and thinner. The physical standard of a nation, comprising of such individuals, is bound to be poor. We all know ordinarily the diets of most of the working people in India, especially those in the mills, factories, Government offices, and other semi-industrial and semi-agricultural concerns, consist of poor and inadequate rations and the amount of work they turn out is exceedingly poor. The deplorable inefficiency of our men, compelled to live on poor rations, can neither be overlooked nor under-rated.

It is possible to assume that both as a group and as individuals children in these families are psychologically handicapped by their socio-economic and professional status of their elders. A diet with adequate nutrition cannot be had cheaply. Adequate nutrition is therefore fundamentally an economic question depending on the financial resources of the family.

II. Mal-nutrition, mal-nourishment and under-nourishment—the ideal diet. A diet necessary to preserve mental and bodily health should consist of energy-yielding substances like proteins, fats, carbohydrates, besides certain minerals and organic substances called vitamins.

Malnutrition is a state of reduced health caused by malnourishment or under-nourishment. Malnourishment refers to a defect in the quality of food rather than its quantity. It is due to ignorance or carelessness in selecting the right foodstuffs. Under-nourishment, on the other hand, is due to a defect of quantity as well as of quality and is usually the result of poverty as well as ignorance in the selection of foodstuffs.

The ideal diet must have sufficient energy value to maintain the body heat and also enable a man to perform various psycho-motor tasks. These psycho-motor tasks vary enormously in different persons, being comparatively slight in persons leading a sedentary life and raising to a maximum in persons engaged for many hours every day in very heavy responsible work. The energy of the body and the nature of the profession. The whole subject of Hindu classification of foodstuffs into *Satvika*, *Rajasa*, and *Tamasa*, is still untouched by modern students of research in the field of Nutrition. A healthy mixture of *Satvika*, *Rajasa* and *Tamasa* qualities is what the Hindus aimed at.

According to the energy value a diet adequate for a man should yield at least 3,000 calories, a little less for women and children.

Inadequacy Symptoms Associated with Mal-nutrition in Children

The diagnosis of psychological inadequacies in children associated with mal-nutrition was based on the following symptoms:

1. *Loss of weight*:—The weight of these children was invariably found to be below average and in a number of cases it was a diminishing figure. Along with this there was a corresponding loss of appetite. It looked as though a habit for poor appetite was being formed in them and efforts on the part of elders to make them eat more were futile.

2. *Chronic fatigue*:—It looked as though these children were born tired. Their exhaustion was marked and they fatigued very easily.

3. They usually complained of unlocalised somatic-aches and pains.

4. Dizziness and faintness.

5. Low grade resistance to infection.

6. Disturbed sleep in the nights.

7. Poor circulation—cold and clammy hands and feet.

8. Bad teeth.

Aetiology:—Out of 670 children examined and treated, in 270 (Table II vide page 440) i.e., in about 40% of the cases, the usual aetiological factors associated with mental abnormalities, namely, heredity, physiological epochs, physical diseases, or mental shocks, were of less significance. It was found that their psychological inadequacies was the resultant of poor food and mal-nutrition.

As already pointed out these children were definitely psychologically handicapped by their sociological background. More than 1/3 of them lived in very poor homes and neighbourhood and nearly 1/3 came from homes where the conditions were marginal. In a few homes, there was extreme poverty and neglect. All these are associated with poor nutrition.

That there is widespread under-nourishment among school children coming from the middle and low income families cannot be doubted. These children of school age are so physically and mentally inadequate that they are not able to derive the desired benefit from the State education. A good bit of money spent on educating them is a waste. The medical inspection of schools should make provision for assessing the nutritional status of children attending schools, and plan remedial measures to improve their nutritional standards. In the absence of other adequate and reliable tests, the following index (Pirquet's index) i.e., $\sqrt[3]{\frac{\text{weight} \times 10}{\text{sitting height}}}$ may be usually employed in assessing the nutritional status of school children. (Here the weight is expressed in grams and the height in centimeters). If the ratio falls below 0.95 the state of nutrition is regarded as inadequate.

Experimental Findings

Pre-school and school children considered inadequately nourished were investigated psychologically and their performances in the various mental tests recorded.

An attempt was made to persuade the parents and guardians to feed the children with one pint of milk daily. Milk alone of all the foodstuffs is rich in good protein, minerals and all the vitamins. The Hindus are fully justified in pinning their faith to milk rather than any other foodstuff.

The unique character of milk in human dietary has been emphasised by leading authorities on health and nutrition. It is the only food which comes to us *naturally* from a living body. It is a vital fluid, almost a living food. Milk is food—the only perfect food.

The addition of 1 pint of milk to the diet every day caused the greatest improvement of all in the average weight and height of these children. After a period of six months the children gained 0.52 lb. a month in weight and 0.21 inches in height. Exceptionally

poor children who could not afford even this pint of milk were also tested. They also showed gains of 0.42 lbs. in weight and 0.17 inches in height.

TABLE III.

Significant inadequacies as per Gesell's 'Mental Capacity Scale'	Present in the number of children	
	Before treatment	After treatment
A.—Developmental defects.		
1. Cannot lift head when lying on back	25	1
2. Cannot follow slowly—moving bright light. (4 to 6 months old children).	28	6
3. Cannot sit up with slight support	41	6
4. Cannot sit up alone (6—9 months)	44	7
5. Cannot walk alone (9—14 months)	49	10
B.—Problem Solving Tests.		
1. Cannot pick up spoon from table	20	3
2. Cannot express recognition of strangers as being different from familiar faces (6—9 months).	22	3
3. Cannot grasp and pull down ring dangled on string just above head.	26	6
4. Cannot lift inverted cup to recover cube after seeing it placed under a cup (9—12 months).	30	8
5. Cannot pile three blocks on top of each other to make a stable pile after seeing one do it.	36	8
6. Cannot place cubes in cup when told (12—18 months)	36	7
7. Cannot make attempts to turn knob when wanting door open.	31	6
8. Cannot point to two parts of the body (eye, nose, mouth) 18 months—2 years).	32	4
9. Cannot make drawing of a circle if done	40	10
10. Cannot obey propositions (2—3 years)	41	12
11. Presented with several cubes and cups—cannot put one block into a cup and two blocks in a cup.	42	9
12. Cannot carry out three commissions without asking further (3—3½ years).	40	10
13. Cannot answer reasonably two out of three questions	41	8
14. Two oblong cards are divided by a diagonal cut into two triangles, child presented into two triangles and asked to put them together so that they look like the figure (3½ to 4 years).	42	9

As it was expected the psychological inadequacies of these children minimised themselves to an appreciable extent. Improvement was shown by 75% of these children. However, it does not

mean that in those children where improvement had been recorded they had become free from their psychological failings only due to milk. Other environmental factors might have also had their parts in correcting the behaviour and improving the performance scores of these children. It does however mean that the children had been immensely profited by this line of treatment as no other treatment was given to them during this period.

This investigation to measure objectively the psychological status of children before and after feeding them with an extra pint of milk amply proves the assumption that a large percentage of such psychological inadequacies among children may be prevented by insisting on adequate nutrition for them right from infancy.

Out of 270 children tested and treated, 125 were of pre-school age ranging in age from a few months to 4 years. Gesell's pre-school tests of mental capacity were employed with the following results. (*vide Table III, page 444*).

A very detailed examination of above 125 pre-school children after treatment showed that more than 75% of them had improved in their psycho-somatic status. Out of these 125 children, no less than 60 of the children had unemployed parents and their inadequate psychological status must have been largely the result of mal-nutrition. They were anæmic, majority of them having a hæmoglobin index of 60% or less.

School Age Children

The following tests were administered to the 145 school age children before and after treatment:

(1) *Tapping test*:—To measure psycho-motor efficiency.

(2) *Memory test*:—To measure memories span for shapes, digits and pictures.

(3) *Span of apprehension*:—To determine the number of letters that can be grasped in one act of attention.

(4) *The cancellation test*:—To determine the ability to concentrate.

TABLE IV.

Results of tests in 145 school children before and after treatment.

	Of speed tapping	Mean scores in memory span			Span of apprehension	Cancellation scores
		Shapes	Digits	Pictures		
Before	20.4	3	3	4	4	20
	38	7	7	8	8	52
After treatment	31.8	6	6	7.5	7	48

The severer the nutritional deficiency, the farther from the normal is the mental test performance. Such a difference can be interpreted as demonstrating the effect of nutritional status on mental test performance. The mean scores for normal children are in brackets.

2. In most of the children, significant deviations from normal performance were observed and they varied considerably in degree from one type of antisocial behaviour to another. These deviations were characterised by (a) a general lowering of performance, and (b) a more marked lowering in some types of tests than in others.

3. This lowering may be attributed to difficulties in attention as these children would get fatigued much earlier and start complaining of unlocalised pains and aches all over the body. There is a general lowering of the power of the voluntary attention resulting in inability to make inferences and development of a sort of mental evasion.

4. The development of mental evasion easily takes place in them because (a) these children by reason of their low physical standard and psychological inadequacy feel that they are not quite the equal of the other competitors, and (2) for the same reason they try to escape from situations in which their deficiency appears prominently.

If this feeling is not early recognised and properly handled by parents and teachers; it is likely to become chronic and make serious trouble in later years.

The improvement in mental functioning as well as in general behaviour after the treatment is a noteworthy feature.

Proper food would prevent much psychological illness in school age children.

Iron Metabolism and Diet

Iron from food, is taken up from the digestive tract, and combined with a protein in the mucosa. The quantity absorbed thus from this source may vary, from 1 mg. in normal individuals to 50 mg. in cases of severe iron-deficiency anaemia. Only from 0.8 to 1 mg. is daily excreted from the system through urine and bile.

60 % of the body iron is present in Hb.; 20 per cent (primarily ferritin) is stored in the tissues and so readily available. A mixed diet which may contain 5 to 10 mg. of iron per day is usually deemed adequate in normal cases. If the reserves of iron are saturated a loss of blood to the extent of 30 to 35% of the total can be restored from these reserves. If not saturated, the Hb. restoration will necessarily have to depend on iron from the daily intake of food.—*Jour. Clin. Path.*, 7: 57, 1948.

T.N.S.R.

Nutrition in Surgery

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"THE nutritional state of a patient has long been recognised as an important factor in surgical risk and convalescence, but the importance of thorough understanding is only now beginning to be appreciated" Remington *et al.* The value of fluid and electrolyte balance, of blood and plasma transfusions, of high protein diet, of chemotherapy and intestinal decompression, have all aided in the better care of patients.

It is essential to diagnose a patient in the early stages when there is any abnormal physiology at work which depletes the patient of the essential body constituents or alters the normal mechanics of the body. There is frequently a tendency to the formation of a vicious cycle when it is often too late to save the patient. Every patient in a poor nutritional state is a bad surgical risk and the nutritional state is maintained by the following:

I. Fluid balance.—This is the most important factor in maintaining the nutritional status of the patient because a loss of about 20% of its water content results in death.

The average daily intake of fluid varies from 1,800 to 3,500 cc. and is made up of two sources *viz.*, imbibed water and from ingested food. The body as a result of its own tissue catabolism produces about 500 cc. of fluid per day. The kidneys are the most important in maintaining water balance. If there is plenty of available water, the urinary wastes are put out in a large volume of urine of moderate or low specific gravity and *vice versa*. One should never forget the fact that a small volume of urine, particularly of high specific gravity means insufficient available water. The daily normal output of urine should be about 1,000 to 1,500 cc. on an average day while the minimum gravity necessary to excrete the waste products of the body is 500 cc. provided the kidneys are functioning at maximum capacity.

Water forms about 70% of the body weight and is distributed as follows:—(a) In the blood plasma, 5–9%; (b) In the cells 45–50%; and (c) in the interstitial or extra-cellular tissue spaces, 15 to 16%. The intracellular part is very stable, while the extracellular part is labile and serves as a reservoir in cases of emergency. The normal fluid distribution is maintained by a balance between hydrostatic and the osmosis pressure in the capillaries.

A dehydrated surgical patient is a grave risk unless the fluid balance is restored. Evidence of dehydration appears when the loss of water is equivalent to 6% of the patient's body weight (Coller) and this very often occurs in patients suffering from vomiting, diarrhoea,

excessive perspiration, hæmorrhage, gastro-intestinal fistula, burns, etc. The intensity of the thirst and dryness of the skin, tongue and buccal mucosa serve as a good guide in the wards, if laboratory information is not available. It is a wrong practice to withhold water to a patient who is actually crying for it—if oral fluids are contraindicated in that particular case then it is a different matter, as in this type of case parenteral fluids must be given. There is often a tendency to underdose the amount of fluids than to overdose it. Signs of early dehydration are commonly seen even in patients during convalescence from an operation, it is easy to understand when we consider the following data—

(1) The usual fluid requirements of 3,500 cc. per day is inadequate when body fluid has been lost abnormally.

(2) Each degree rise in temperature above the normal leads to an additional loss of 500 cc. per day.

