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EDITORIAL

Though practised since pre-historic ages, the specialty of neurosurgery is less than 100 years old. It was enabled to develop by the increasing knowledge of neuroanatomy and neurophysiology and by the achievements in anæsthesia and asepsis. The discovery of the excitable cortex by von Fritsch and Hitzig, subsequent research by Ferrier, and the clinical studies of Hughlings Jackson permitted the more accurate localisation of cerebral lesions. Operative techniques were then developed to permit the successful application of this knowledge to the healing or palliation of those patients previously considered to be hopelessly afflicted. The nature of the disease processes were clarified. Men such as Sir Victor Horsley, Sir Geoffrey Jefferson, Harvey Cushing, Walter Dandy and Percival Bailey created the specialty. They correlated the knowledge which the basic sciences offered with the clinical course and pathology of the diseases. They developed new techniques and the operations which they carried out frequently resulted in new knowledge of the nervous system. The specialty is still growing, like all fields of medicine, for many diseases such as malignant tumors and vascular accidents still give disappointing results. Neurosurgery is beginning to converge once again with the fields of neurology and psychiatry. All inquire, directly or indirectly, into Man's nature and his behaviour, and all seek ways of remedying him of the physical limitations of his existence. The results of the research activities of workers in these fields will be fruitful in the years ahead. It is a very exciting period in medicine and particularly in these three specialties. The common bonds between them are very strong and our knowledge of man and the treatment of his ills will greatly benefit by their close co-operation.

In Asia neurosurgery first developed in Japan and Australia and on a small scale in China. Subsequently, units were established in India, Pakistan, Persia and the Philippines. Neuro-surgical procedures have been carried out from time to time in a number of other countries but not in an organized manner and usually not by trained neurosurgeons. Neurosurgery is a distinct specialty, for it requires considerable knowledge of those aspects of anatomy, physiology, pathology and radiology having to do with the nervous system, and the application of this knowledge to the surgical alleviation of diseases of the nervous system. Training in neurosurgery requires much more than the developing of those manual skills of technique. It requires of the doctor the slow accumulation of information and judgment. And it requires a never-ending curiosity about the nervous system and its afflictions.

An awareness of the need to develop neurosurgical services in the Federation of Malaya prompted the recommendation that the Ministry of Health establish such a unit. In August, 1963, with the aid of CARE-MEDICO, Inc. a neurosurgical unit was opened at the General Hospital in Kuala Lumpur. CARE/MEDICO is an American/Canadian organisation sponsored by public donations which enables volunteer doctors and nurses to go to those countries where assistance is sought. Since August the unit has grown considerably. A theatre and basic instruments have been obtained. By the end of March, 1964, two hundred and thirty seven procedures had been carried out. This figure is comparable with the number of procedures done in some of the larger university centers in the United States. Arteriography, myelography and pneumœncephalography are done routinely. Though cranial and spinal trauma constitute about 30% of the patients many cases of brain and spinal cord tumor have been encountered, though frequently advanced and associated with blindness or paraplegia. And yet this is to be expected. Over the following years such lesions will be diagnosed earlier. At the present time pre-operative and post-operative care is improving and there is a slow but heartening decline in the mortality rate. It is hoped that a ward for the neurosurgical unit will be obtained by the end of the year which will result in considerable improvement in the services of the unit. These improvements are permitting more successful attacks on major lesions.

The aim of the unit is not only to provide neurosurgical services but to provide the means whereby Malayan doctors and nurses will be able to assume complete direction and

responsibility for the unit by 1969. Two nurses have been sent to Montreal for a year to learn neurological and neurosurgical nursing. It is planned to send one every two years. It is also planned to send five neurosurgical trainees overseas for a complete course of training, in the hope that by 1973 there will be five neurosurgical centers in the Federation.

The neurosurgical unit here is in its infancy. Much work and a number of years will be required to build it. In the years ahead it is hoped that the development of neurosurgery will make a significant contribution to the medical services of this country and will achieve results which will contribute to the growth of the specialty throughout the world.

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HÆMOPHILIA DUE TO FACTOR VIII DEFICIENCY IN AN INDIAN BOY

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Hæmophilia is a well-known inherited disease in Europe, but it is thought to be very rare in Asian countries.

It is a disease characterized by a life-long tendency to prolonged hæmorrhage and markedly delayed coagulation time in affeced males. It is due to a plasma factor deficiency, factor VIII, also called "anthihaemophilic globulin (=AHG). It is inherited as a recessive Mendelian trait and is sex-linked.

Recently, similar inherited coagulation defects were described, resembling hæmophilia. They were due to a deficiency in other factors or due to circulating anticoagulants. These conditions were given different names such as pseudohæmophilia, parahæmophilia, etc. A disease resembling almost completely the classical hæmophilia, is factor IX deficiency, called hæmophilia B. Much confusion has arisen from this variety of names and it has therefore been proposed to designate the conditions by the name of the coagulation factor which is lacking. Since it is of great importance for the treatment and management of the disease to know exactly what is lacking, attempts should always be made to arrive at an accurate diagnosis as regards the deficiency state.

In this paper a case of classical hæmophilia due to deficiency of factor VIII in an Indian boy is described.

METHODS

Routine hæmatological examinations were carried out according to standard methods.

The blood for study was obtained from the antecubital vein with a new disposable Monoject needle without traumatisation in order to prevent contamination with tissue thromboplastin. The two syringes technique was employed and all syringes were siliconized in order to prevent the activation of certain coagulation factors by contact with glass. The quantity of blood was calculated in advance and all necessary equipment was kept ready in order to avoid undue delay.

As a control, the blood of a normal healthy person was used whose blood was known to us by previous examinations.

Bleeding time was studied by the method of Ivy (1935). With this method the normal bleeding time is 1 to 9 minutes.

Capillary fragility test was that of Rumpel Leede, using a tourniquet to study the resistance of the capillaries.

Coagulation time was done by the method of Lee and White (1913), using glass and siliconized test tubes. Our normal values obtained with this method are 6 to 12 minutes in glass and 25 to 35 minutes in siliconized tubes.

Clot retraction. The simple qualitative (Budtz-olsen 1951) and the quantitative test of Didisheim (1961) were employed.

Clot lysis was evaluated as recommended by Wintrobe (1961), by tilting the tubes, used for the estimation of coagulation times, 90 degrees at 8, 24, 48 and 72 hours. If a clot was found initially and subsequently the blood has become completely fluid, lysis has taken place. This does not occur within 72 hours in normal blood.

Prothrombin time. The one-stage plasma prothrombin time of Quick (1945) was employed, using a commercial thromboplastin made from rabbit brain (Difco-Bacto-Thromboplastin).

Plasma fibrinogen was estimated as described by Ratnoff and Menzie and modified by Holburn (1955).

Thromboplastin generation test. The rapid screening test for disorders of thromboplastin generation as modified by Hicks and Pitney (1957) and the modified thromboplastin generation test of Biggs and Douglas (1953) using inosithin instead of platelets were employed. This test reveals any deficiency due to factor V, VIII, IX and X as well as Hageman factor and PTA.

Test for anticoagulants was performed as described by Wintrobe (1961).

CASE REPORT

Prak., a 5½ year old Indian boy has suffered from easy bruising and a tendency to prolonged haemorrhage since 6 months of age. In 1960 when he was 1½ years old, following a cut on his tongue, the bleeding was so prolonged that a blood transfusion had to be given. However, after a transfusion of 2 pints of blood, the bleeding did not stop and the condition became critical. He was transferred from Kuantan to Penang where plasma and another blood transfusion were given before the bleeding stopped. He had never had joint bleeding. He was the only son in the family, a younger sister was healthy and had no bleeding tendency. No other members were known to suffer from easy bleeding. On December 11, 1963, we saw a resonable well nourished boy, who did not look anæmic but who showed bruises on body, arms and legs. According to the father these bruises were obtained from slight knocks and bumping. Other physical findings were normal. Spleen and liver were not enlarged. Hæmatological findings were as follows. Hb 14.3g%, RBC 6.04 per cmm, WBC 9600 per cmm, PCV 41.5%, MCV 68.7, MCH 23.7 uug, MCHC 34.5%. Platelets 270000 per cmm. Reticulocytes 4.2%, differential count of the leucocytes normal.