(3) The average loss of fluid during the first four hours of a major operation is 1,000 cc. and during the early postoperative period 2,000 cc. daily.

Hence to maintain fluid balance 4,500 cc. are required, while in a dehydrated patient the amount is 8,000 cc. per day. This will clearly explain the shrivelled look on the faces of the cases with a deficient fluid balance and very often because of unnecessary restricted intake of fluids.

Treatment of dehydration :—It is important to realise that in dehydration the salt balance is also altered and hence this must be replaced simultaneously. The caloric value is obtained by the inclusion of glucose, hence the ideal fluid is 5% glucose in normal saline. The optimum rate of injection is 300 to 500 cc. per hour for full utilization by the body. It is important that for the metabolism of the injected dextrose, reserves of vitamin B₁ are heavily called upon, besides the B complex and vitamin C. They may be added to the same solution. The plasma chloride content should be estimated daily, if possible, 0.5 gm. of sodium chloride per kg. of body weight for each 100 mg. fall in the plasma chloride level serves as a guide for replacement (Bartlett). As long as the patient excretes more than 1 gm. of salt per day in the urine there is little danger of serious salt depletion (Bartlett). Excretion of sodium chloride in the urine is easily tested with silver nitrate solution. The intravenous route is the best of the parenteral methods of administering fluids. The rate of flow depends upon the therapeutic indication. In cases of shock and hæmorrhage four to five hundred cc. may be given within a few minutes to restore the blood pressure to a respectable level and then the fluid injected at a slower rate.

In other circumstances a slow rate should be used especially when there is evidence of congestive heart failure, arteriosclerosis, pulmonary oedema and kidney damage.

In infants suffering from congenital pyloric stenosis the dehydration is rapid and, unless adequately treated, fatal. Fluid replacement has to be thorough and should utilize the subcutaneous method, injection into the superior longitudinal sinus has often been utilized and this combined method is more efficacious than hypodermoclysis alone. Blood transfusion also helps in a very weak infant but not more should be given than 10 cc. per lb. of body weight. Post-operatively the fluids should be continued as the method of oral regime differs with different clinics and the quantity given is inadequate in the early stages. Proctoclysis is too uncertain to be of value.

Rapid dehydration is common in adults suffering from high and mid-intestinal obstruction. Replacement of the fluid and the ionic contents should be started no sooner the patient is under observation. Operations can easily be withheld for 30–60 minutes during which time the patient is resuscitated to stand the operation better. A Bevine's or Miller-Abbott tube introduced to decompress the bowel is often a life-saving measure. It may be retained post-operatively till the bowel function has returned to normal. The fluid output chart should be strictly maintained as a proper idea of the amount of fluid to be given can only be obtained by this method. The presence of this tube is no bar to oral feeding—part of it may be removed but oral feeding has a great psychological effect on the early recovery of the patient and helps to start the psychological functions of the bowel.

In cases of extensive superficial burns serum exudates into the blisters and dehydration of rapid onset occurs. During the early stages of primary shock, besides the other measures, plasma transfusion very often saves the patient. Blood is not indicated as there is already hæmoconcentration due to serum loss. The amount of plasma necessary depends upon the extent and severity of the burn in relation to the size and age of the patient, by the intake and output of fluids, by blood-pressure readings and by laboratory tests, such as hæmoglobin determination, erythrocyte count, blood chloride estimation and urine examination. Harkin's method of calculating the amount of plasma is useful and consists of giving 100 c.c. of plasma for every point the hæmatocrit is above the normal of 45, on the assumption that the patient's hæmatocrit reading before the injury was normal. If the plasma protein is below normal and additional 25% of the calculated amount of plasma should be added for every gramme the protein level is below 6.0 gm. per 100 c.c. Oral fluid should be encouraged and a diet of a high protein, high vitamin and caloric value. In cases where secondary anæmia occurs blood transfusions of 75–100 c.c. each will help in early recovery.

Intestinal fistulae :—In external fistulae situated in the small intestine the amount of secretions drained out is very large and

rapidly dehydrates the patient in spite of parenteral replacement therapy. Fortunately most of these fistulae have a tendency to close up within 4 to 6 months. Administration of adequate 5% protein hydrolysate helps to maintain the nitrogen balance and avoids wasting of the body muscle mass which is the labile source of endogenous proteins.

In fistula from the duodenum and jejunum the acid and the base are lost equally, while in the case of a fistula in the ileum the base loss predominates as in cases of diarrhoea and external biliary drainage.

Sudden hæmorrhage, either, external or internal, has very often to be dealt with. External variety is very often traumatic and ing should be first stopped, and, if possible, rapid blood transfusion given of about 200-400 cc. according to the amount lost and then a slow drip maintained till the patient's general condition returns to normal. Blood loss is best replaced by whole blood, substitutes like plasma, glucose-saline are inferior in that they temporarily restore the fluid in circulation and raise the blood pressure. Glucose-saline passes out into the tissue spaces and is excreted rapidly by the kidneys—hence it acts as a stop-gap measure till blood is available.

Massive internal hæmorrhage may occur from peptic ulcers, ruptured tubal pregnancies. The latter demands immediate surgery to stop the bleeding. A slow drip transfusion should be given even in the presence of a leaking blood vessel as it acts as an aid to hæmostasis. By the drip method the blood pressure is not raised to such an extent as to start fresh hæmorrhage. Uncontaminated blood collected in either the peritoneal or pleural cavity has been utilized in autotransfusion after adding citrate and has proved a life saver on many occasions.

II. Acid base imbalance.—The human body by a delicate mechanism is able to maintain a fixed reaction of the blood. Sodium is the important salt in the extracellular tissues while potassium plays a similar role in the intracellular protoplasm while the bicarbonate of the body serves as the important buffer system with the help of the lungs and the kidneys to keep the body from either acidosis or alkalosis; to a certain extent this mechanism may succeed but may be so severe as to require medical aid.

A. *Acidosis*: This condition is often noticed in diarrhoea, low intestinal fistula, defective carbohydrate metabolism as in diabetes, in starvation and in extreme anæmia conditions. The urine is scanty, highly acidic and contains ketone bodies, while the CO_2 combining power of blood is lowered (normal 55-80 volumes per cent). Values under 20 should cause grave concern.

The treatment of acidosis is to remove the exciting cause. 5% glucose in normal saline helps to restore the electrolyte balance, the glucose acting as a diuretic and combating the ketosis. Insulin when indicated should be given. In severe cases alkalies are indicated but must be given with caution lest it overcorrect the acidosis and produce alkalosis. This is true in children where the ionic balance is very delicate. The CO_2 combining capacity of the blood should be done and the dosage of alkalies calculated to Palmer and Van Slyke's formula—0.5 gm. of Sodium Bicarbonate will elevate the CO_2 combining power of blood plasma by one volume per cent for each 42 pounds of body weight. Roughly 5-10 c.c. per kilogram of body weight of a 5% solution of Bicarbonate can be given per day. Hartman advocates the use of Sodium Lactate in 1.8 per cent strength and 10-20 c.c. per kilogram of body weight per day.

B. *Alkalosis* is commonly seen in cases of pyloric stenosis with excessive vomiting, in voluntary and involuntary hyperpnoea due to the throwing out of a large amount of carbondioxide. When in pyloric stenosis the dehydration is marked dextrose in normal saline should be given while in the absence of dehydration 2 per cent saline acts well. Acid forming salts like Ammonium Chloride in gram doses six hourly will combat the alkalosis, if tetany of alkalosis is present 5 c.c. of a 10 per cent calcium chloride intravenously is indicated.

Gamble has emphasised the fact that whether an inorganic acidosis or alkalosis is present, both can be corrected by a combination of the same two solutions: (1) Isotonic Saline solution which provides water and the chief electrolytes needed, and (2) Dextrose solution.

The kidneys with a good supply of water can be depended upon to use this water for the excretion of the less needed electrolyte.

III. Carbohydrates, proteins and fats.—These are the main constituents of a normal diet and yield the necessary calories which should range from 2,500 to 3,000 calories per day. Fat yields twice the amount of calories as carbohydrate or protein, while protein has its specific dynamic action. The diet should contain 100 gms. of proteins, 50 gms. of fat and 400 to 500 gms. of carbohydrate.

Inoperative starvation is inadvisable as it produces acidosis and dehydration—it is essential to omit any food for about 3 hours before operation. Postoperatively the appetite remains poor for a day or two and naturally the patient requires well-cooked, diversified, yet not spicy, food, sent in an attractive manner to stimulate the dormant appetite. The individual taste and idiosyncrasies should be considered. Some patients do not tolerate the amount of carbohydrate orally then it may be replaced parentally, more

especially if shock is anticipated during operation, where 500-1000 cc. of 10 per cent glucose should be given. Insulin 1 unit per 3 gms. of glucose helps, half the Insulin is given 15 minutes before the injection and the remaining on completion of the drip (Fisher).

The proteins are essential to maintain the nitrogen balance of the body. The essential amino acids maintain the vital functions of important organs like the liver, heart and brain. Protein is an essential element of the cellular structure. A low plasma protein level is detrimental to proper healing of wounds (Thompson *et al*). Ravdin states that a liver high in fat and low in protein content is maximally susceptible to injury especially with narcotic drugs so often used in surgery. The plasma protein level should be estimated preoperatively and restored if low, a level below 4 gms. per cent is a hazard to operation. A gastroenterostomy stoma does not function well after operation if the plasma proteins are low (Ravdin).

Treatment is to feed the patient with a high protein diet and give casein hydrolysate prepared as a drink with fruit juice. In urgent cases it can be given intravenously in the form of 5 per cent protein hydrolysate. Hypoproteinaemia must be restored to normal in patients requiring biliary surgery where liver damage is a common accompaniment.

Fats: The normal intake per day should be about 50 gms. Fat is useful for its high caloric value. Excessive fatty intake is detrimental as also is a fat person a poor surgical risk. Fat necrosis is common in the operated area, the operative approach is made more difficult with a thick fatty layer under the skin and post-operative period is not without anxiety as chest complications are common. If an interval operation is to be performed it is advisable to keep the patient on a restricted diet and exercise to reduce weight.

IV. Vitamins.—The Romance of the vitamins might well be the title of the story of the accessory food factors which Hopkins in 1909 described as being indispensable for the maintenance of growth and life, which Funk in 1911, called "vital amines" and which now threaten by their increasing numbers to exhaust the alphabet in their designation (Emile Holman).

Deficiency of vitamins generally occur in all the vitamins rather than a particular one. Minot observes that "Borderline states of nutritional instability are much more common than is usually appreciated. There is a wide zone between optimal nutrition and the level at which classic symptoms of recognised dietary deficient states develop."

Vitamin A deficiency is most felt on epithelial tissues which undergo degeneration and hence spontaneous infection is common

in the affected areas. At the same time parotitis, purulent bronchitis, pneumonia and urinary infections are post-operative bugbears and are more prone to occur in young children.

The vitamin B group is helpful in cases of neuritis, atony of the intestinal tract, anorexia, loss of weight; it helps during the early convalescent period. It is said that the symptoms of vitamin B deficiency remain latent at times and appear only with the onset of some intercurrent infection after a surgical operation and during increased body metabolism. Cardiac reserve is lowered in vitamin B deficiency and circulatory failure is a postoperative danger.

Adequate intake of fresh orange and lemon juice will supply the daily vitamin C requirement. Holman has shown that 44% of the free clinic patients were on the borderline of scurvy. Yavorsky *et al* found the vitamin C content of all tissues like adrenal, brain, pancreas etc., to be at a lower level in patients over 46 years of age than those below 46, hence the older group requires a longer period of preparation by high vitamin intake than the younger patients. Of real surgical importance are Hojer's original observations that ascorbic acid is intimately concerned with the synthesis and maintenance of the intercellular supporting materials, which provide the frame work of healing. The drain on the vitamin C content is increased in acute infections like acute osteomyelitis, pneumonia, pulmonary abscess as well as in pulmonary tuberculosis.

In the preoperative preparation of a jaundiced patient vitamin K is imperative—when given orally it should be accompanied with bile salts or else the absorption of vitamin K is negligible (Butt.) The quantity to be given depends upon the prothrombin level in the blood. Unfortunately when the liver itself is injured by poisons, infection or new growth, vitamin K is useless as the site, where prothrombin is formed, is damaged.

Conclusion.—Nutrition in a surgical case is an important aspect on which the surgeon has always to keep a watch. Expectancy is the watchword of good surgery—any complication that is likely to arise must be prevented by adequate measures from the start. The fluid balance has a tendency to alter rapidly and may be missed in the early stages—an adequate urinary output should be maintained at all times. An intravenous drip as a routine in all major operations is a sound surgical method, it avoids incipient dehydration.

The patient after operation must be made to return to normal diet as soon as possible. Collier and Maddock emphasise that the best way to maintain proper nutritional balance is to give food and drink by mouth—Parenteral therapy tides over an emergency; the sooner the patient is restored to food and drink by mouth, the greater his chances of recovery.