Specialized studies. The bleeding time was 6 min. Tourniquet test of Rumpel Leede was negative. Coagulation time was very much prolonged, it was 47 minutes in glass and 7 hours in siliconized tubes. The normal control showed a coagulation time of 10 minutes

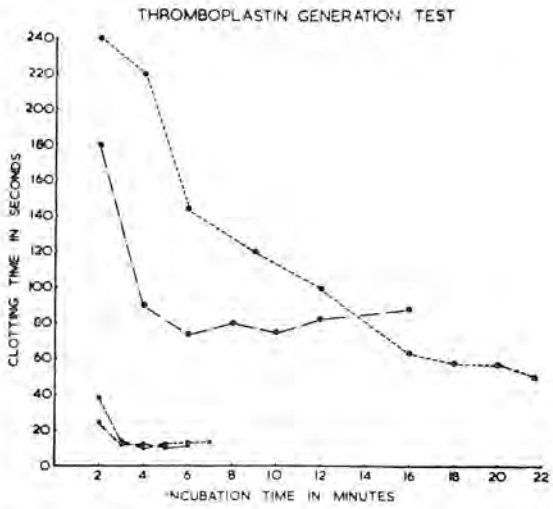

Fig. 1. Showing the abnormal thromboplastin generation test in patient Prak. when patient's absorbed plasma was incubated either with his own serum or with normal serum.

o - - - - - - o Normal absorbed plasma + normal serum.

o - · - · - o Normal absorbed plasma + Patient's serum.

· · · · · · · · Patient's absorbed plasma + Patient's serum.

· - · - · - · Patient's absorbed plasma + normal serum.

in glass and 30 minutes in siliconized tubes. Clotting time of recalcified plasma was 11 minutes and that of the normal control was 3 minutes. Clot retraction was normal. Although the clotting time was very much prolonged, the clot retraction was satisfactory once the clot was formed. Clot lysis was also normal, taking place only after 72 hours. Prothrombin time was 13.6 seconds (75%), normal control 13.7 seconds (75%). Fibrinogen content of the blood was 276.5mg% (normal range 200—400mg%). Protrombin consumption was very poor. The rapid screening test for disorders of thromboplastin generation of Hick and Pitney was found to be grossly abnormal, so the thromboplastin generation test of Biggs and Douglas was carried out to determine the exact nature of the deficiency. This last test showed that thromboplastin formation was deficient when patient's absorbed plasma, patient's or normal serum, inosithin and Ca Cl_2 were incubated together (see fig.). It was normal when patient's serum or normal serum and normal absorbed plasma, inosithin and Ca Cl_2 were incubated. This test points

to a deficiency of either factor V or factor VIII. No circulating anticoagulants were detected.

DISCUSSION

The thromboplastin generation test showed that either factor V or factor VIII was deficient. Since the prothrombin time was normal, factor V was not deficient. Therefore the deficiency was in factor VIII. In agreement with this, is the abnormal prothrombin consumption test. No deficiency of any other coagulation factors could be demonstrated and the platelet number and capillary resistance were normal. The patient is therefore a classical type of hæmophilia due to factor VIII deficiency. Recently cases of hæmophilia were reported which were due to circulating anticoagulants directed against factor VIII. (Horowith and Fujimoto 1962, Ehrenworth, 1963). However, no circulating anticoagulant could be demonstrated in the patient. A condition resembling almost completely the classical type of hæmophilia is that due to factor IX, (Aggeler *et al* 1952), also called hæmophilia B or Christmas disease, (Biggs *et al* 1952). Factor IX, also called PTC (plasma thromboplastin component) is found in serum. Our patient's serum incubated with normal absorbed plasma, inosithin and Ca Cl_2 showed normal thromboplastin generation, which demonstrated that factor IX was not deficient in the patient. The father and mother did not show prolonged coagulation times. It is a pity that no maternal uncles could be included in the study.

This is the first case of hæmophilia due to factor VIII deficiency described in Malaya.

Up to now no therapy has been found which can cure hæmophilia. However, once it is known exactly which factor is lacking in the patient, the management and symptomatic treatment can be carried out more effectively. The patient must of course be protected from wounds and abrasions and he should be warned not to cut or bruise himself. However, it is important that the patient lives normally and does not become an invalid. Operations should be avoided. If this can not be avoided the factor VIII level in the blood should be raised before operation takes place. In a bleeding crisis the basis of therapy should be the administration of a sufficient quantity of antihæmophilic factor to restore the normal coagulation of the blood. This should be maintained until the crisis has passed. Factor VIII should be given intravenously. It can be administered as a transfusion of fresh whole blood if the patient has anæmia, otherwise fresh plasma, freshly frozen or freshly lyophilized plasma should be given. This has to be given frequently or by continuous drip, since the effect of factor VIII lasts only a few hours. It may take some time before the bleeding stops. Bleeding in the region of the throat should be treated vigorously since asphyxation may be the result of swelling of the soft tissue. Also acute joint bleeding should be treated intensively.

Factor VIII has been produced in purified form but it is still very difficult to obtain and these preparations are only available in small amounts.

Bleeding from accessible places can be treated by applying thrombin to the exposed bleeding points. The care of teeth is very important. Special precautions have to be taken in case dental surgery is required.

A paper by Boudreaux (who himself was a hæmophiliac) and Frampton in 1960 reported that the ingestion of peanuts or peanut flour gives symptomatic improvement of bleeding tendency. However, this observation has not yet been confirmed on scientific basis.

A complication of repeated administration of blood or plasma is the development of a circulating anticoagulant directed against factor VIII rendering the patient refractory to therapy. This makes prevention of bleeding the more important.

SUMMARY

A case of classical hæmophilia due to factor VIII (Antihæmophilic globulin) in an Indian boy is described. The diagnosis was revealed by the finding of an abnormal thromboplastin generation test and an abnormal prothrombin consumption, while other factors than factor VIII were found not to be deficient. No circulating anticogulants were detected. This is the first case of hæmophilia due to factor VIII deficiency described in Malaya.

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REFERENCES

Aggeler, P. M., White, S. G. Glendening, M. B., Page, E. W. Leake, T. B. and Bates, G. (1952). Plasma Thromboplastin component (P.T.C.) Deficiency: A New Disease Resembling Hæmophilia, Proc. Soc. Exper. Biol. & Med., **79,** 692.

Biggs, R., Douglas, A. S., Mac Farlane, R. G., Dacie, J. V., Pitney, W. R., Merskey, C. and O'Brien, J. R. (1952). Christmas Disease. A condition previously mistaken for Hæmophilia. Brit. M. J. **2,** 1378.

Biggs, R. and Douglas, A. S. (1953). The Thromboplastin Generation Test. J. Clin. Path. **6,** 23.

Boudreaux, H. B. and Frampton, V. L. (1960). A Peanut Factor for Hæmostasis in Hæmophilia. Nature, **185,** 469.

Budtz-Olsen, O. E. (1951). Clot Retraction. Oxford. Blackwell Scientific Publications.

Didisheim, P.: quoted in Wintrobe, MM (1961). Clinical Hematology. Lee & Febiger, Philadelphia.

Ehrenworth, L. (1963). Spontaneously occurring Anticoagulant against Antihæmophilic Globulin in a Previously normal subject. Am. J. Med. **34,** 272.

Hicks, N. D. and Pitney, W. R. (1957). A rapid screening test for disorders of thromboplastin generation. Brit. J. Hæm. **3,** 227.

Holborn, R. R. (1955). Estimation of fibrinogen in small samples of plasma. in The Coagulation of Blood. Tocantins, L. M. New York, Grune and Stratton.

Horowitz, H. I. and Fujimoto M. M. (1962). Acquired Hæmophilia due to a circulating Anticoagulant. Am. J. Med. **33,** 501.

Ivy, A. C., Shapiro, P. F. and Melnick, P. (1935). The bleeding tendency in Jaundice. Surg. Gynec. and Obst. **60,** 781.

Lee and White (1913), quoted in Wintrobe M. M. (1961). Clinical Hematology. Lee & Febiger, Philadelphia.

Quick, A. J. (1945). Determination of Prothrombin. Am. J. Clin. Path. **15,** 560.

Quick, A. J. (1949). The Prothrombin consumption test. Blood, **4**, 1281.

Quick, A. J. (1957). Hæmorrhagic Diseases. Philadelphia, Lee and Febiger.

Wintrobe, M. M. (1961). Clinical Hematology. Lee and Febiger, Philadelphia.

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SOME USES OF HYPNOSIS IN GYNECOLOGY

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The interaction of psyche and soma with the resultant complex of symptoms is probably more frequently encountered in the specialty of Gynecology than in any other area of medical practice. The female generative tract is extremely susceptible to the physiologic expression of emotions. A high percentage of gynecologic symptoms have a psychosomatic basis. Hypnotherapy is thus of great value in many instances.

Functional Dysmenorrhea: This is a "disease of theories." Many authorities place the etiology in the realm of disorders of the autonomic nervous system, much like the manifestations of bronchial asthma (1–4).

In a large number of women the belief exists that as soon as their periods begin, they are "unwell." In many cultures menstruating women are regarded as "unclean." In some primitive tribes menstruating women were confined to special houses and were temporarily taboo. To this day the women of certain Australian tribes are forbidden, on pain of death, to touch anything belonging to men (5). In western civilisation generations of school girls have learned to refer to their menses as "the curse." With such a background, therefore, it need not surprise us to find that numerous women suffer from dysmenorrhea.

The orthodox methods of treatment range from analgesics, sedatives, endocrines to surgical procedures such as D & C and pre-sacral sympathectomy, and as a last desperate resort marriage or childbirth is recommended. By its very name, Functional Dysmenorrhea raises a doubt of the wisdom of attempting treatment by these methods. Rather, hypnotherapy or psychotherapy is unquestionably the most rational approach. Brenman & Gill (6) and other investigators have reported good results with hypnosis. Kroger & Freed (7) have reported that more gratifying results can be obtained by hypnoanalysis.

In the writer's experience he finds that dymenorrhea responds remarkably well to hypnotherapy. Usually the patient is regressed, and the first occurrence of menstruation is hypnotically revivified in her mind, since most of these patients have received no preparatory instruction concerning the onset of the menarche. In the trance state such patients are given adequate information in direct relationship to the memories and impressions of their first menstrual experience, and posthypnotic instructions to continue to regard it as a normal matter-of-fact body function.

These two cases are illustrative.

Case 1. Age 15, menarche at 11, first few periods were normal but thereafter developed such severe pain that she had to be confined to bed at every period. Under hypnosis it was learned that the disturbing onset had occurred while she was at a show. When she discovered that she was bleeding, she went home in a state of panic, and since she had received no informative instructions, she dared not say anything about her state until late that night when she told her mother. In the state of regression she was properly and adequately informed and given proper posthypnotic instructions. Her next period was marked by little discomfort, and two more hypnotic sessions led to lasting therapeutic benefits.

Case 2. Age 37, a midwife, married with no children, suffered from severe menstrual pain, often requiring pethidine injections for its relief. She was a tense, hypersensitive type of woman. She responded well to hypnotherapy, accepted re-education correcting her hypersensitivity, and was given strong reassurance that her menstruation would be regular and painless. Her period has now been regular and painless.

Nausea and Vomiting of Early Pregnancy: The treatment of nausea and vomiting in early pregnancy is still a common problem in obstetrics. Psychogenic factors are chiefly responsible for the majority of cases. Hyperemesis gravidarum is unknown in some cultures. As psychogenic factors play a very important part in its etiology, nausea and vomiting and even hyperemesis gravidarum are astonishingly susceptible to hypnotherapy. Kroger, a

former Associate Professor of Obstetrics and Gynecology, Chicago Medical School, who had practised hypnosis for over 30 years, claimed that the incidence of cure by hypnosis is over 85% (8). Platonov treated 583 grave cases of hyperemesis gravidarum in Russia and cured over 84% with an average of seven hypnotic sessions (9). DeLee (10) believed that hyperemesis was most amenable to suggestion, and observed that most of the cures we accomplish are due to it.

These two cases are illustrative.

Case 1. Age 29, Gravida 4, para 3, had symptoms when her period was overdue two weeks. At first medical treatment was used to relieve her symptoms, but when the drugs finished, her symptoms returned. Hypnotherapy was then used. After three sessions she was completely well, and she delivered at term under hypnosis. She had had nausea and vomiting during all her previous pregnancies.

Case 2. Age 25, Gravida 3, para 2, also had nausea and vomiting in all her previous pregnancies. Symptoms began when her period was overdue for one week. Hypnoanesthesia had been used for her last delivery. Only one session was needed to cure her condition.

Abortion: It is well-known that psychic shock may be responsible for the termination of a pregnancy at almost any stage of its development. Cases of miscarriage and premature labour have frequently been reported as the result of emotional trauma caused by shelling and bombing during wars, the witnessing of unpleasant scenes or the hearing of unpleasant news. Authorities the world over are agreed that there is an idiopathic group deriving from as yet unknown causes who react to psychological stress and emotional crises, usually recurrent in character, to which may be attributed a large proportion of habitual abortions. Many authorities advocate simple psychotherapy for those patients (11). Reuter in 1937 quoted in F. J. Browne's Antenatal and Postnatal Care (12) reported good results from the use of cold baths in habitual abortion. The patient is advised to have a bath filled with cold water ready by her bedside. On first waking in the morning, she is to get into the bath, stay while she counts eight slowly and then, without drying herself, get into bed again! Thus it is to be expected that hypnotherapy can be of value in abortion. W. S. Kroger (13) has treated three apprehensive patients, who developed signs and symptoms of threatened abortion, with hypnosis. He had also used hypnosis to prevent premature labour in a case of abruptio placentae (14). Platonov (15) quotes Miloslavsky's recent systematic studies which showed that hypnosis could reduce uterine excitability, terminate bleeding and salvage a large number of fetuses.

Case 1. Age 24, married 4 years, and had had two spontaneous abortions despite medical care under proper conditions in a modern hospital. She sought the care of the writer for her third pregnancy, complaining of vaginal bleeding and slight abdominal pain. Her history disclosed her to be hyperreactive to stimuli, emotionally hypersensitive, and subject to panic states. She responded well to hypnotherapy, accepted re-education correcting her hypersensitivity, and was given strong reassurance, and after six therapeutic sessions succeeded in achieving a normal delivery.

Case 2. Age 34, married 2 years, had been infected with gonorrhea by her husband, a seaman. She responded to therapy but her husband reinfected her upon each return home from the sea. She was desirous of having a child and did conceive, but in the second month she developed a threatened abortion. Hypnotherapy was employed to relieve her tensions, fears and anxieties, and posthypnotic suggestions for a peaceful pregnancy were given. Two sessions served to allay the bleeding and to prevent the threatened abortion.

That hypnotherapy was the therapeutic agent in these cases cannot be asserted dogmatically. But it can be stated definitely that hypnosis can bring about mental and physical relaxation, peace of mind and comfort of body, and these are conducive to an uneventful pregnancy. The assumption that hypnosis may produce a relaxed condition or paralysis of the uterus is not without scientific evidence. Hypnosis has been shown to have profound effect on organs controlled by the autonomic nervous system, with depressive changes in temperature, pulse, blood pressure and B.M.R. (16) Hypnosis may thus effectively block adverse stimuli passing down the autonomic pathways from the higher sensorium and vegetative centres to the uterus.

Infertility: Psychosomatic infertility is receiving more attention from clinicians. The last decade has seen a number of good papers dealing with psychosomatic aspects of infertility (17–19). It is often heard that many women who have long been sterile conceive within a short time of adopting a child. Many factors are responsible for psychosomatic infertility (17). Kroger evaluated a series of infertile patients by psychological tests (20). He found that behind the outward desire to get pregnant was the deeply repressed wish not to get pregnant, on the basis of emotional immaturity associated with fear of motherhood or feelings of inadequacy. He believed that such conflicting emotions, mediated through autonomic, somatic, behavioral and endocrine mechanisms, often can affect the physiology of ovulation, implantation and, perhaps, even the viscosity of the cervical mucus, to produce the so-called hostile cervix. Other factors he mentioned are avoidance of coitus during ovulation; transitory or persistent tubal spasm; and conflicts in the male which may affect the fertilizing capacity of the sperm. Thus it is reasonable to expect that hypnotherapy is often of value in the treatment of infertility. Wollman (21) recently described successful treatment of several cases of infertility by hypnotherapy. Infertility may aften involve functional as well as organic factors. One should never accredit one cause because no other was found. Hypnosis provides an additional tool for the physician in the study of infertility.

The writer has one case to illustrate.

Case: Age 34, had been married 4 years, was unable to conceive though infertility investigations of both husband and wife proved to be normal. She consulted the writer because "she was so thin." A careful history, however, disclosed her intense desire to bear a child. Hypnotherapy was employed to relieve her tensions and anxieties. Under hypnosis she was reminded that infertility investigations showed both she and her husband were normal. She was also told about the fertile period, and was given strong reassurance that soon she would conceive. She was given a total of five sessions. The mental and physical relaxation secured through hypnosis benefited her and soon she conceived, and a Toad's test confirmed it.