Holman states that there is little justification for the almost universal practice of admitting the candidate for an elective operation on one day and operating on him the next. The ordeal of operation with its attendant risks should, whenever possible, be undertaken only in patients in a state of optimum nutrition. To end, let us remember the words of Lord Moynehan, "Surgery has been made safe for the patient, we must now make the patient safe for surgery."

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Queen's Road,
Bombay.

Tooth Decay in Relation to Diet and General Health

The teeth of 1046 children aged 6 months to 18 years, of different nationalities and resident in Hawaii, were examined and compared with the teeth of 104 immigrant Filipino children. From these observations and studies of Japanese plantation children on low and high intakes of protective foods, it was concluded that general health and ordinary resistance to disease are not related to tooth health. The comparative freedom from tooth decay of the native Hawaiians was attributed to their consumption of large quantities of taro and seafoods. The former, unlike rice, when digested gives an alkaline residue and is high in oxalates, and the latter contribute fluorine, all of which were considered to favour the prevention of caries. Another unknown protective factor was postulated.—LARSEN, N. P., *J. Amer. Med. Assoc.*, 1948, 137, 832—838. [Honolulu, Hawaii.]—*Nutritional Abstracts of Reviews*, Vol. 18, 103.—E. D. Baird.

Nutrition in Surgical Cases

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THE subject of nutrition is a very vast one but we will restrict here only to its adaptability in the vegetarians who constitute a great bulk of our surgical cases. The special need of maintaining nutrition in surgical patients is because:—(1) the resistance of the patient is increased; (2) quick healing of the wound occurs and consequently a shortened period of hospitalization; (3) of prevention of certain complications; (4) reduction of post-operative shock and asthenia; and also (5) prevention of liver damage by the toxic effects of the anæsthetic agents.

One of the important advances in surgery is the recognition of the direct relationship of the patient's state of nutrition to his chances of withstanding well the shock of the operative procedure and the anæsthetic agents, of resisting post-operative infection, of achieving primary closure of the operation wound and of avoiding post-operative complications. The damage done to the tissues during operative work superimposed by starvation diet during post-operative period produces a great negative nitrogen balance and hence a great need arises of taking proteins in the diet.

Hypoproteinemia and hypovitaminosis are the two conditions that adversely affect the convalescence of the surgical patients. The causes of this hypoproteinemia are:—

(1) Insufficient protein intake especially in vegetarian diet which is supposed to be lacking in correct balance as far as the body needs are concerned. It contains more of carbohydrates and fats and less of essential proteins and hence a great necessity of supplying amino-acids to the vegetarian patients.

This being stated so in books, our observation is that these vegetarian patients who are considered poor operative risks, have stood well the major operative procedures like gastro-jejunostomy and ileo-colostomy with no postoperative troubles. This is only an observation for which no scientific explanation can be put forward.

(2) Impaired digestion or absorption as in chronic diarrhoea, peptic ulcer or carcinoma of the stomach.

(3) Inadequate synthesis of the plasma proteins in cases of liver diseases, such as chronic alcoholism etc.

(4) Increased breakdown of body protein stores as in fevers and hyperthyroidism.

(5) Excessive loss of proteins in bleeding, burns, intestinal fistulae, empyema, huge abscesses, continuous gastric suction and nephrosis.

Hypoproteinemia may present clinically as:—(a) peripheral oedema; (b) visceral oedema with diminished gastro-intestinal motility if the bowel-wall is affected; (c) diminution or complete anuria; (d) anaemia; (e) liver damage—if methionine component is less; (f) delayed wound healing; (g) infection; (h) thrombosis and embolism; and (i) tendency to form decubitus ulcers because they are said to be caused by local pressure on tissues of lowered vitality due to protein deficiency. The condition of hypoproteinemia can be corrected by giving protein in the diet and if the digestive enzymes are deficient because of the illness or disease of the stomach, then pre-digested protein are preferred. The dietary proteins are acted upon by the proteolytic enzymes of the intestine and broken down finally to the amino-acids in which form they are absorbed by the intestinal mucosa and carried to the liver by portal circulation for synthesis and utilization. They are then distributed to the tissues by the systemic circulation:—(a) Synthesis of tissue cells; (b) Utilization for some special purpose such as synthesis of haemoglobin, fibrinogen, plasma proteins, insulin and prolactin, hormones, histamine, enzymes and antigens; and (c) Deamination etc.

Surgically, we are most concerned with the plasma proteins because they are useful for tissue repair and growth, maintain osmotic equilibrium because of their colloid nature and also maintain acid-base balance. For the proper healing of the wound, we must guard against hypoproteinemia because if the protein content of the blood is reduced, there will be loss of fluids from the capillary bed into the intracellular spaces resulting in oedema of the tissues. The edges of the wound thus become oedematous and it retards healing. In case of gastro-jejunostomy operation, the oedema of the stomach may bring about narrowing or complete closure of the opening and subsequent distension of the stomach and vomiting. The intestinal anastomosis are much less apt to function properly during the first few days following operation, if oedema associated with hypoproteinemia is present.

Carbohydrates.—Carbohydrates spare the body proteins. This is given in the form of dextrose or rice conjees. The dextrose increases the glycogen reserve, relieves thirst, promotes diuresis, protects against ketosis. Its administration alone is not likely to result in oedema of the tissues but, on the contrary, it is said to diminish the oedema that has already occurred. It is given as glucose orally or by glucose—Saline infusions or by skimmed milk powder—the 100 gms. of which when dissolved in 200 cc. of water will yield 34 gms. of proteins and 52 gms. of carbohydrates; thus the volume

is also less if we do not want to load the system by fluids. Intravenous administration of large quantities of neutral salts with which the glucose is generally incorporated, results in oedema of the tissues if hypoproteinemia also exists. Intravenous glucose alone helps to reduce the oedema if present.

Vitamins.—Surgically, we are concerned more with vitamins C and K. The evidence is less convincing with regard to vitamins A, B and D but they certainly merit consideration.

Vitamin A:—Its deficiency results in decreased ability to maintain specialised epithelial structures. The deficiency is generally associated with urinary calculi and pulmonary complication—the latter because of the damage done to the cilia of bronchi and trachea. To correct it, we should give Haliverol capsule, generally 2 capsules b.d.

Vitamin B:—It is believed to be good for increasing appetite. The components of B group are concerned with carbohydrate metabolism (thiamine), cell-respiration (riboflavin) and cellular oxidation (nicotinic acid). These are frequently added to intravenous fluids or protein diets on merely empirical grounds. The choline component is concerned with prevention of liver disease by mobilizing the fatty acids. Pyridoxine—B₆ is concerned with the metabolism of amino-acids and the treatment of certain nervous diseases.

Vitamin C:—Out of all the vitamins, this is supposed to be the most important because of the quick healing of the wound and prevention of bleeding in the muscles and petechial haemorrhages and blood diarrhoea. Its deficiency may result in periosteal haemorrhages in the lower extremities bones of artificially fed infants.

In surgery, its absence prevents the formation of collagen fibres in the wounds and proliferating mesodermal cells fail to mature so that a scar of poor healing results. It is, so to say, concerned with the synthesis and maintenance of the intracellular supporting materials and hence we give vitamin C to patient with disruption of wounds and we have noticed satisfactory results following its administration. It also increases the resistance to infection. Our daily requirement ranges from 30 to 75 mgm. and sufficient amount can be had from orange juice, milk, fresh fruits and vegetables. Otherwise parenteral administration of 500 to 1000 mgm. daily with glucose saline or plasma infusions will be an ideal alternative.

Vitamin D is concerned with the absorption of calcium and phosphorus from the intestinal tract into the blood stream. Hence adequate amount of vitamin D is of aid in the healing of the fractures, the clotting of the blood and in maintaining normal capillary permeability. Haliverol capsule is therefore beneficial in supplying the vitamin D also,

definitely more than those of vegetarian origins. Meat proteins have generally unpalatable taste. As these are pre-digested proteins without the addition of any preservative, they deteriorate quickly if put up in bottles and hence they are generally available in ampoule form for oral use. If the bottle is kept well air-tight and the product consumed in a tolerably good time, then even powder and liquid form will suffice. Such hydrolysates are Ledinac of Lederle Laboratories—which is a liver protein augmented with B-complex factors and C; protein hydrolysate of Arcies; Prohepax of T.C.F.; Protocasein of Zandu which is in a liquid form made palatable by addition of glucose; Hydramin powder of Hion. The unpalatable taste can be masked by giving it in fruit juices or whey etc. There are many conditions where this protein diet is indicated but at present the cost is so prohibitive that the use is extremely limited to the affording class of patients only. Because of the infirmary attached to our hospital, we have here an ample opportunity of observing the post-operative condition of wounds in the infirms and in the patients coming from outside. Infirms are generally on poorly balanced diet and are poor operative risks while patients from outside come with a tolerably good nutrition. In a case of an infirm, a suprapubic wound took a very long time to heal and that too healed after a few injections of vitamin C and glucose whereas a suprapubic opening closed very early in another patient. A simple operation of circumcision in an infirm cost him his life because of the wound getting absolutely unhealthy and sloughing due to his undernourished condition. A disruption occurred in a hernia wound in a case of an absolutely undernourished Brahmin patient—a strict vegetarian and his wound took nearly 2 months to heal by granulations after some vitamin C injections. A very bad case of gall-stones in an old lady, stood well the operation of cholecystectomy because of the intravenous plasma and the post-operative period too went off uneventfully. Plasma has also saved many lives. Where oral feeding is impossible or insufficient, the deficiency can be made good by whole blood transfusions, plasma or hydrolysates of casein—intravenously. Thus intravenous Amigen is extremely used but at times it gives a febrile reaction and thrombosis in the vein,—but it can also be given intramuscularly though this route is a bit painful one. Approximately 1 gm. of protein per 2 lbs. of body weight daily is the optimal requirement for the ordinary patient; thus 2 liters a day of protein digest containing 6.5 gm. of Nitrogen per one liter and equivalent to 40 gms. of proteins will keep most patients in positive nitrogen balance. One liter of plasma contains only 18 gms. of proteins and hence a large quantity is to be given to make up the correct requirement and thus a large quantity of fluid introduced in the circulation may cause pulmonary oedema by loading the system. Oral route is preferable for protein hydrolysates as far as possible.

Nutrition in appendix case.—As routine, we give milk, orange-juice, Ovaltine, Glucose water etc. supplemented by oral hydrolysates, if need be. The progress is generally remarkable.

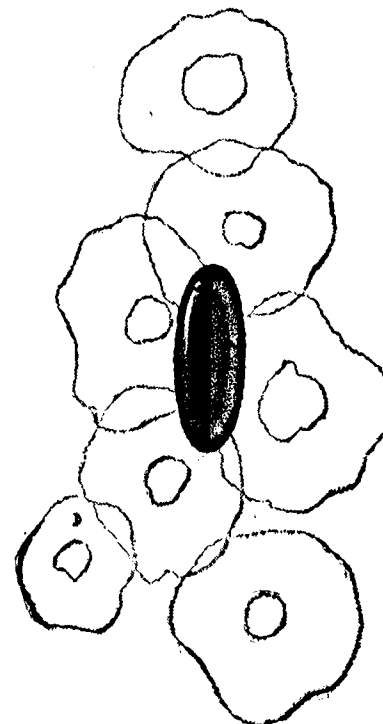
Gastro-jejunostomy.—Pre-operatively such cases are built up in nutrition by advising them to take plenty of milk, orange juice, and protein hydrolysates, and post-operatively for the first 24 hours, he takes only boiled water and then next day boiled glucose water and from the third day onwards milk, orange juice, protein hydrolysates; with this routine all of them have done well so far. In this case, another alternative suggested is the passage of a Miller-Abbott tube which contains two compartments of unequal lengths. The tube is passed into the stomach before the operation and the end of the tube is guided into the jejunum at the completion of the gastro-jejunostomy. The short compartment which reaches only to the stomach is used for aspiration purposes keeping the stomach empty while the food is introduced into the jejunum through the longer compartment. The nutrient introduced is the protein-glucose salt mixture. Similarly the diet can be adjusted in cases of gastrosomy, ileo-colostomy and iliac-colostomy according to the need of the particular patient.

We are very much indebted to the following books for supplying us with lot of information regarding nutrition so far as theoretical consideration is concerned: (1) Surgical Treatment by Baneroff and Wade; (2) Recent Advances in Surgery by Edwards; (3) Medical Bulletins of Lederle Nos. 3 and 4.

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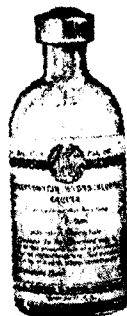


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Vitamins and their Relation to Cardiovascular Disorders

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THE problem of post-war economic adjustment with rising cost of living has increased the food problems of large group of wage earners with fixed incomes all over the world. The influx of large number of refugees who have been deprived of their homes and earnings have been an additional problem for our country, seriously affecting their intake of foodstuffs and the composition of their diet which has been imbalanced and inadequate. It would seem reasonable to assume that under such conditions all grades of vitamin deficiencies will be met with in the population, from characteristic clinical features of beriberi, scurvy and pellagra to those totally unrecognizable by present diagnostic methods.

Secondary vitamin deficiencies arising in the course of various diseases and conditional deficiencies which may arise in spite of a normal diet when abnormal states impose certain special nutritional needs have only recently been appreciated. Deficiency disease may therefore arise not only from insufficient intake of food, but also from poor absorption from gastrointestinal tract from faulty utilization of substances within the body or indeed from increased need of food because of heightened metabolism.

Recent clinical work has conclusively demonstrated that cardiovascular disturbances are of frequent occurrence in vitamin deficiency states. Elevation of portal venous pressure and impaired capillary circulation of the intestines account further for a poor absorption from the latter in a cardiac case. According to Pollack and coworkers (1940) and Orieg and Govier (1943) liver congestion further inhibits utilization of vitamins, explaining the common occurrence of a-vitaminosis, especially vitamin B₁ deficiency.

Vitamins are not considered as mere food accessories today but some of them at least should be regarded as possible chemotherapeutic agents for indications quite as strict and clearly understood as those for Sulphonamides and Penicillin. With this conception in mind Vogelsang and Shute (1948) have recorded their observations of the effect of vitamin E on many pathological cardiovascular disturbances.

Vitamin B₁ is the only vitamin known to be important to the functional integrity of the heart. Jolliffe (1939) experimentally produced vitamin B₁ deficiency in normal subjects and found that the symptoms of cardiovascular disturbances may appear early. Palpitation and tachycardia are usually the earliest symptoms. Dyspnoea and precordial oppression and pain appear early. These

are also the early symptoms of beriberi heart. If the disease is allowed to progress, more characteristic changes appear such as cardiac dilatation, gallop rhythm, systolic murmur over the pulmonary area, venous distension due to increased venous pressure, increased pulse pressure, congestion of hili and of pulmonary bases and diffuse peripheral oedema. Weiss and Wilkins (1937) drew attention to warm flushed skin and rapid blood flow, due, it is believed, to generalised arteriolar dilatation. Wilbur (1938) demonstrated that whereas in congestive heart failure from any cause other than hyperthyroidism, the circulation is slowed in that due to vitamin B₁ deficiency both the circulatory time and minute volume are increased.

The electrocardiogram shows atypical changes. Inverted T wave, slight aberration and low voltage of the ventricular complexes and prolongation of Q-T or premature beats and auricular tachycardia have been described but the persistence of sinus rhythm is usual and is important in diagnosis. On fluoroscopy we may observe an enlarged cardiac shadow and decreased amplitude of contraction, the latter may be caused at least in part by some degree of precordial effusion. Pulmonary artery and its branches show dilatation.

At autopsy the heart is flabby and dilated especially the right chamber. Microscopical examination reveals diffuse but not severe lesions of the myocardium, intracellular and intercellular oedema and occasional scattered areas of necrosis and even petechial hæmorrhages.

The exact mechanism of the syndrome is still debated. On the one hand, the constituents of vitamin B complex have a direct influence on the metabolism of carbohydrates. On the other, the oedema is partly the result of low blood protein level, which is favoured by mal-nutrition. Therefore peripheral oedema may be caused both by this mechanism and by heart failure. Whether the oedema of cardiac muscle has the same colloid osmotic mechanism or is a primary factor is not known; in any case, it may be a contributory factor to heart failure. The possibility of neurogenic disturbance of the cardiac functions because of sympathetic and vagal lesions has been discussed Blankenhorn and coworkers (1946). The increased speed of circulation, first observed in Oriental beriberi by Wenckebach (1934) frequently present in Occidental thiamine deficiency also, may be one of the causes of failure by decreasing peripheral utilization of oxygen. It resembles the phenomenon observed in thyrotoxicosis.

The diagnosis becomes difficult especially when patients with valvular lesions and other organic disease of the heart have thiamine deficiency. This disorder should be suspected in patients with heart failure, who are alcoholics, or who show any other manifestations of nutritional deficiency particularly of the vitamin B

complex. Blankenhorn (1945) based his diagnosis on insufficient evidence of their etiology, signs of neuritis or pellagra accompanied enlarged heart with sinus rhythm, dependent oedema, high venous pressure, recovery with decreased size of heart, autopsy consistent with beriberi heart disease. In case of congestive heart failure with beriberi heart disease. In case of congestive heart failure attention is sometimes drawn when response to digitalis is incomplete and the presence of vitamin deficiency is suspected as it may be corrected by full doses of vitamin B₁. Sometimes the duration and severity of the deficiency may lead to irreversibility of the process and Blankenhorn (1946) reports that the Thiamine treatment may not cause a prompt improvement.

Relief in these cases is obtained neither by digitalis nor by diuretics but by administration of antineuritic vitamin B₁ 50 to 100 mg. daily intravenously in severe case, otherwise intramuscularly and supplemented with orally, preferably vitamin B complex. High protein diet is indicated. Food rich in vitamin B₁ should be included. These prolong the action of vitamin B₁ given by injection, even if they might not be effective without the latter (Freedberg and Blumgart 1943).

Pellagra, although an important deficiency disease, has not been found to be associated with any specific cardiac pathological changes; circulatory changes found in such cases are to be ascribed to lack of vitamin B₁ or hypoproteinemia of starvation or a coincident heart disease. Feil (1936) and Rachmileitz and Braun (1945) did not find any enlargement of the heart.

The electrocardiographic changes of the T-wave which is low, absent or inverted and ST segments and prolonged Q-T interval are probably of metabolic origin as they promptly disappeared after administration of Niacin in daily oral doses of 300–500 mg. Niacin is essential to the metabolism of carbohydrate.

Vitamin C has diuretic properties even in health and when given to patients with congestive heart failure as ascorbic acid tablets 50 mg. each three times a day is capable of inducing and maintaining a profuse diuresis even when other therapeutic measures have failed. It may actually induce a diuresis in excess of that produced by digitalis though not with the same degree of clinical improvement nor with reduction of ventricular rate.

Dilatation of right heart has been described in severe cases of scurvy. It is still to be demonstrated whether it is actually caused by vitamin C deficiency or by associated cause including deficiency of the Thiamine. White (1946) draws attention to the tendency to hæmorrhage in cases of scurvy which may involve the heart and pericardium as well as other parts of the body; the heart muscle is said to show degeneration in scurvy.

Recently some workers have claimed that rheumatic fever and its complications are due to a deficiency in vitamin C. They derived

their conclusions from the fact that they found low blood values for cevitamic acid in these cases, especially during febrile stages. Whether the low blood content of cevitamic acid is the result or one of the causes of the disease is still undetermined. Also the therapeutic test to determine if adequate vitamin C supplied to the blood stream will prevent or clear up rheumatic fever must be investigated. One thing seems certain that in the acute stage of rheumatic disease vitamins C and K are beneficial and may prevent haemorrhagic manifestations.

Rickets due to lack of vitamin D, when very severe, is also associated with dilatation of heart. In a series of 11 children dying suddenly with rickets, Meixner (1928) showed left ventricular dilatation in each child post mortem. It is possible in many cases the thoracic deformity and not the vitamin D deficiency is the cause of observed clinical changes. Moreover associated vitamin deficiencies may be involved.

Mason (1942) observed vascular abnormalities in vitamin E deficient animals. Taking this clue Vogelsang and Shute (1947) in coronary thrombosis and other cardiac processes. This stimulated them to investigate the effect of tocopherol upon the pathological conditions of certain types of peripheral vascular diseases.

Shute and coworkers (1948) have reported an encouraging influence of vitamin E on vascular disease. With a daily oral dose of 200-300 mg. of alpha tocopherol they recorded marked improvement in 22 cases of thrombophlebitis and phlebothrombosis. Early thrombus melted away, while older thrombi required doubling the dose. The authors studied the effects of tocopherol in 3 cases of gangrene of extremities, 2 cases thromboangitis obliterans and 12 cases of cerebral thrombosis with good results. In the last condition the patients treated most promptly did best but even in old cases some improvement was achieved.

As far as known vitamin E seems to work on these cases in four main ways: (1) It decreases oxygen requirement of heart muscle by 50% or more so that it cuts down the amount of work that the heart muscle has to do. (2) It decreases excessive capillary permeability or leakage of blood from injured vessels into tissues. (3) It attacks any thrombus or blood clot that is present in adequate dose; it may even prevent thrombus. (4) It sometimes slowly softens scar tissue.

The results of Shute and coworkers have not been accepted by many other workers, who failed to confirm the effectiveness of vitamin E in many of the cardiovascular conditions described by the authors.

Vitamin K is finding a new, still highly controversial, place in relation to acute coronary occlusion and its treatment. Doles (1947)

gave Vitamin K 50 to 72 mg. every 6 to 8 hours until the prothrombin level is normal in acute coronary occlusion. This was based on his earlier findings (Doles 1941, 1942) of hypoprothrombinemia in patients during and after an attack of acute coronary occlusion and Wartman's (1938), subintimal haemorrhage theory as the basis of occlusion established after study of serial sections of 41 occluded coronary arteries. Within 30 minutes to 3 hours after the first dose there is marked relief from acute pain, sometimes it controlled the pain, when Morphine failed to do so, sometimes a second dose of Morphine was not required after the administration of vitamin K. The blood pressure usually reached its original level next day in his series. The mortality rate among the 55 adequately treated patients was 3.6%.

The use of vitamin K in acute coronary occlusion is diametrically opposed to the uses of anticoagulants like dicumarol which has reduced the mortality rate and reduced the vascular complications of acute coronary occlusion as evident from the large number of published reports. The explanation probably lies in the two different types of cases with different pathology.

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Prakash Bhawan,
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Agra.

Nutrition in Enteric Group of Fevers

P. K. KRISHNANKUTTY NAIR, M.D. (Madras),
Colombo.

Introduction.—Anybody who has been in charge of an enteric ward would not have failed to observe the difference in physical appearance of patients entering the ward and leaving it. However muscular and strong the patient had been, by the time he gets over his attack of typhoid fever, he is reduced to his bare anatomical features. Why is the patient in such astonishingly poor nutrition, in spite of the fact he had been under the care of an able physician and had been looked after well by the able nursing staff? The answer is that enteric fever is a long febrile illness and the patient had been almost starved out, thereby satisfying an old adage, "starve a fever." It is high time that we break the custom and look to the feeding of the patient adequately so that when the patient is discharged he looks fit and well fed.

It is surprising to note that rules regarding metabolism, energy requirements and output have not been studied in these fevers, even though we fully understand the factors controlling the basal metabolism. (Barring diabetes mellitus and thyrotoxicosis the studies of metabolism have not been fully utilised in the appreciation of any other disease).

Pathological physiology of enteric fevers in relation to nutrition.—1. Basal metabolism is raised in all fevers. "For every rise of 1°F in the internal temperature of the body the basal metabolism is raised by 7%." The chemical reactions of the body like those occurring in a test tube are speeded up by the rise of temperature." So when a patient is running 103°F his metabolism is raised by 35% i.e., even if he is lying in bed he is as good as doing some manual labour. So we can understand that his body requirements are 35% more than normal. If this requirement is not satisfied by suitable food, necessary energy will be obtained by breakdown of the body tissues. Hence the need for careful and proper nutrition.

2. The person who is suffering from enteric fever, unlike the healthy individual, has to fight against the virulent enteric group of bacteria and their toxins. The immune bodies are formed only from globulin. Further during this prolonged disease process, there is extreme wear and tear of tissues due to the toxic process, and the most important tissue builder is protein, and so it is only right that we supply an adequate amount of protein to replenish the wasted tissues. Also as mother substance for the production of anti-bodies, protein is essential.

3. The usual complication of peripheral neuritis has been proved beyond doubt, as entirely due to vitamin B₁ deficiency and

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it can be completely avoided by giving adequate doses of vitamin B₁ which is relatively deficient in these fevers.

4. The flaring up of old tuberculous lesions is in no less extent due to lack of proper nutritional care during these prolonged fevers.

Having accepted the essential need for carefully feeding the patient, we have to see that the patient assimilates the required amount of water, carbohydrates, fats, proteins, minerals and vitamins.

Water.—It has not yet been fully realised that one of the early manifestations of toxæmia is dehydration. When there is dehydration toxins are not eliminated completely, because no living cell can work at its optimum in the presence of dehydration, since it involves the shift of salts from the cell to tissue spaces and so the toxæmia increases by interfering with the cell metabolism. So the primary aim is to avoid dehydration, by giving adequate amount of water. It is best given as half normal saline since it gives the required amount of salt as well. If the patient cannot swallow, an intra gastric drip method will ensure the proper supply of water and other substances. One must insist that the patient takes at least 6 pints of fluid per day.