REFERENCES

1. Kroger, W.S. & Freed, S.C.: Psychosomatic Gynecology. 1962. p. 235.
2. Theobald, G.W.: Some Gynecological Aspects of Referred Pain, J. Obst. & Gyn. Brit. Emp. 53:315, 1946.
3. Taylor, H.C.: Vascular Congestion and Hyperemia, Am. J. Obst. & Gyn. 57: 637–688, 1949.
4. Mazer, C., & Israel, S.L.: Diagnosis & Treatment of Menstrual Disorders & Sterility, New York, 1946.
5. Cianfrani, T.: A short history of Obst. & Gyn., Charles C. Thomas, 1960, p. 7.
6. Brenman, M: Quoted by Gill, M.M. Functional Disturbances of Menstruation, Menn. Clin. Bull. 7:10, Jan. 1943.
7. Kroger, W.S. & Freed, S.C.: Psychosomatic Treatment of Functional Dysmenorrhea by Hypnosis, Am. J. Obst. & Gyn. 46:817–822, Dec. 1943.
8. Kroger, W.S.: Clinical & Experimental Hypnosis, Lipp. p. 200.
9. Platonov, M.V.: *et al.*: Quoted by Volgyesi, F.A. in the recent neuropsychiatric and biomorphologic justifications of hypno-therapeutic empiricism, Brit. J. M. Hypnot. 2:6–25, 1950.
10. DeLee, J.B. & Greenhill, J.P.: Principles and Practice of Obst. & Gyn. 10th Ed. Saunders, 1951.
11. Tupper, Weil, Javert: B.M.J. Aug. 18, 1962, p. 465.
12. Browne, F.J.: Antenatal and Postnatal Care, 1955, Churchill, p. 200.
13. Kroger, W.S. & Freed, S.C.: Psychosomatic Gynecology, 1962, p. 148.
14. Kroger, W.S.: Clinical & Experimental Hypnosis: Lipp. 1962, p. 199.
15. Platonov, K.: The Word as a Physiological and Therapeutic Factor, Moscow Foreign Languages Publishing House, 1955.
16. Gorton, B.E.: Physiologic Aspects of Hypnosis in Schneck's Hypnosis in Modern Medicine, 2nd Ed., p. 246–280.
17. Kroger & Freed: Psychosomatic Aspects of Sterility, Am. J. Obst. & Gyn. 59:867–874, 1950.
18. Mandy, T.E. & Mandy, A.J.: The psychosomatic aspects of infertility, Internatt. J. Fertil. 3:287–295, 1958.
19. Marsh, E.M. & Vollmer, A.M.: Possible pyschogenic aspects of infertility, Fertil. & Steril. 2:70–79, 1951.
20. Kroger, W.S.: Evaluation of Personality Factors in the Treatment of Infertility. Fertil. & Steril. 3:542–551, 1952.
21. Wollman, Leo: The role of hypnosis in the treatment of infertility, Brit. J. Med. Hypnot. 2:38046, 1961.

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A SIMPLE METHOD OF SUPRAPUBIC CATHETERIZATION:

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Paracentesis vesicæ by a trocar and cannula is a method of relief of retention of urine well-known in the days of John Hunter. In 1806, John Bell wrote of this operation in his "Principles of Surgery," Volume II as follows:

> "This is a desperate operation, which is yet our sole resource, in our last stage of misery.
>
> This operation is less frequently resorted to in saving the lives of young and imprudent people, it is more commonly resorted to as a means of prolonging the life of the aged and infirm.
>
> Usually a man, far advanced in years, with the prostate gland diseased, who has had repeated attacks of obstruction. The bougie gives no relief, the catheter, even with any degree of force or rudeness, cannot be driven into the bladder."

He described six methods of paracentesis vesicæ: suprapubic, posterior perineal, anterior perineal, per perineal urethrostomy, per rectum and per urethra "forcing the catheter." The suprapubic method, he regarded as the most obvious and easiest — even the most ignorant tyro cannot miss the bladder here. But the experienced surgeon avoided it because as the bladder emptied, the trocar lay in a very oblique position; it never drained well and extravasation of urine always occurred, the tissues soon mortified and the patient died on the third or fourth day.

Though it is easy to be wise one-and-a-half centuries later yet it seems obvious that the operation was left too late to be of use; it was indeed a desperate resort in "the last stage of misery." The passage of a flexible catheter down the cannula to improve drainage was not thought of and it was apparently not known by Bell and his colleagues that a flexible catheter had been invented by Avicenna nearly a thousand years ago.

Present-day results of suprapublic bladder drainage are far better because of the invention of rubber tubing. De Pezzer's or Malecot's suprapubic catheters can be introduced into the bladder via the cannula, thus prolonging the period of drainage indefinitely. This well-established and widely used method was first suggested by Morson in modern times. Owing to the large size of the trocar and of the catheter, leakage of urine and resulting sepsis were inevitable, leading to their use being now condemned and to the advocacy of a formal suprapubic cystostomy to introduce the catheter with less likelihood of undesirable complications supervening.

When retropubic prostatectomy and other forms of prostatectomies with primary bladder closure were devised, and as these operations gained in popularity, it became important to have some form of temporary bladder drainage for the acutely retained patient from enlargement of the prostatic gland, and that this is to be effected without the introduction of sepsis so as not to preclude a subsequent prostatectomy with primary bladder closure. Riches (1943) devised a very clever and elegant instrument for the introduction of a small rubber catheter into the bladder by suprapubic puncture to effect drainage in the interim period between retention and prostatectomy. We have used his technique in the Professorial Surgical Unit, General Hospital, Singapore, for some two years and found some drawbacks: the rubber catheter being small in size and thin-walled perished rapidly in our climatic conditions; the catheter though small is still for larger than that necessary for adequate drainage, thus increasing the likelihood of pain or discomfort during its introduction and sepsis subsequently; the technique itself is not simple and inexperience had on several occasions led to the catheter slipping out of the bladder when the latter contracted in size on emptying.

Lane (1952) invented an instrument for the introduction of a small Malecot's catheter by suprapubic puncture and Swinney (1957) devised a method of introducing a Foley's catheter by the same route. The use of an

improved type of urethral cather made of poly-vinyl-chloride, (P.V.C.) tubing among paraplegic patients was reported by Ross *et al* in 1957. In the following year Gibbon reported its use in a wide variety of non-paraplegic cases with equal success. The Gibbon catheter is now widely used in all cases requiring bladder drainage by an indwelling urethral catheter. Owing to the inert nature of P.V.C. and other plastic tubings such as polythene, irritation and sepsis are minimal even after prolonged contact with living tissues.

The following method of suprapubic catheterization is so simple and easy to carry out even in the most unfavourable conditions that the author is surprised that it has not been more widely used. At first it was thought that the technique was novel though it is likely that it has been used by various doctors in various places but has yet been unreported. I am grateful to Gibbon (1962) who informed me that it had been previously reported and a further search of the literature showed that Davis had described essentially the same method in 1953.

Technique:

The procedure is carried out under aseptic conditions in the operating theatre. The patient is prepared as for a formal suprapubic cystostomy operation. After cleaning the skin, towels are applied leaving a small strip of midline subumbilical skin. A wheal is raised in the midline, 5 centimetres above the pubis symphysis with 1% lignocaine hydrochloride (Xylocaine). Through it the abdominal wall is infiltrated with the anæsthetic down to and including the bladder wall. The needle is then plunged into the bladder cavity and some urine is aspirated: first to confirm that the correct viscus had been entered and second to allow an assessment of the nature of the urine.

If the urine is clear, a small trocar and cannula, (about 6 Charrière, 2 millimetres in external diameter) of the type used for tapping hydrocœles is pushed into the bladder in one quick, firm movement. The trocar is withdrawn and a 3-foot length of P.V.C. or polythene tube of a suitable external diameter (a 6 Charrière cannula will take tubing of 1.7 millimetres external diameter) is immediately threaded into the bladder via the cannula. About 15 to 25 centimetres of tubing are pushed in, the length depending upon the obesity of the patient and the site of suprapubic puncture. (Figure 1). The cannula is then withdrawn and the "catheter" secured by either a stitch or a long piece of adhesive plaster or Scotch tape. (Figure 2). The other end is connected to a suitable container to establish "closed aseptic drainage."

The polythene tube has previously been prepared — one end being rounded off by lightly flaming it and moulding it with the fingers; several side holes are made near the tip by a heated metal probe of suitable size as described by Gibbon (1958). It is advisable to try the tubing for size by threading it through the cannula to be used before commencing the operation, otherwise, when the bladder empties the cannula will no longer be

Fig. 1

Fig. 2

within the lumen of the viscus and catheterization will be unsuccessful. It is also important to choose a tubing with a slightly loose fit, for after boiling it expands slightly and softens, making intubation difficult or impossible. It is also necessary to see that a fair length of tubing lies within the bladder to prevent it from slipping out accidentally as the bladder empties and contracts down.

If the urine is turbid, a larger trocar and cannula, e.g., 7 or 8 Charrière, allowing the insertion of large bore tubing is used in anticipation and prevention of blockage of the catheter by debris. A small nick of the skin with a knife will allow easier introduction of the larger trocar.

The whole procedure even in the hands of the most inexperienced takes no more than 5 minutes. The patient should feel no discomfort. Trauma, exposure and handling of tissues is reduced to the minimum and if strict asepsis has been observed, no sepsis can supervene in a case with uninfected urine.

This technique has been used in substitute for Riches' suprapubic catheterization for the emergency and temporary relief of acute retention while the patient waits for prostatectomy. It will also afford temporary relief of other causes of urinary retention.