Carbohydrate is essential since it forms the immediate source of energy. It can be given as imperial drinks. The practice of giving 50% glucose I.V. is to be deprecated, since this hypertonic solution will act as irritant to the cells of the kidneys, which are already under the influence of the toxins. About 50 grms. of glucose should be given per day; it may be given as 5% glucose saline by intra gastric drip failing which intravenous route may be used.

Fats:—Since they do not form an immediate source of energy they can be withheld for the time being.

Proteins are essential for reasons stated above. Whey and albumin water are good enough as sources of protein, but the best form is to give predigested or hydrolysed proteins. The advantage of predigested proteins is that they are already digested and so ready for absorption. It does not call upon the intestines to expend energy for digestive process. Further, they are in the elementary stage of Amino Acids and so absorption is better and utilisation immediate. About 400 c.c. can be given in a day and if the patient cannot absorb, it must be given by the intravenous route. (Incidentally it may be mentioned here that these protein hydrolysates saved the lives of many people during the Bengal famine.)

Salts:—Apart from the Sodium Chloride an alkaline mixture will be helpful since it will prevent acidosis and flush the kidneys.

Vitamins:—It is imperative that vitamins in full doses should be supplied. Vitamin C which is essential for the development of immune bodies should be supplied in 100-200 mgms. doses per day. Vitamin B complex which contains a minimum of 25 mgms. of vitamin B₁ should also be given daily. This is essential from two points of view: (1) The utilisation of vitamin B₁ is directly proportional to basal metabolism and so it must be supplied increasingly when the B.M.R. is raised, as in fevers; and (2) normal healthy bowel synthesises a certain amount of vitamin B complex in the presence of the normal bacterial flora. When the bacterial flora is changed as in enteric fever, the bio-synthesis is at abeyance.

Conclusion.—If the above dietetic fundamentals are insisted on I think that the lot of the patients suffering from continued type of fevers like typhoid will be much improved and they will get out, in a better state of nutrition.

No. 8, Mount Pleasant,
Baselines Road, Colombo, 9.

Significance of Milk Sugar in Infant Feeding

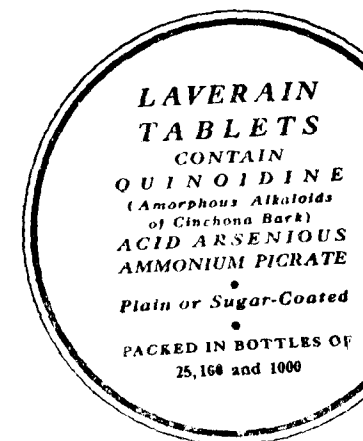
Conflicting views on the relative merits of cane sugar and lactose in promoting weight gain in infants are discussed. According to the author, lactose is superior only when used in a food mixture of about the same composition as human milk. He dismisses the view that B lactose is superior to α-lactose, on the grounds that in the preparation of an infant food with either the α- or B-form, an equilibrium mixture is established containing 60 per cent. of B-lactose. He suggests that the proportion of fat to lactose in the diet is important.

It is known that fat-enriched artificial foods are not always well tolerated, but when they are tolerated the infants so fed nutritionally approach the breast-fed child most nearly. Authors who used diets similar in composition to human milk, with high fat and lactose content, make no mention of diminished fat tolerance, but this is common when cane sugar is given with fat-enriched diets. It has been shown in the Kiel Children's Clinic that diets containing butterfat give excellent results if lactose is included. The diets used contained 500 g. whole milk, 500 g. of 2 per cent maize flour soup, 100 (or 80) g. lactose, and 35 (or 25) g. butter. Lactose gives good results only when used in foods with at least the same fat content as human milk. It should not be used in foods prepared by diluting cow's milk. The main value of lactose lies in its influence on the intestinal processes in creating conditions favourable for acidophile bacteria.—Miller, E. Ueber die Bedeutung des Milchsuckers für die Säuglingsernährung, *Ztschr. Kinderheilk.*, 1947, 65: 269-282 [Kinderklin., Univ. Kiel.]—*Nutrition Abstracts and Reviews*, Vol. 18, No. 3.—M. B. Richards.

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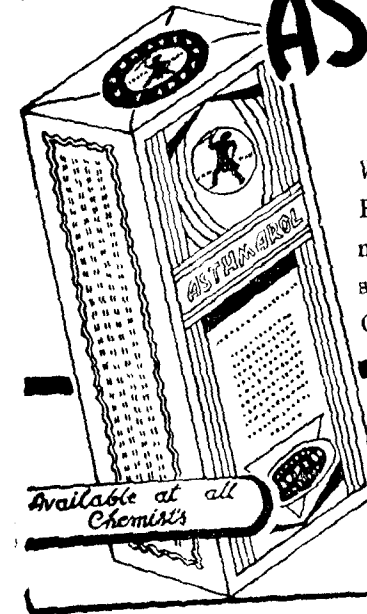
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Food and Nutrition

OUR readers will be delighted to learn that at least now we have been enabled—thanks to the partial removal of the stringent restrictions under the Newsprint Control order—to resume the publication of our Special Numbers. These special numbers deal with the subjects taken up in all their aspects by medical men and women competent and experienced, and they also serve as a mine of information and a refresher for the members of the profession. They were also very highly appreciated by foreign journals as best publications satisfying the thirst for knowledge of the members of the profession.

We have taken up for this Special Number the subject "Food and Nutrition". This subject has assumed considerable importance in recent years owing to the introduction of rationing and the high cost of the essential necessities of life. Even in ordinary times, many people in the country were not having even a square meal a day, that it had become one of the serious charges against the alien administration that it had not only done nothing to solve the problem but also had accentuated its seriousness by its various acts of commission and omission. With the advent of independence from foreign yoke, the public had naturally expected a change for the better; and the promises made by the Congress while it was fighting for independence that it would give top priority to the solution of the poverty problem in the country if it was placed in power, had filled the people with great hope that something tangible will be done to relieve their distress. The Congress has taken up the reins of administration not only in the Centre but also in all the Provinces of the Dominion of India; but circumstances beyond their control have supervened. Even in ordinary times India was dependent upon Burma for a good slice of its rice requirements. With the fall of Burma to Japan that source dried up.

Even after the termination of the war and the restoration of the country to its normal position things have not in any way improved. On the contrary, they have worsened consequent on the chaotic condition created by the Communist revolt.

The food and nutrition problem is not only our country's problem; it is a world problem on the successful and satisfactory solution of which, the peace and happiness of the world depend to a very large extent. That is why special attention was devoted to a national health policy in all advanced countries and its aims and objectives have been declared to be :

"To improve the individual or the community life of the future, to insure a better second generation, and a still better third generation and to promote a healthier and better nation and race."

For the attainment of such an object a balanced diet for all is considered necessary by many eminent men. But in view of the possible delay in the achievement of this ideal due to various reasons it has been suggested by some realists that at least a satisfactory diet must be assured to the people. It is for this reason that the Royal Institute of International Affairs, London, recommended the following ameliorative measures which could help solve the problem within a five year period :

- (1) The use of manures and fertilisers on lands, where water supply is already adequate.
- (2) Improvements in water supply and drainage facilities including contour bounding of fields.
- (3) The use of improved varieties of seeds.
- (4) The control of diseases in crops and of pests in stored grains.
- (5) The control of malaria and other parasitic diseases.

Every country, barring of course those which are under alien domination, is trying to give effect to this plan of action, so as to effect an improvement in the position. We, in India, have also heard much of the "Grow More Food Campaign" during the last years of the alien administration. Even after the advent of independence the talk has not grown less; and the expenditure on such schemes has also mounted up; and yet, the position has not in any way improved; and, to add to this, the failure of the monsoon for two consecutive years in many parts of the country has considerably worsened the situation with the result that the governments had to reduce even the small quantity of rations allowed to individuals to tide over the crisis. The quantity of foodstuffs allowed in rationed areas is hardly sufficient to enable people to keep their bodies and souls together. While the question of eliminating deficiency diseases by adopting suitable and effective measures is being taken on hand earnestly by every advanced country and given effect to as far as possible, our country is yet in the most

primitive stage of having to chalk out measures and plans to assure the people of at least sufficient quantity of foodstuffs to keep them going.

In the circumstances it might appear far-fetched to think of nutritional problems when the people are not even assured of a diet sufficient at least quantitatively to meet their requirements. It cannot be gainsaid that clinical conditions attributable to faulty and deficient dietaries are widespread in the country. As has been pointed out by a learned contributor elsewhere, a fairly correct picture of nutritional diseases in India would not only be of scientific interest, but if put together and presented in a proper form may provoke the authorities to take determined action not only for their eradication but also to take steps for the removal of the prevalence of the background of mal-nutrition among the population which increases its susceptibility to various infectious and dangerous diseases like tuberculosis. It is in this hope that we have devoted this special number to this subject and we trust that the authorities will realise before it is too late that mal-nutrition and under-nutrition are the major public health and clinical problems in the country today and take effective steps to solve them. "Prevention is better than cure."

Gleanings From Medical Press

Food-problems in war-time.—George R. Cowgill, *Yale University Review*, 1943, and *Smithsonian Institute Report*.

The planned feeding of the armed forces in the Second World War afforded a marked contrast to that of the First War (1914-1918). It was then considered that any combination of foods that furnished enough energy, protein, and certain mineral nutrients like calcium and iron would meet all the requirements of satisfactory nutrition. But this will no longer hold good. A rough classification of the requisite factors, that make for adequate nutrition, would now be as follows: (a) food energy measured in calories; (b) protein; (c) mineral nutrients; (d) vitamins. Besides a knowledge of these we now have some extra information bearing on the question of how much food the average adult needs, and the factors that change that requirement. The nutrition expert is frequently asked to say if armed forces fighting in

various parts of the world have different requirements of food, according to the climate or to other factors peculiar to the respective regions. Must the soldiers in Iceland be fed differently from his comrade in Morocco? Marked differences no doubt characterize the food habits of the people inhabiting different parts of the globe. It is, however, erroneous to assume that the nutritive needs of the fighting forces should be readjusted. The kinds of foods eaten by any group of people constitute a reflection of the economic factors prevailing such as availability and relative cost and socio-religious factors involving established customs, religious training, prohibitions and the like. Naturally therefore, foods that are readily produced in a given area will predominate in the dietaries of the inhabitants of those areas over foods that must be imported or obtained with difficulty from distant or neighbouring areas. If differences in food habits do not connote differences in the basic requirements of different places, a basic ration

could serve for all troops irrespective of where they are stationed. The soldier in Iceland must face greater heat loss to the environment and use warmer clothing and heated quarters. The soldier in Libya and his comrades in the humid tropics must face the problem of greater sweating due to the warmth. Workmen in steel rolling mills, and other industrial plants, whose activities result in profuse sweating, often lose so much salt through the sweat as to develop muscle cramps and pains. Tablets of refined salt are administered to them as a routine to replace the loss. Recently it has been learnt that appreciable quantities of various water-soluble vitamins are also lost from the body by way of sweat. How significant this loss may be remains to be determined by research. Conditions in the tropics have played an interesting role in determining the specification of certain emergency rations such, for example, as an experimental chocolate bar, lightly sweetened, designed to be carried in the soldier's shirt pocket to be eaten only as a last resort when separated from his troop units.

The selection of foods for aviators has got its special problems. One that might be mentioned here is the necessity for avoiding foods that readily produce gas. When the pilot and his crew rise to high altitude gases expand in corresponding degree; if much gas is present in the alimentary tract, its expansion can cause considerable discomfort and nausea.

The Axis Nations, particularly Germany and Japan, entered the War with the soldiers well equipped for Blitz tactics and jungle fighting. The Japanese soldier's remarkable equipment includes rice and certain other dry foods valuable in supplementing rice with the nutritive factors it lacks. Dr. T. Saiki, Director of the Imperial Nutrition Institute in Tokyo, by concentrating on the problem of finding new but cheap food resources with which to feed the Japanese masses who are extremely poor, evolved a nutritionally adequate diet that would cost only 5 cents a day. As a result of the War, the shipment of silk from Japan was stopped and they found other uses for the silk-worm. Recently it was learnt that the silkworm cocoon

contains a significant amount of vitamin B₁. This is now being extracted and used as food!! In spite of the varied useful researches being carried out in U.S.A., the author deplores the fact that many valuable foods are not being used in the quantities that their nutritive values warrant. As examples, he gives skim milk powder, peanut-meal, soyabean products, pig liver, and dried yeast. Peanut-oil is expressed and used for soap-making and manufacture of munitions and other products. The meal residue, valuable as a source of protein and vitamins, is not used as food by men, although it has been proved that a tasty bread can be had by incorporating 20% peanut-flour. Animal tissues, left over after extraction of the anti-anæmic principle, are rejected as worthless, while really tests recently made have revealed qualities quite unsuspected hitherto and such as to give them high ratings as foods.