No complications have been noted so far. Those pertaining to the administration of local anæsthesia and to suprapubic puncture will apply, but these are rare and not serious. Indeed, in view of the simplicity of the whole procedure no major complications are expected to occur. 20 cases of urinary retention have been managed by this way with satisfactory results. A microscopic hæmaturia to the extent of 20 — 50 red blood cells per high power field is usually present as with an indwelling Gibbon catheter but the urine remains clear on naked eye examination. In one case, macroscopic hæmaturia occurred but ceased spontaneously after a few hours; it is not clear as to the cause of this — it is possible that the hæmorrhage was due to the prostatic pathology as this is a known complication of indwelling per urethral drainage of the bladder for prostatomegaly. Cystoscopy with the catheter in situ has been performed in several cases and no leakage has been noted on filling up the bladder; the site of entry was uninflammed. At the definitive operation no extravasation has been noted and indeed the site of puncture of the bladder could not be identified. (The tube is removed before the skin incision is made).

A review of the various special trocars, cannulæ and catheters that have been devised has convinced the author that the instruments and catheters used should match the technique in simplicity, safety and reliability.

This technique owing to its very simplicity — the few and simple pieces of apparatus and equipment required — should have wide and ready application, especially so in the more undeveloped countries of the world.

REFERENCES

Avicenna. See "A History of Urology" (1953). American Urological Association, Vol. 2, p.355. Baltimore. Williams & Wilkins Co.

Bell, John. (1806). Principles of Surgery, Vol. II, quoted by Hopkins (1959). Med. J. Australia, i, 114.

Davis, J. A. L. (1953). Some Aspects of Catheterization in Retention of Urine. Brit. Med. J., i, 257.

Gibbon, N. (1958). A new type of catheter for urethral drainage of the bladder. Brit. Jour. Urol., 30, 1.

Gibbon, N. (1961). Some recent developments in bladder drainage. J. Roy. Coll. Surg. Edinburgh, 6, 215.

Gibbon, N. (1962). Personal communication.

Riches, W. (1943). The methods and results of treatment in cases of paralysis of the bladder following spinal injury. Brit. J. Surg., 31, 135.

Ross, J. C., Gibbon, N. and Damanski, M. (1957). Recent developments in the treatment of the paraplegic bladder. Lancet, ii, 520.

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THE GENUS CEPHALOSPORIUM AS A CAUSE OF MADURA FOOT IN MALAYA

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Madura foot is a distinct clinical entity presenting a uniform pathological and clinical picture although it may be caused by several species of fungi and by other micro-organisms, such as Actinomyces and Nocardia.

The fungi incriminated in the condition may be divided into two large groups, viz., I. **The Ascomycetes,** which are characterised by the formation of ascospores, and which have a sexual life-cycle. Members of this group are Allescheria, Aspergillus, Sterigmatocystis, and Penicillium; II. **The Fungi Imperfecti** which only show an asexual life-cycle, members of this group being Indiella, Glenospora, Monosporium, Cephalosporium and Phialophora.

Four different species of Cephalosporium are recognised as incitants of Madura Foot. They are **Cephalosporium recifei** (Leao & Lobo, 1934), **Cephalosporium granulomatis** (Weidman and Kligman, 1945), **Cephalosporium falciforme** (Carrion 1951), and **Cephalosporium infestans** (Gaind, Padhye, and Thirumalachar, 1962).

This report is the first recorded case demonstration of Madura Foot in Malaya caused by Cephalosporium falciforme.

Case History

The patient, a 47 year old male Indian labourer, was first admitted to an estate hospital in North Selangor 5 years ago with swelling of the right foot. He gave a history of injury with a thorn one year previously to the second toe of the same foot while clearing an oil palm estate in the district where he lived. The thorn was withdrawn from the site of penetration and the patient had forgotten about the incident till a year later when he noticed a small papule at the site of injury. This ruptured, discharged pus and soon healed spontaneously. Some weeks later another papule appeared, breaking down, discharging a serous fluid and then healing spontaneously. The condition progressed slowly over a period of 5 years characterised by remissions and relapses.

In November, 1963, he was admitted to the General Hospital, Kuala Lumpur, for further investigation. The right foot distal to the ankle was swollen and showed a number of healed scars, (Fig 1). No œdema was present and X-ray of the foot showed no bony involvement. Histopathological examination of a piece of tissue removed at biopsy revealed the presence of septate mycelium and fungus "grains" (Figs 2, 3 & 3a), surrounded by chronic inflammatory cells mainly lympho-

Fig. 1. Patient's foot 5 years after injury showing several healed scars and swelling.

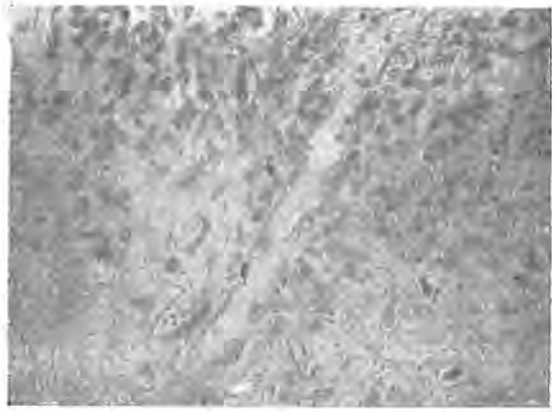

Fig. 2. Microscopic appearance of fungus in tissue showing septate mycelium.

Fig. 3. Gomori silver stain showing fungus 'grains.'

Fig. 3a. Fungus 'grain' magnified to show individual mycelium. (Gomori silver stain).

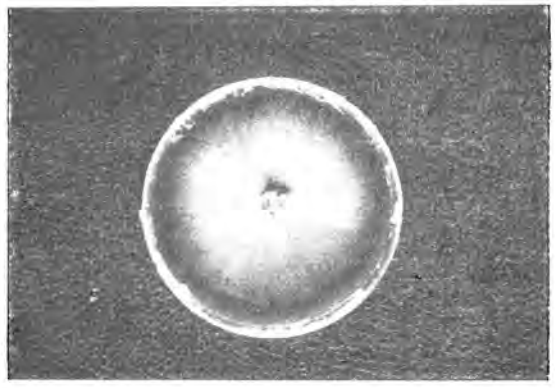

Fig. 4. A colony of Cephalosporium falciforme on Sabouraud dextrose agar—4 weeks old.

Fig. 5. A mount of the culture in hactophenol blue.

cytes, eosinophils, and macrophages. Gomori silver stain showed the mycelial wall and spores to be black. As the tissue was fixed in formalin, identification of the fungus was not possible. Examination of the pus showed the absence of the characteristic granules, and no fungus could be cultured. A repeat biopsy was carried out. The tissue was sliced into thin pieces and washed in several changes of sterile physiological saline. The granules were yellow in colour with a diameter of 2.5mm. These were surface sterilised and cultured onto Sabouraud dextrose agar containing 250 units of chloramphenicol per c.c. of the medium to inhibit the growth of bacteria.

Mycology. Cultures were maintained at room temperature. Growth on Sabouraud's dextrose agar was fairly rapid. About 10 granules were cultured and the same fungus was found in all instances. Young colonies were white later turning to brownish grey with deep radiating furrows extending from the centre to the periphery, (Fig. 4). The back of a mature colony of 4-5 weeks was brown to tan. A mount of the culture in lactophenol blue showed the presence of slender, unbranched conidiophores bearing a cluster of conidia at the tip (Fig. 5). Some of the conidia were sickle shaped (hence the name **C. falciforme).**

Treatment. This was not carried out as the patient discharged himself against medical advice. Amphotericin B has proved to be disappointing when given for 22 days in a case of arthritis of the knee due to Cephalosporium rosea-griseum as reported by Ward *et al* (1961) from the Mayo Clinic, Rochester.

Cases of Madura foot due to the genus Cephalosporium described in the literature are very few, and reports of treatment are still fewer in number. Gaind *et al* (1962) found treatment with large doses of penicillin, sulfa drugs, synermycin and griseofulvin without marked beneficial effect; potassium iodide given in increasing doses over a period of 4 weeks resulted in slight improvement; finally the whole area was excised and skin grafted.

ACKNOWLEDGEMENT

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REFERENCES

Conant, N.F., *et al*, 1959. Manual of Clinical Mycology. W. B. Saunders and Company.

Gaind, M.L., Padhye, A.A., and Thirumalachar, M.J., 1962. "Madura foot in India caused by Cephalosporium infestans," Sabouraudia Vol. I, Part IV.

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THE TREATMENT OF ASTHMA BY HYPNOTHERAPY

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There is extensive literature on the psychogenic aspects of asthma (1-3). Hippocrates, over two thousand years ago, shrewdly remarked that the asthmatic, if he were to master his condition, must "guard against his own anger" (4). Salter (5) himself a martyr to this condition, expressed the prevailing view that "its cause lies within the nervous system." A pioneer contribution from the psychoanalytic viewpoint was made by French and Alexander (6) of Chicago, who regarded asthma as a substitute for crying. Abundant clinical evidence points to a close association between feeling states and bronchial asthma. Many asthmatic patients are aware that an emotional disturbance, e.g., feelings of intense anxiety or unexpressed resentment, may provoke or aggravate an attack. The classic story of MacKenzie, 1886, of the asthmatic lady, reputed to be allergic to roses, who had an attack of asthma on smelling an artificial rose, supports the hypothesis that emotions or ideas, inter alia, may act as casual agents (7). Magonet (8) mentioned the case of a patient who was sensitive to ragweed pollen, but who, interestingly enough, did not have these attacks when his mother-in-law was out of town. A boy working in his father's flour mill was allergic to flour. He was told to move to a different town and to avoid working in a flour mill. He moved, but, as things worked out, the new job offered was in a flour mill, yet he was no longer allergic to flour. When he returned, however, and worked for a brief period in his father's mill the asthma returned (8). Thus, it is reasonable to regard bronchial asthma as a "neurosis" of the lungs." Characteristically, these patients suppress all intense emotions involving threats to their dependent relationships, deprivation and insecurity induced by sexual conflicts. As children, they are anxiety-ridden, lack confidence and are dependent to an extreme degree. Numerous authors (9-13) have treated asthma by hypnotherapy, and often the results were dramatic. Recent observations (14) showed that typical attacks of asthma could be hypnotically produced, and the attacks thus provoked could be immediately terminated by appropriate hypnotic suggestions.

Methodology: Usually the asthmatic patient has a long history of suffering and seeking of help from various sources, often to no avail. Such patients by the very nature of their illness are anxious, fearful, and highly motivated in their search for help. Consequently, they are usually good subjects and develop deep trances easily. My procedure is ordinarily two-fold. First, an effort is made to regress the patient to the time of his first attack and to re-educate him to the effect that the cause of that first attack is no longer operative and that he need no longer have fears and tensions about the constantly recurring need to breathe. Posthypnotic suggestions are given that he will enjoy sound physiological sleep, and that should he awaken, there still will be no asthma, and that in the morning he will feel at ease and comfortable and will so continue. In many cases, in one or two sessions, despite long previous sufferings, lasting therapeutic results can be secured. In more resistant subjects and where precipitating situations are not recovered a second methodology is employed. It is that of encouraging the patient, when he feels an attack developing, to sit down or to lie down, to close his eyes, to breathe deeply, then to hold that breath deliberately for a brief while; then slowly and comfortably to exhale with ease and comfort and without fear. This procedure is to be repeated at least five times and has the immeasurable effect of re-educating the patient's breathing attitudes, relieving his fears and tensions, and so it tends to abort the attack. Thus the patient is given the confidence of a ready and sufficient remedy in times of need.

Cases: The following cases are illustrative.

Case 1. Girl age 20, had asthma since menarche at 14, several attacks each month. She was regressed to the time of her first period, and it was learned that the onset was one of complete surprise, fear, anxiety and actual terror, and occurred when her mother

was not at home. Two hypnotic sessions were sufficient to give her permanent relief. Asthma could be hypnotically induced and terminated in her.

Case 2. Housewife age 42, had asthma for 16 years, husband a mechanic and had six children ages from 7 to 16 years. She was a tense, sensitive woman, worried about her children, about their poverty, etc. During the past few years her asthma was so bad that she had to pay daily visit to the Hospital past midnight and early hours for adrenalin injections. Finding no improvement in her condition she went in despair from G.P. to G.P. She showed dramatic relief after the first sessions. Four sessions cured her completely.

Case 3. Staff nurse age 30, had asthma for 2 years. Attack occurred a fortnight after birth of first child. Had to take ephedrine tablets every night. Marked improvement after the first session. During the second session, a week after the first, I asked her while she was in a trance whether she knew the cause of her asthma and she abreacted in tears that it was due to "anger." She wanted her own mother to nurse her baby when she returned to work, but her mother-in-law did not allow her. Two sessions gave her a lasting therapeutic cure.

Case 4. Girl age 6, had asthma for 1 year. Onset found to be associated with severe bronchitis. Whenever she had coughs and colds, and these were quite frequent, she had attacks. Four sessions stopped the asthma.

Case 5. Undergraduate science student age 24, had asthma since childhood. In recent years attacks occurred every night. Hospitalised many times and on several occasions "lost consciousness" on arrival in hospital. Under a trance it was learnt that his asthma dated back to the age of 4 or 5 when one of his grand parents died and he was forced to kiss the forehead of the dead man, which terrified him. He was a tense and irritable young man, and had a strong dislike for cats. There was marked relief after the first session. After three sessions at weekly intervals, he was comparatively free from attacks and they were so mild that half a tablet of Tedral would stop them. Normally he had to take 2–3 tablets Tedral, and often had to resort to cortisone tablets.

Case 6. Boy age 16, had asthma for 8 years. Onset of asthma started from prolonged bronchitis. After five sessions he was free from attacks. Asthma could be hypnotically induced and terminated in him.

Case 7. Age 26, housewife, had asthma for 20 years. For the past $1\frac{1}{2}$ years she had attacks almost every 1–2 days, and had to take tablets daily. After 6 sessions she was relatively free from attacks.

Case 8. Housewife age 40, had asthma since a very small child. Recently attacks occurred every day, and she had to take increasing doses of tablets. After 6 sessions she was relatively free from attacks.

Case 9. Schoolgirl age 9, had asthma for 4 years. Onset of asthma started after measles when she had severe prolonged bronchitis. Attacks occurred every day, and on many occasions she had to be warded in hospital under oxygen tent. After 5 sessions she was relatively free from attacks.

Case 10. Girl age 24, had asthma since the age of 9 when she had chronic cough. She was able to go into a very deep trance. Four sessions led to a lasting cure.

REFERENCES

1. Dunbar, H. F.: Emotions & Bodily Changes, N.Y. Columbia, 1938.
2. French, T. & Alexander, F.: Psychogenic Factors in Bronchial Asthma, Psychom. Med. Monog. Ser. 2:34, 1941.
3. Vaughan, W. T.: Practice of Allergy, St. Louis Mosby, 1939.
4. Sclare, A. B.: Psychological Aspects of Bronchial Asthma in "Bronchial Asthma A Symposium." Chest & Heart Ass. Report of a meeting held on 25/2/1959 at the Royal Society of Medicine, London.
5. Salter: Asthma, its pathology, and treatment, 1860.
6. French, T. & Alexander, F: Psychogenic Factors in Bronchial Asthma, Psychom. Med. Monog. Ser. 2:34, 1941.
7. Mackenzie: Amer. J. Med. Sci., 91, 45, 1886.
8. Magonet: Hypnosis in Asthma, Heinemann, 1960.
9. Ambrose, G. & Newbold, G.: Handbook of Medical Hypnosis, Bailliere, 1959.

10. Kroger, W. S.: Clinical & Experimental Hypnosis, Lipp., 1963.

11. Van Pelt: An Answer to Asthma, J. Am Inst. Hyp. Vol. 3, No. 2, April, 1962.

12. Diamond, H. H.: Hypnosis in Children: The complete cure of 40 cases of Asthma, Am. J. Clin. Hyp. No. 3, Jan. 1959, p.124.

13. Maher-Loughnan, G. P., Macdonald, N., Mason, A. A., & Fry, L.: Controlled trial of hypnosis in the symptomatic treatment of asthma. B.M.J. 1962, 5301, 371-6.

14. Platonov: The Word as a Physiological and Therapeutic Factor, Moscow, Foreign Language Publishing House, 1955.

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PRESIDENT'S VALEDICTORY ADDRESS 1964

By Dr. ABDULLAH bin AHMAD, L.M.S. (S'pore), P.J.K.

THE TWIN PROBLEMS OF RURAL POVERTY AND ILLHEALTH

Poverty and illhealth go hand in hand wherever they exist in rural areas. The solution to illhealth in rural areas does not lie in the medicine chest of the doctor. The key to many such medical problems can be found in the rectification of the basic social or economic ills that afflict the community. How can the vicious cycle of Illhealth — Low Productivity — Poverty — Malnutrition — Illhealth be broken in the rural areas? The rural environment contains the greatest hazards to health. Would changing the environment help to lay the foundation for the takeoff into rural prosperity and good health? Would the people, who are traditionally bound to a rural way of life accept the sociocultural changes of a semiurban way of modern living? I shall try to deal with some of these problems.