It is becoming increasingly obvious that every citizen should receive sound education in the principles of nutrition and dietetics—how to select the food diet, how to think practically of food energy, protein, minerals and vitamins, in terms of common foods, classes of foods, from which substitutes may be chosen, when told to conserve some foods and how to pack a "lunch a man can work on" and kindred topics. Every citizen today has ample opportunity to learn how, through selection of food, he can in simple fashion utilize to the best advantage any given set of food materials.

T.N.S.R.

Vitamins and their occurrence in foods—(Smithsonian Institute Report, H. E. Munsell.)

The author who is a renowned nutrition chemist in Washington (U.S.A.) in the course of an elaborate thesis on vitamins and their occurrence in foods has set down certain important things to remember, for the information and guidance of the general public. This is what he says:—

"The array of information relating to the vitamins is extensive and complex. Unless one is making constant use of it, it is almost impossible to remember even

the essential details and very few people wish to be hampered by the need of a pocket book in order to remember their vitamins. In the selection and preparation of foods for a diet adequate in vitamin content a few rules or summary statements are usually sufficient. Those given below are suggested as very helpful:—

1. Use a variety of all types of foods giving special attention to the use of milk and milk products, eggs, green leafy vegetables, fresh fruits, all vegetables, lean meats and whole grain cereals and breads.

2. To avoid loss of vitamin value in cooking or dressing for the table:—

(a) Cook foods as quickly as possible.

(b) Use small amounts of water and use any that is left or strained. Special utensils are not necessary for the so-called waterless cooking.

(c) Steaming is an excellent way to cook most vegetables and some other foods.

(d) Do not peel vegetables or cut them up and then let them stand before cooking; cooking them whole and with the outer covering on, helps to preserve their vitamin contents.

(e) Never add soda to vegetables during cooking; it serves no useful purpose but makes for destruction of vitamins.

(f) Cook green vegetables in an open vessel and they will remain green.

(g) Serve foods as soon as possible after they are cooked.

(h) Do not fry foods if they can be cooked in some other way. Frying and roasting are very destructive of vitamin.

3. Give very careful attention to sources of vitamin B₁ in the diet. It is more difficult to obtain an adequate amount of this vitamin than any of the others and it is perhaps the one in which many diets are very deficient. As it is liable to serious loss in cooking (next to vitamin C) overcooking is to be rigorously guarded against.

4. Store foods in closed containers and at low temperatures.

5. Do not chop or crush fresh fruits and vegetables and allow them to stand

60

as the vitamin C content is rapidly lost thereby.

6. Frozen foods have practically the same vitamin content as fresh foods; care should, however, be taken to see that it is not lost in preparation for serving. Do not defrost and allow to stand. If frozen foods are to be cooked, put them on to cook while they are still frozen and use all the liquid.

7. Dried foods are not especially recommended for vitamin value.

8. Canned foods retain vitamin value well with the possible exception of vitamin C, provided they have not been stored too long. To obtain full value, use the entire contents of the can. Canned foods are cooked foods and need no re-cooking.

9. In canning foods observe the same precautions for conserving vitamin content as suggested for cooking. T.N.S.R.

Nutritional requirements of man.

—By C. A. Elvehjem, Abstract from Annual Report of the Smithsonian Institute, Washington, 1945.

The establishment of nutritional requirements has had a rather interesting history. The actual requirement has in a few cases been studied on human beings of different ages. The data on protein requirements are somewhat limited, but there is today general agreement amongst most workers as to this constituent of our diet. Some useful data have been collected on the Ca, P, and Fe requirements of man. In the case of many vitamins, the data have been obtained through animal experiments and often from work not initiated specifically to answer this question. Many are guilty of concluding from animal experiments that a vitamin is needed at a certain level and then for food measure they suggest that the daily human requirement would be 50 per cent greater than the value found. This method would, however, err on the side of excess and safety; but might lead to piling up a considerable and needless excess in some cases.

The following figures may be used as a guide for the daily requirement of the better known nutrients. These values

will vary depending on the age of the person and on superimposed requirements above the normal due to overwork, ill health, pregnancy and lactation. These are, therefore, largely physiological.

Protein	.. 70 grms.
Calcium	.. 0.7 grm.
Iron	.. 12.0 mg.
Vitamin A	.. 4 to 5000 I.U.
Vitamin B ₁	.. 1.5 to 2.0 mg.
Vitamin C	.. 50—75 mg.
Riboflavin	.. 2—3 mg.
Nicotinic Acid	.. 10—15 mg.
Vitamin D	.. 400 I.U.

Elvehjem (a leading authority on the subject) then discusses how these values were obtained, their limitations, and how more satisfactory values may be evolved by continued researches by food chemists.

In spite of the extensive values for the caloric requirements of men, under varying conditions, much attention has not been paid to the effect of external environment on the caloric intake. Thus, excessive external temperatures may reduce the intake of calories to the point that adequate food is not consumed to supply normal vitamin requirements; until accurate values for the amino-acid content of all our food supplies, we have to continue to refer to the protein requirement in total grammes of protein in the diet—which is really not what we are interested in. The value of 70 grammes for protein per day for an adult man, is a safe figure, provided it is derived from a variety of sources, some from animal sources also.

Fat:—No definite figures have been fixed for the fat content of human dietaries. Fat should certainly be a constituent of the normal diet and liberal amounts are advised to supply the fat soluble vitamins; but this latter suggestion is not absolutely essential, as we can now get concentrates of these. The importance of fat as a source of linoleic acid, should not, however, be overlooked. There is some difference of opinion concerning this. A high intake of linoleic-acid-containing-fats has been used in the treatment of eczema. Butter fat is superior to vegetable oils. Fat has a sparing effect on vit. B₁ and

decreases in fat content of dietaries may aggravate the B₁ deficiency, brought about by refined, milled foods.

Calcium: The generally accepted figure as adequate is 0.7 to 0.8 grammes per day. This holds good *only* when the diet contains an adequate supply of vit. D. The optimum Ca: P ratio often found in many textbooks as 2:1, is obviously wrong. A ratio of 1.2:1 is ample according to latest researches.

Iron: The daily requirement is fairly well established as varying from 12 to 15 mg. per day. It should be made quite clear, however, that the iron is of no value to the body *unless* it is accompanied by copper which has a definite role to play in the conversion of iron to haemoglobin. Then the iron must be in an assimilable form. Extensive studies on the iron content of foodstuffs and the values thus obtained check well with those obtained through feeding experiments with rats. We still do not know if the availability as measured in rats is similar to what we may find in humans. Again, *Ferrous* iron, is the only form to be absorbed by the alimentary tract. The ferric iron consumed is reduced in the stomach by substances which are commonly found in the diet. The diet may also contain substances which oxidise the *ferrous* into *ferric* iron. The phosphatides of egg-yolk are substances which probably could inhibit the absorption of iron, through the auto-oxidation of the *ferrous* iron. The value of 12 to 15 mg. per day is liberal enough to compensate for iron not utilized. The daily copper requirement is probably about 2 mg. per day. Elements such as Mn, Zn, and Cobalt are important in the diet and values for these are being worked out.

Iodine:—Most of the work on iodine requirements has been done directly on human beings. It has been found that 50 to 100 micrograms of iodine per day may be sufficient. The value usually given is 2 micrograms per kilogram of body weight (1 kg. 2.2 lb.). Where these small requirements cannot be met by foods or drinking water, salt (sodium chloride) is fortified in America by adding sodium or potassium iodide to

maintain the iodine content of salt at 0.015%.

Vitamins:—The main difficulty with regard to these is that there are different chemical compounds which may have similar physiological effects. The quantitative activity of these different compounds varies considerably.

Vitamin A:—Foods contain both the classical vit. A, and carotene as well as other related carotenoids. A value of 5,000 I.U. has been suggested on the basis that the average diet supplies a third of the total activity as vit. A and the remaining two thirds as carotene. 2,000 I.U. of vit. A as such will undoubtedly meet the daily needs. The absorption of vit. A from the intestinal tract is definitely related to the fat content of our diet; the actual requirement will differ drastically depending on the presence of other materials in the diet.

Vitamin D:—This depends on the total Ca and P intake as well as the Ca: P ratio. The quantity of milk consumed will largely decide this factor. It will be safe to accept a value of 400 I.U. per day.

Vitamin K and E:—Under normal conditions there is sufficient synthesis of vit. K in the human intestinal tract to meet this requirement. Changes in the intestinal flora might make it necessary to supply preformed vit. K in the diet.

Vitamin C:—It is possible to determine this factor by simple methods not only in foods but also in the blood stream. 70 mg. of vit. C has been found by several groups of workers to maintain the vit. C content of the blood at high levels. The important question is whether it is necessary to supply sufficient vit. C to produce complete saturation of the blood stream or whether optimum health can be maintained at lower levels of saturation.

Vitamin B₁:—This has been the subject of great interest at present. It has not yet been feasible to study the vitamin B₁ content of the blood, so we have to rely largely on balance studies. A high fat diet reduces the vit. B₁ requirement. Recent studies in the Banting Institute have suggested that high fat diets may eliminate all symp-

toms of B₁ deficiency except those changes taking place in the heart. If this is true, the intake of B₁ should be liberal and adequate regardless of the fat intake. 1.5 to 2 mg. per day will be adequate for most individuals.

Riboflavin:—Recent researches have shown that 3 mg. per day will be adequate. The studies were conducted on human beings receiving average diets, but again changing the fat and carbohydrate ratio may alter the riboflavin requirement. An increase in the fat diet produces considerable increase in the riboflavin requirement. Under normal conditions there may be a certain degree of riboflavin synthesis in the intestinal tract and we are measuring the riboflavin required above this synthesis. Sudden changes in the intestinal flora necessitate the supplying of the entire riboflavin requirement through the diet.

Nicotinic acid:—Tremendous difficulties have been experienced in attempts to determine the nicotinic acid content of foods or to measure the quantity excreted in urine. Using the results of animal feeding experiments as basis it has been laid down that the daily human requirement is about 15 mg. The average human diet has been calculated to contain about 20 mg. of nicotinic acid. Bacteriological methods for nicotinic acid determinations have recently been evolved and these have been found to yield satisfactory results.

Vitamin B₆:—This is as important as any of the other factors but there is no way at present of fixing a specific figure. It is, however, safe to assume that the B₆ requirement is of the same order as B₁.

The values laid down, though based largely on feeding experiments on animals of different species, are still of great importance in constructing adequate diets, provided we still obtain a considerable proportion of the nutrients from natural foods. T.N.S.R.

Vitamin B-complex—A resumé of present knowledge.—A general summary of the more significant

facts relating to the vitamin B complex, has been prepared at the request of the Council on Foods and Nutrition of the American Medical Association by Dr. C. A. Elvehjem, D.Sc., of Wisconsin. This exhaustive summary stresses the importance of these vitamins to nutrition and brings into a single compass all that has so far been authentically published on the basis of extensive researches, animal and human experiments. It is, therefore, bound to be of great and abiding interest to readers of the ANTISEPTIC.

The B complex is now known to consist of at least a dozen separate factors, ten of which have been obtained in crystalline form. The following concise abstract of the above paper which covers nearly 24 closely-printed columns of the *Journal of the American Medical Association* (27-11-'48) will give the reader all that there is to know (about each of these ten ascertained factors comprised in the B-complex) regarding mode of occurrence, result of deficiency, minimal human requirements, and general reactions.

B₁, Thiamine:—Thiamine hydrochloride is a white crystalline substance readily soluble in water, having a nut-like and salty taste and a yeast-like odour. It is rapidly destroyed in neutral or alkaline solutions but in acid solutions it stands sterilization at 120°C for 30 minutes. In the dry form it is very stable and not readily destroyed by oxidation. In foods and tissues it occurs in the free form and as thiamine pyrophosphate or cocarboxylase. In thiamine deficiency the utilization of carbohydrate is incomplete and pyruvic acid accumulates in the tissues. The symptoms of deficiency depend to a great extent on the rate at which the deficiency develops, but in all cases the subjects become depressed, irritable, quarrelsome, fearful, and uncooperative. A relationship exists between thiamine deficiency and cardiac function. Anaemia of the hyperchronic type and the "beriberi heart," have also been described in a number of thiamine-deficient patients.

The daily average thiamine allowance for an adult man has been fixed at 1.2 to 1.8 mg. by the Food and Nutrition

Board of the National Research Council. Some recommend 1 mg. as sufficient. Active growth periods, pregnancy and lactation require larger amounts as also pathologic conditions like fever hyperthyroidism.

Only a few foods can be classified as rich sources of thiamine; they include fresh peas, beans, oatmeal, whole wheat, peanuts and lean pork. The staple foods like milk, vegetables and fruits contribute smaller but appreciable amounts of thiamine in the diet. Significant amounts are lost during preparation of foods, as moist heat readily destroys them. Thiamine is not destroyed in the digestive tract under normal conditions; intestinal disturbances might retard its absorption. Thiamine has been proved to be synthesized by the bacterial flora of the gastro-intestinal tract, but only to a lesser extent than some of the other B-vitamin factors. Recently a factor destructive of thiamine has been found in raw fish. This inactivation is considered to be due to the action of an enzyme.

B₂, Riboflavin:—It is a practically odourless, orange-yellow crystalline compound which shows an yellowish-green fluorescence in aqueous solution. Riboflavin is heat-stable, especially in acid media, but extremely labile when exposed to light. In nature it may occur as such as riboflavin phosphate, or as a constituent of specific flavoproteins, which function as valuable enzymes in tissue respiration.

The symptoms of riboflavin deficiency include inflammation of the lips, fissures at the corners of the mouth, glossitis, dermatitis, and vascularizing keratitis. The ocular symptoms appear to be constant and to precede all others. There is still considerable difficulty in distinguishing the glossitis or riboflavin deficiency from that of pellagra, pernicious anaemia or iron deficiency, and in distinguishing the ocular ariboflavinosis from other conditions involving corneal vascularization.

Riboflavin is widely distributed in plant and animal materials. Liver, milk and green leafy vegetables are the best and most dependable sources in the

human dietary. Seeds, which are so important as a source of thiamine, are poor in riboflavin.

The Food and Nutrition Board of the N. R. C. recommends 1.8 mg. of riboflavin as the daily allowance for men. There is also considerable evidence for the bacterial synthesis of riboflavin in the intestinal tract and some of this can be utilized. Riboflavin is believed to exert a pronounced beneficial effect on the economy of food utilization for the synthesis of the body tissues. The biological value of the proteins in bread is greatly improved in the presence of extra B-vitamins.

Nicotinic acid occurs as white needle-like crystals, non-hygroscopic and stable in air. The sensation of flushing and erythema of the skin often noticed on the administration of nicotinic acid is not produced by the amide of the nicotinic acid. Nicotinic acid is a weak acid and its alkaline salts in solution show but a slight alkaline reaction. It stands sterilisation under pressure when in solution and shows no loss of potency when exposed to dry heat.

In the human body nicotinic acid functions as two important co-enzymes, similar, instructive, but differing in the phosphoric acid units. In nicotinic acid deficiency a decreased cozymase content of the liver and muscle tissue is to be seen. The early symptoms of pellagra are weakness, lassitude, anorexia and indigestion, followed by sore and ulcerated mouth and diarrhoea. The typical dermatitis simplifies diagnosis usually. In endemic pellagra areas even little children suffer from nicotinic acid deficiency. Nicotinic acid is widely used in treating pellagra, but its use is most successful in conjunction with other vitamins and specific natural foods.

The best sources of nicotinic acid include liver, yeast, lean meats and, to a lesser extent, potatoes, peanuts and vegetables. The Food and Nutrition Board has fixed 12 to 18 mg. as the daily allowance for an adult man. Wheat contains 5 to 7 mg. per 100 gms. a large quantity of which is lost in milling. Nicotinic acid is a stable compound. No loss occurs in cooking (if the cooking

water is not discarded). Milk and eggs are low in nicotinic acid, but carry a good supply of Tryptophan and so are important in the prevention of pellagra.

B-6, Pyridoxine hydrochloride:—It is a white crystalline powder slightly better in taste, but odourless. In 1941, it was shown that two derivatives of pyridoxine, *viz.*, pyridoxal and pyridoxamine were more active for the growth of certain lactic acid bacteria, than the original pyridoxine.

No clear cut symptoms resulting from pyridoxine deficiency have been described in human beings; but case reports have been made of additional and more rapid improvement in pellagrins with pyridoxine therapy after treatment with nicotinic acid, riboflavin and the amine. Rapid healing of the typical lesions in cheilitis followed pyridoxine therapy.

The vitamin B₆ requirement has been put down as 1.5 mg. a day. The best sources of this factor B₆ are rice bran, liver, yeast, cereals, legumes and milk. Whole wheat contains 0.4 mg. in 100 g. Swaminathan (*Indian Jour. Med. Res.*, 29:561, July 1941) found that diets normally eaten by the Indians supplied 3.5 to 5.0 mg. per day.

Pantothenic acid called also the "filtrate factor" of vitamin B-complex, is now available as the dextrorotatory calcium salt which occurs in dense fine crystals, white in colour, odourless and slightly bitter in taste. It is fairly stable to moist heat but destroyed by prolonged dry heat. Recent researches have shown that pantothenic acid is related to enzymatic acetylation. It was shown that a coenzyme was present in liver preparations, which was essential for the acetylation of aromatic amines. The same co-enzyme was then found to be necessary for the acetylation of choline in brain tissue. This coenzyme was next prepared in concentrated form and was found to contain 10 per cent pantothenic acid.

This factor has now been demonstrated to be essential to the nutrition of human beings. As this factor is widely distributed in nature and in the human dietaries, even restricted diets may not be low enough in this factor to cause a deficiency. Liver is one of the richest

natural sources of this vitamin. Meats, cereals and milk are also good sources. Evidence has been adduced to show that only about one half of the pantothenic acid in whole wheat is lost during the milling process.

Choline is a colorless viscous fluid and the more familiar choline chloride is a very hygroscopic crystalline substance with a salty bitter taste. It is not stable to alkali treatment but is very stable to acids even at high temperatures. It is a component part of the phospholipid lecithin and its function has now been more or less well established, as being related to the mobilization of fatty acids in the body. It has been estimated that the choline intake in the normal human diet may range from 250 to 600 mg. The best dietary source of choline is egg-yolk while soyabean meal, dried yeast, brain, kidney pancreas and liver are good sources. Most edible fats are low in choline. Choline is present in food materials in the complex form of phosphoric acid esters. Most diets contain sufficient choline and its precursors to meet normal human needs. Evidence is accumulating that some types of clinical cirrhosis are favourably affected by dietary treatment including choline and other vitamins along with a high protein diet.

Biotin was first recognized as a growth factor for yeast and was later identified with the anti-egg injury factor or vitamin H in animal nutrition. It is a very stable compound, resisting heat at high temperatures. Pure bio in shows appreciable liability to alkali. Both the free and bound biotins are inactivated by oxidising agents.

Recent work has established that biotin functions in the carboxylation of pyruvate to form oxalacetate. Dermatitis and changes in the colour of the skin responded to biotin. It is ubiquitous in distribution but liver, yeast, kidney and egg yolk are the chief sources. Biotin is present in most tissues in a "bound state" in which it cannot be extracted by hot water.

Inositol is a crystalline substance with a sweet taste comparable to the cyclic form of glucose. It is a very stable

compound resisting strong acid and alkali treatments. It is found in plants where it occurs in free form, and as "phytin", a calcium magnesium salt of inositol phosphoric acid. In the animal body it is found in muscle (accounting for the term muscle-sugar), brain, blood erythrocytes and eye tissues and as "phytin" in R.B.Cs. of chicken and turtles. The spleen, heart, brain, kidney, thyroid and testes are specially rich in inositol content. Regarding the role of inositol in human nutrition recent reports show that inositol improves the action of tocopherol in the treatment of progressive muscle dystrophy. It is believed to form a complex with tocopherol in the intestinal tract and this complex is essential for normal creative metabolism.

Para-aminobenzoic acid: It was first recognized as an essential metabolite when the antagonistic action between this compound and sulphanilamide was discovered in 1940 by Dr. Woods. The possible role of para-aminobenzoic acid in the treatment of grey hair in human subjects has been studied and found to have some measure of support. The most striking observation regarding this metabolite is its chemotherapeutic effect on typhus and Rocky Mountain spotted fever (1946).

Folic acid—It is a yellow compound sparingly soluble in water and has three different components: the pteridine part, the para-aminobenzoic acid and glutamic acid. The part consisting of the first and second is called pterotic acid.

Folic acid is widely distributed in nature both in free form and in compounds with added glutamic acid. Most knowledge concerning the function of folic acid has come from studies on bacterial metabolism. Greatest progress in the isolation of this vitamin was made when it was recognized that a growth factor was needed for the growth of *Lactobacillus casei* and related organisms. The activity of synthetic folic acid in various types of macrocytic anaemia in relapse has been demonstrated. Commercial preparations of the classic pernicious anaemic factor contain practically no folic acid, and a pure substance vitamin B₁₂ has now been

isolated and is very effective in small amount in the treatment of pernicious anaemia.

The results of a preliminary report (1947) on the folic acid content of foods show that liver and deep green leafy vegetables (spinach, amaranth etc.) to be very high and green vegetables, cauliflower, kidney, muscle meat, and wheat cereals to be high in folic acid values. The loss of folic acid in cooking may be very high.

The availability of the several synthetic water-soluble vitamins has been of great value in chemical practice. But the common natural foods still remain the best source of these vitamins in practical nutrition. Proper selection is however essential for ensuring the availability of all factors in required amounts. Modern methods of processing and overcooking for satisfying the palate, are to be carefully watched and controlled.

T.N.S.R.

Parboiling treatment of rice.—(Nature: 162, 913-15, 11-12-1948). Dr. J. J. C. Hinton of the Cereal Research Station, St. Albans, Hertford, England.

(The treatment of paddy with hot water or steam, for parboiling before hulling is an ancient and established commercial practice in India and other rice-eating countries of the world. Prior to the advent of rice-mills which mill raw rice and also parboil paddy and then convert it into rice, paddy was parboiled in homes and then handpounded. Parboiling has since been found by scientific experiments to fix the vitamin B to the rice, which otherwise was apt to be destroyed by overhulling and polishing.)

The distribution of vitamin B₁ within the rice grain, is very similar to its distribution in wheat, and about 50 percent is found in the scutellum. In parboiled rice, the scutellum portion of the germ appears to remain in the grain, in contrast to untreated raw rice. Hinton carried out a series of interesting experiments, using carefully conceived delicate tests, on freshly treated samples of parboiled rice. Small samples of paddy were kept soaked in water and heated to 75°C for

4 hours, dried on a cloth, and steamed in an autoclave for 15 minutes at one atmosphere (15 lb. per sq. in.): 15 to 20 grains were dissected by first removing the embryo and scutellum. The end of the grain was then cut off and the pericarp, aleurone layer, and endosperm removed from the centre portion of the grain. The outermost 1-yr (15 to 20 percent) of the endosperm is described as No. 1, and the rest as No. 2. The vitamin B₁ content of these several portions was then carefully assayed. The results are set down below:—

No.	Sample	Vitamin B ₁ content (I.U. per gram) of various samples			
		Embryo	Scutellum	Pericarp aleurone layer	Endosperm No. 1 No. 2
1	Raw rice	23.0	63.0	10.5	0.10
2	4 hours after parboiling	5.9	0.4	0.5	0.45
3	Do.	Nil.	Nil.	0.6	0.56
4	72 hours after parboiling	Nil.	Nil.	2.3	0.54
5	12 days after do.	Nil.	Nil.	0.9	0.73
6	4 hours after do.	21.4	11.2	3.6	0.40
7	Heated to 75°C for 4 hours and not steamed.	24.4	30.7	6.2	0.88
8	12 hours after parboiling	3.2	2.1		0.80
9	23 days after do.	5.3	2.8		0.77
10	23 days after do.	6.4	3.8		0.71

These results show that the parboiling leads to a re-distribution of vitamin B₁ in the grain, with the result that the endosperm is considerably enriched. Simple steeping in hot water (the method used in Indian village homes) alone enriches the endosperm appreciably. There is in addition a marked retention of the scutellum in milled parboiled rice as compared with milled raw rice, due largely to the gelatinization of the endosperm immediately beneath the germ, which acts as a cementing layer, so that the germ is not readily knocked out or dislodged during the subsequent milling.

This retention may be responsible for the higher vitamin B₁ content of par-boiled rice. The results of sample No. 7 show clearly that mild soaking in hot water (the Indian traditional household method) followed by light milling is calculated to improve the adhesion of the germ by partially gelatinizing the endosperm beneath, without seriously affecting the vitamin B₁ content.

[This investigation throws light on and seeks to offer a scientific explanation for the well-established and long-known fact that parboiling of paddy prior to milling helps the retention of the vitamin B content and the germ in tact. This and other valuable observations are embodied also in the interesting and exhaus-

tive "memoir on rice", published by the Indian Research Fund Association about twenty five years ago as a separate monograph.] T.N.S.R.

Food from grass.—A method of extracting important food elements from grass, which, will provide millions of people with an excellent diet at a very low cost, has been perfected by Dr. C. F. Schnabel, according to whom a full day's ration for an adult can be produced for 16 cents (9 d) [about 10 annas at the present rate of exchange]. (*Reuters Trade Services cited in Food Manufacture London, Oct. 1948, p. 487.*) T.N.S.R.

Book Review

Nutritional Diseases in India—By R. PASS-MORE, M.A., D.M. Oxford, Capt., Indian Medical Service, Nutrition Research Laboratories, Indian Research Fund Association, Coonoor, S. India. 1948, pages 128. (U. N. Dhur & Sons, Ltd., Calcutta.) [Price Rs. 10 net.