Poverty is a problem not limited to the backward developing countries of the world. A highly industrialised country like the United States of America, which is the richest country in the world as 35,000,000 poor people out of a population of about 180,000,000. It is estimated that this 20% of the population who are poor enjoy only 5% of the National Income of the United States, while the affluent 80% enjoy 95% of the National Income. When the individual income falls below 10% of the per capita National Income of $M7,000 per annum, one is considered to be poor in the United States. A per capita income below $M80/- a month cannot buy you the necessities of life in the United States. It is interesting to note that in Malaya more than 60% of the population are subsisting on a per capita income of about $10/- a month or a family income of $30 - $40 a month.

Poverty in the United States is different from poverty in a developing country. When a country's production is far in excess of the needs of its people, measures directed at a more equal and more efficient distribution of wealth will wipe out poverty. In a developing country like Malaya where a large majority of the 7,000,000 people are peasants, who are eking out a subsistence on one to three acres of land, any move to divide the National Income, even equally amongst its people will find no solution to poverty nor will it generate prosperity. If the standard of living of the people is to be increased, the production of wealth in the form of goods and services should be increased many times.

The per capita National Income of Malaya is $900/- per annum and is the second highest in Asia. When compared with the per capita National Income of $M7,000 in a highly industrialised country like the U.S.A., or the per capita national income of $M4,000/- in Australia, it will be evident that Malaya cannot afford to immediately invest large amounts of money to fight poverty and illhealth. It is estimated that 60% of Malayans who live in rural areas enjoy only 10% - 15% of the Malaya's National Income of $6,000 million per annum. The seriousness of rural poverty will be appreciated if it is known that half the economically active persons in Malaya live in the rural areas. About 60% of our National Income is enjoyed by the urban Malayan Chinese population who are actively engaged in the commercial and industrial enterprises of the Nation, and who also pay most of the taxes. A large part of the balance of 30% probably goes as profits to the foreign commercial and industrial organisations which operate in Malaya; the balance is income accruing to Governmental organisations from property and entrepreneurship.

Poverty is not limited to the Malays, it also exists side by side amongst the Chinese and Indian peasants and rural workers in Malaya. The rural people are primarily occupied in small scale farming in uneconomical lots of land. The riverine and coastal people are employed in small scale fishing. The people in the interior get their income from hunting and collecting forest produce. People like the rural Malays lead a simple life. They

produce just sufficient for their needs. According to our economist Prof. Ungku Aziz some of these people live completely outside the money economy. A family income of $25 to $30 a month is average amongst the Malay peasants. This is less than $10 per capita in the family, and below 10% of the per capita National Income of $70/- per month. The Health and Nutrition of the family cannot be maintained on such a meagre precarious income. Poverty, Ignorance, Insufficient Land, and Illhealth are the causes of the family food being deficient in calories, vitamins, minerals, and the sorely needed proteins to nourish the children. Malnutrition is endemic in rural areas and it reduces the output of work.

The rural family who are malnourished because of poverty, soon become prey to the many diseases caused by an unfavourable environment. Malaria is the biggest scourge, and recurrent attacks of this fever soon make them unfit for hard work. Tuberculosis is a common problem. Ankylostomiasis and other worm infestation affect a vast majority of the rural people. Gastro-enteritis, dysentry and typhoid take a heavy toll of infant life. Cholera is beginning to rear its head in the rural areas in North Malaya. Filariasis and yaws are common in certain areas. Most of the people suffer from anæmia which saps their vigour. About 20,000 of the 30,000 deaths that occur annually in the rural areas are certified by the village policeman as due to fever or "Pyrexia of Unknown Origin." The exact cause of death of a large majority of the rural people is still a mystery to the Registrar General of Births and Deaths. The death rate in the rural area is more than 13.0 while the death rate for urban areas is about 8.0 or 9.0 Very few of these rural people seek admission to hospitals when they fall ill. However, they are now, not averse, to calling at the Government Rural Dispensary or at the Government Travelling Dispensary for outpatient treatment. About $\frac{1}{4}$ of the 4,000,000 rural people visit these travelling dispensaries. More than 60% of the people treated are Malays, and to most of them this is the only contact they have with a Modern Health Service. The treatment of sickness that is caused by the health hazards in the rural environment is not the answer to their suffering. These rural people need a healthy home environment in order to remain healthy.

It is obvious one cannot enjoy glowing health in an environment of mud, swamps, leeches, mosquitoes, flies, worms and germs, when even the water you drink and bathe in, smells and tastes of decay and dirt. To a doctor who works in this area there does not appear to be much of a dividing line between life, disease, and death.

Most of the wooden houses in which these people live may look shabby from the outside, but they do take pains to keep the inside clean. Since the water they use is already polluted, washing the house creates new dangers for the growing infant who is beginning his life within the protection of the house. Since the houses are uniformly spread in one or two acre lots over vast expanses of land there is no hope in the forseeable future to provide each house with filtered and treated running water.

Most of the houses have no latrines either within the house or outside it. The bush in the neighbour's land or in one's own land is the toilet for the older members of the family, and the land outside or under the house is used by the children. The pollution of the wells, ponds and rivers which are the only sources of the family water supply, is invariable under such circumstances. Gastro-enteritis, Dysentry, Worm infestation take their toll of infant life and infant health. The infant mortality in the rural areas is about 80 – 100 deaths per 1,000 live births, while the rate is half of this in the urban areas, where a more sanitary environment exists.

I may have painted a gloomy picture of the state of affairs in the rural areas. Those of us who live a lifetime in urban areas tend to overlook the want and misery that exists so close to us in the villages and kampongs. Our Government like all enlightened Governments is making a many pronged attack to eradicate rural illhealth and rural poverty. Land reforms to increase the size of the family holding to about 10 acres each are being introduced so that the family income will be raised from $30/- per month to $300/- per month. Agriculture, the mainstay of any rural economy, is being modernised. Labour saving

agricultural machines are being introduced into the farms. High yielding seed, and fertilisers are being distributed. Large schemes to improve the irrigation facilities are being undertaken. The diversification of crops and double cropping are some of the measures being introduced to increase the income of the farmer. Co-operative capital, and co-operative marketing facilities are being introduced to break the vicious holds of the money lender and middlemen.

General education and technical educational facilities are being provided in most rural areas to lay the foundations for creating the human resources required for rural progress. Roads, bridges and additional transport facilities are being extended into the rural areas at a rapid rate. This will facilitate the rapid exposure of the rural people to the modern urban ways of living, commerce and production.

The Health Services are being extended into the rural areas at a rapid rate. The midwife is the spearhead of the service. Every 2,000 people get a resident midwife and a midwives clinic costing $10,000/-. Every 10,000 people receive a Sub-health Centre, costing $100,000/- where there is a resident Nurse, two Assistant Nurses, Public Health Overseer and other staff. Five of such centres serving 50,000 people come under the control of a Resident Doctor, Resident Dental Surgeon and Resident Public Health Inspector. The whole Health Unit serving 50,000 rural people costs $1.6 million to build and large sums of money to maintain. The main function is to treat sickness amongst the rural people, make child bearing safe, provide child care, improve personal hygiene and personal care, and improve the sanitation in the environment.

Despite the concerted efforts of many Departments to provide the extension services to alter the environment and make it safe for the rural inhabitants, the planners appear to have overlooked the enormous cost in manpower, money and materials required to make safe more than 4,000 square miles of land occupied by the rural people. Most of us who are engaged in the construction of permanent anti-malarial works will appreciate the heavy expenditure required to make a few acres of land free from a single disease like malaria. The main problem to be faced when trying to improve rural sanitation is the fact that about 4,000,000 people are spread out in one, two, three or four acre lots, over 4,000 square miles of land in about 1,000,000 wooden houses of the primitive type. The vast majority of these houses have no purified water supply, no latrines, no drainage and no proper refuse disposal. What is the solution? How shall we provide a clean home and healthy environment to these 4,000,000 people so that illhealth can be eradicated?

No community can enjoy all the modern services of water, light, power, drainage, sanitation, shopping and other social services if they are spread out all over the country. The cost of taking such services to them is prohibitive. To enjoy the high standards of living of a modern civilisation it is necessary that we live in a close compact community. It is therefore necessary to introduce modern town planning in our villages and kampongs. In every village suitable high land which is dry and easily drained should be developed as a modern village centre for housing, in the same way that we developed Petaling Jaya as a modern suburb of the National Capital. Rural Re-housing and Rural Re-construction is the fundamental solution to Rural Illhealth.

The Government Commission which investigated the 1963 outbreak of cholera in South Malaya made many recommendations for the prevention of epidemics. One of these recommendations was for village development according to modern town planning. The Commission recommended:—

> "that the Government should introduce legislation to control the development of **villages,** towns and cities, in Malaysia according to modern town planning concepts, which should be binding at all levels of administration, and from which no deviation should be allowed if such action would lead to infringement of health rules, or cause a hazard to the Health of the community."