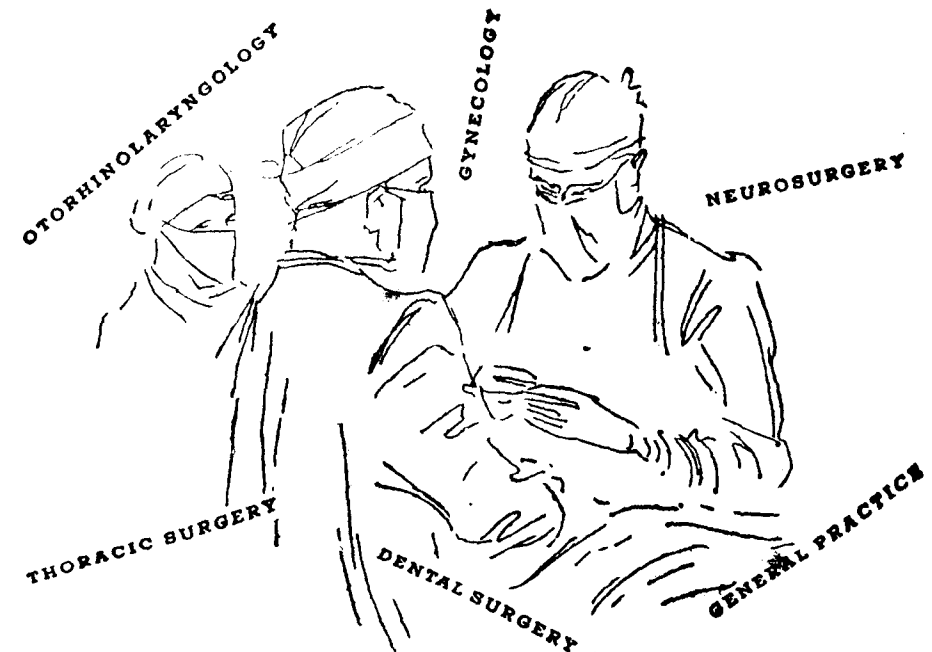
"The first essentials for the prevention of disease are a higher standard of health, a better physique and a greater power of resistance to infection. These can only be obtained if the food of the people is such as will give all the physiological and nutritional requirements of the human frame." All medical men will agree with this statement. In India where the population is broken by poverty and malnutrition, many diseases flourish. The author who has many years of experience on the staff of the Indian Research Fund Association Laboratories at Coonoor has attempted in this little monogram to describe the signs and symptoms of mal-nutrition and deficiency diseases so that these conditions can be readily and accurately diagnosed and treated. The public health importance of nutrition is stressed and the relevant epidemiological factors and their relation to social conditions are described and discussed.

The book is divided into six chapters. The first chapter deals with the principal causes of sickness and death in India and their relation to diet. The second chapter deals with Nutrition. In this chapter, the author gives a general definition and description of mal-nutrition, its etiology, general features etc. The nutritional disorders of the skin such as Hyperkeratosis, Angular Stomatitis, Genital Eczema, Nutritional Disorders of the Eye, such as Xerophthalmia, Superficial Keratitis,

and the Diseases of the Digestive System such as Glossitis, Diarrhoea, and of the Cardio-vascular System, and the Nervous System are described very thoroughly. Details with regard to their treatment are also included in this section. The third chapter is devoted to the description of specific deficiency diseases, such as Beriberi, Pellagra, Scurvy, Rickets and Osteomalacia. In this chapter, the etiology, epidemiology, clinical features and treatment are given in detail. The fourth chapter deals with anaemia in relation to nutrition, with special reference to Anaemia of Pregnancy, and Anaemia in Tea Estate Labour. The fifth chapter is devoted to a description of diseases probably due to food poison, such as Lathyrism, Epidemic Dropsy. The sixth chapter is devoted to a description of the miscellaneous diseases in which errors in diet may be a contributory factor, such as Tropical Ulcer, Goitre, Peptic Ulcer, Diabetes, Cirrhosis of the Liver, and Urinary Stone.

There are useful appendices giving Tables of Food Analyses, Summary of Dietary Requirements, Summary of the Properties of the Important Vitamins, Hospital Diet Scales, Menu of a special invalid kitchen. The hospital diets are those followed in the Army. In this book the clinical aspects of the subject have been cut down to a minimum. Emphasis is laid throughout on food deficiencies, rather than on vitamin deficiencies. Controversial subjects have been reduced to a minimum. The general set-up of the book is very good, and the photographs illustrating some of the deficiency diseases are very useful. We are sure it will be a useful monogram on Nutritional Diseases in India, suitable to students and practitioners.

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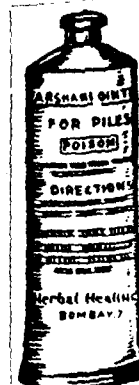
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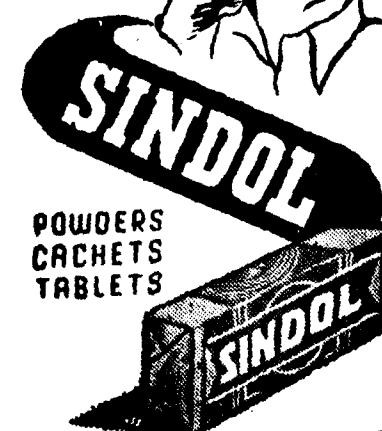
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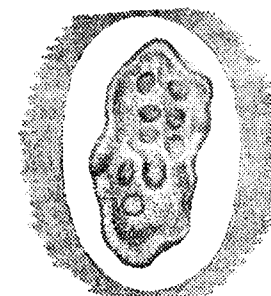
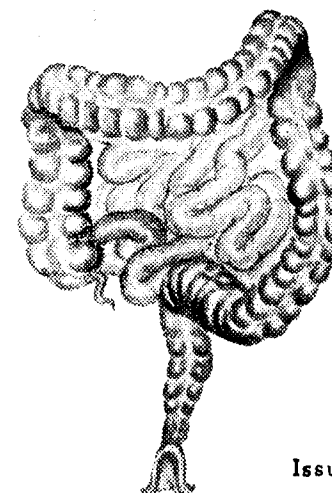


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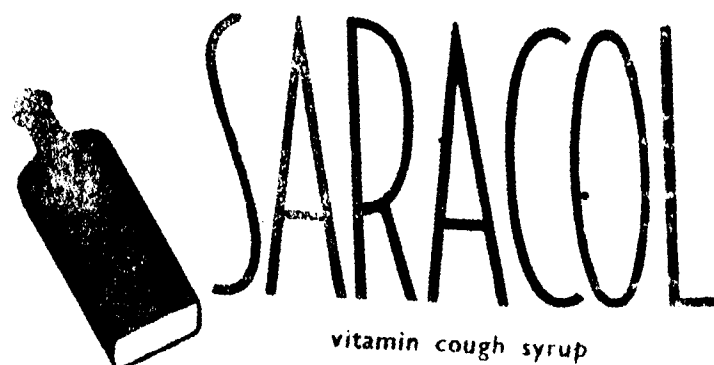
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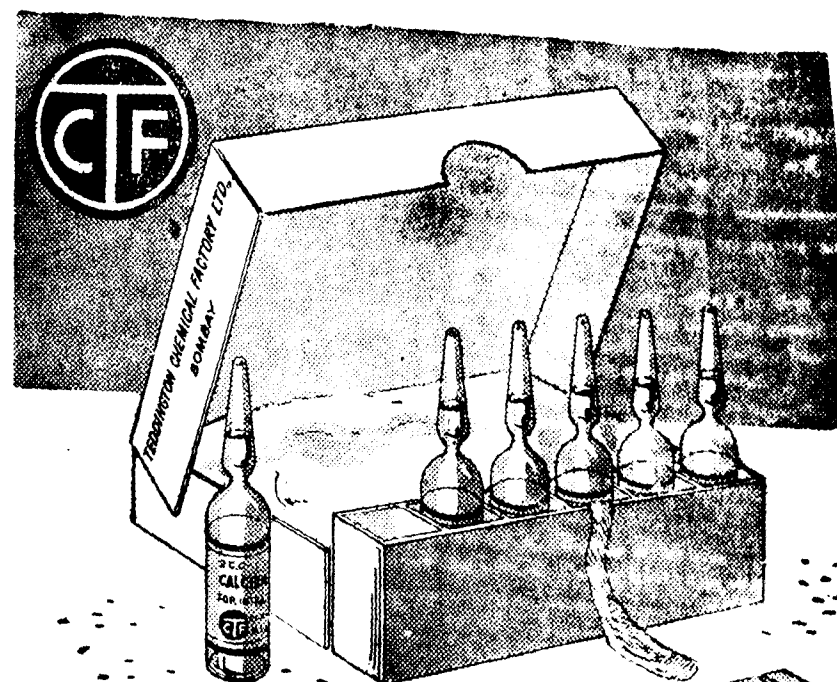
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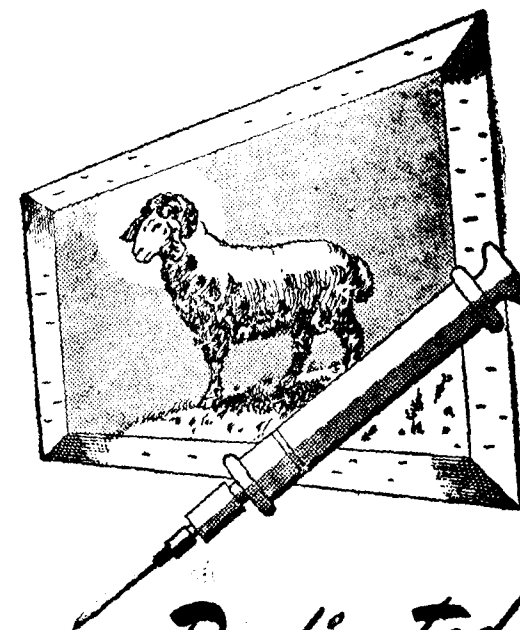
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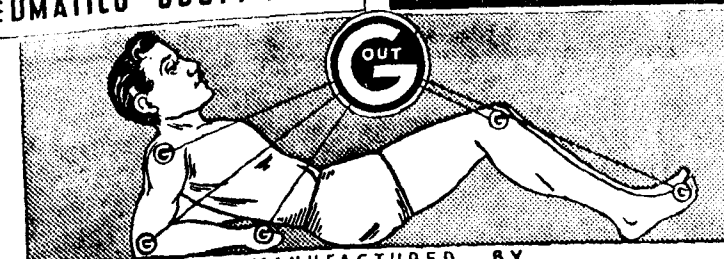
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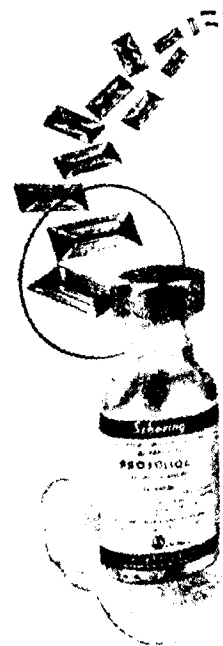
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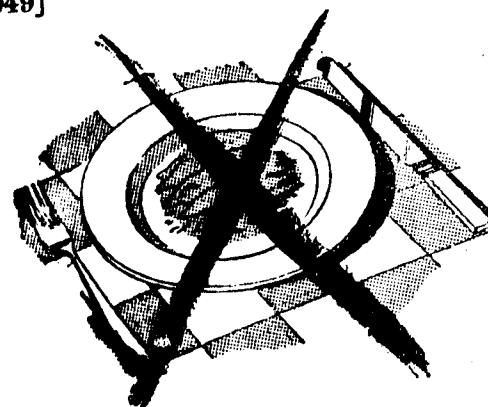
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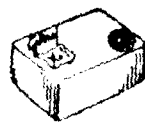
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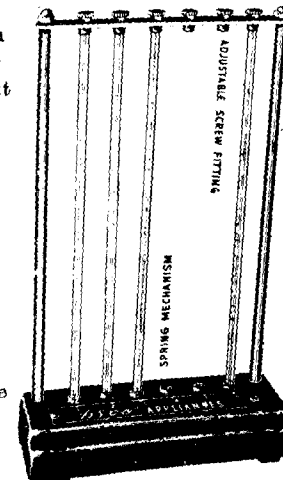
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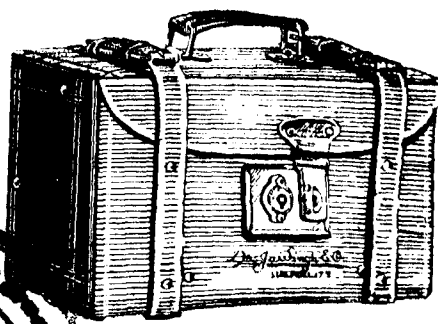
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