(Malaysia being a young country, early action along these lines will pay handsome dividends in health for the future, without causing much hard-

ship to the present generation. The haphazard development of Malacca without the aid of modern town planning should be a lesson for the rest of Malaysia, especially for local authorities).

The words used by the Commission are more or less the very same words used by the Malayan Medical Association when it made this recommendation to the Commission.

The Rural Re-housing which is necessary under this scheme to eradicate rural poverty and rural illhealth is far too vast to be shouldered by the rural people themselves. Government assistance is necessary for the planning and development of sites, for laying down all the common services, and for subsidising the purchase of houses. Government has not given much attention to rural re-housing. The United Nations Secretariat in its report on the World Housing Situation recommends that countries in Asia need to build annually no less than 10 houses per 1,000 of the population. On this estimate the 4,000,000 rural people in Malaya would require at least 40,000 houses every year. At $10,000 per prefabricated concrete low cost house, a sum of about 400 million dollars would be necessary. Can this money be found to provide for Rural Re-construction at an annual investment of about 7% of our National Income? When considering this we should remember that more than half the economically active people of Malaya live in rural areas, which contain about 60% of the population. Despite this only 10 – 15% of the National Income goes into their pockets.

If rural labour is used in the rural re-housing programme, and if most of the materials used are manufactured in the same region, and if skilled labour is trained in the rural areas, Government spending of the magnitude contemplated would boost the rural income and help to eradicate rural poverty. It is interesting to note that a measure contemplated to improve the environmental health should also help to alleviate poverty and thereby help to break the vicious cycle of ill-health — low productivity — poverty — malnutrition — illhealth. I commend this proposal for your contemplation. Should some of the present makeshift rural development plans, which may turn out to be ineffective and uneconomical in the long run, give way to a forward looking plan to modernise our kampongs and villages in gradual stages? Is this the key to eradicate poverty and illhealth and lay the foundations for Rural Progress? If town planning is to be introduced into the rural village have we in the past been siting millions of dollars worth of public and private buildings in the right area? Should we and the District Officers give this more thought in future? I leave these questions for your consideration.

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TUBERCULOSIS IN MALAYA

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SOME FACTS AND FIGURES

Tuberculosis is a major public health problem in Malaya. It kills and causes suffering to more persons than any other single disease. It is by far the biggest killer in the country. More than 5,000 persons die from tuberculosis every year, about 7% of deaths from all causes. Against this there is a yearly crop of 25,000 new cases, which means that for every death from tuberculosis five new cases appear thus swelling quite relentlessly the pool of tuberculosis sufferers in the country. This in turn increases with a vengeance the risk to healthy individuals of contracting the disease.

More than one quarter of our hospital beds are occupied by tuberculous patients, most of them in an advanced stage of the disease. More than one quarter of the time of our doctors and nurses in our hospitals is taken up looking after and treating tuberculosis sufferers. Almost one-tenth of the total Health Budget of the country is expended on tuberculosis.

So large is the pool of infectious cases in the country that one child out of every four is infected with tuberculosis before the age of five years. At the age of 10 years about half the children are already infected. By 15 years of age three out of four children are found to be infected. Fortunately not all these infected children develop disease but they certainly constitute the vast reservoir from which new cases appear in our community. The larger the number of infected persons in the country the greater is the potential for the appearance of new cases of tuberculosis.

Although tuberculosis is usually a disease of townsfolk or unbanised communities, our rural areas are affected almost as seriously as our towns. The prevalence of this disease amongst the aboriginal population in Malaya has been found in recent surveys to be as high as in our towns. The kampong folk, particularly those in Kedah and the east coast states have an incidence almost as high as is found in the slum areas of our big cities and towns.

No community in Malaya enjoys freedom from tuberculous infection and disease; no social stratum is exempt from the ravages of this disease. Malays, Chinese, Indians contract this disease with equal facility and spread it as readily as they contract it. Rich and poor alike are prone to it. Nobody can be safe until all are safe.

THE SITUATION BEFORE MERDEKA

Before Merdeka no organized attempt was made to combat the spread of tuberculosis in Malaya. Only those cases who voluntarily sought treatment in our hospitals were given treatment and even for these cases the facilities for accommodation and treatment in our hospitals were grossly inadequate. All hospitals were embarrassed by the number of cases who sought treatment. They maintained long waiting lists of cases seeking admission. The available beds for tuberculosis were always full. Those patients who managed to gain admission were found almost invariably to be in an advanced stage of the disease requiring long-term hospitalisation. This accentuated further the acute bed shortage. Waiting lists mounted inexorably. Advanced chronic cases for whom little could be done occupied the available beds for several years and early treatable cases were turned away for lack of accommodation.

Little wonder no attempt was made to institute proper public health measures to control the spread of this disease in accordance with the accepted principles governing the control of any infectious disease. One of the cardinal principles is to find the infectious cases in the community and render them non-infectious with treatment and/or quarantine. The other important measure is to protect the susceptible members of the community with reliable vaccination. One can well imagine the dilemma facing the health authorities. When the existing facilities in our hospitals were already bursting at the seams to cope with those cases who voluntarily sought treatment what would happen when a systematic case-finding campaign was launched? Acute and utter embarrassment!

The guardians of our public health decided to tackle the problem by ignoring it completely. Not quite completely, really, because they did make an attempt to introduce at least the

other less difficult measure: BCG vaccination. Even in this the interest shown was so meagre and the general apathy so formidable that the BCG vaccination campaign was poorly organized and enjoyed only lukewarm support. Also in order to appease the public clamour, experts were invited to study the tuberculosis problem in Malaya and recommend suitable measures for its effective control. One after the other these experts came, saw, recommended and went their way. All their reports and recommendations were neatly filed away, never disclosed to the clamouring public. Dr. Andrew Moreland came in 1947, Sir Frederick Heaf in 1952. Their reports were never made public. In the meantime the spread of tuberculosis in the country continued with unabating fury. During the 10 years, 1947 to 1957 the risk to healthy individuals of contracting tuberculosis quadrupled. Whereas in 1947 for every death from tuberculosis two new cases developed, by 1957 this ratio had quadrupled to eight new cases per death.

AFTER MERDEKA

In 1957 came Merdeka and with Merdeka came also a sense of greater responsibility on the part of our Government for the well-being of our people. The elimination of tuberculosis was included in the Alliance Party manifesto, and one of the very first steps taken by the Ministry of Health after the general elections was to create the post at the Federal level of a Senior Tuberculosis Specialist who was charged with the responsibility of studying the tuberculosis problem in the country and drawing up a plan for its control. Assistance was sought from the World Health Organization for the services of a tuberculosis consultant. Sir Harry Wunderly, the chief W.H.O. Consultant on Tuberculosis, formerly Commonwealth Director of Tuberculosis in Australia, a recognized world authority on tuberculosis control who had been mainly responsible for the effective control of tuberculosis in Australia during his tenure of office was assigned to Malaya in 1959. Sir Harry remained in Malaya for two months during which he made a thorough study with the Senior Tuberculosis Specialist of the existing resources for the prevention and treatment of tuberculosis.

They travelled extensively up and down the peninsula visiting all the hospitals and medical institutions in the country, making an exhaustive study of the situation prevailing in Malaya. At the end of his tour Sir Harry submitted a report on his findings and made recommendations. These recommendations were accepted by the Government, and based on them and in close consultation with him a comprehensive long-term plan for the Control of Tuberculosis was drawn up by the Ministry of Health in 1960.

A separate Division of Tuberculosis was set up in the Ministry of Health under the charge of the Senior Tuberculosis Specialist to implement this plan. Funds were allocated under the National Second Five-Year Development Plan, 1961-1965. In 1961 the National Tuberculosis Control Campaign was officially launched to eliminate tuberculosis as a public health problem in Malaya. The control campaign is now in operation and is fast gathering momentum. By 1965 it will be fully developed with all its components functioning in top gear. Ten years from then, that is by 1975, it is hoped to bring tuberculosis under effective control so that it will no longer be a public health menace.

THE NATIONAL TUBERCULOSIS CONTROL CAMPAIGN IN MALAYA

Principles

The National Tuberculosis Control Campaign which was launched in 1961 under the Second Five-Year Development Plan and is scheduled to be fully developed and operational by 1965 is based essentially on two important principles which, incidentally, govern the control of any infectious disease, namely: -

1. find the infectious cases in the community and render them non-infectious with treatment;
2. protect with a reliable vaccination all those who are susceptible to infection

Effective Weapons

The degree of success which may be expected in the control of an infectious disease depends largely on the effectiveness of the weapons which are available and the possibility of their practical application on a mass scale. In so far as tuberculosis is concerned we have not only extremely effective weapons but also ample proof from the experience of technologically advanced countries of their efficacy and practical applicability on a mass scale. We can find the infectious cases in the community very rapidly by carrying out simple bacteriological tests on those found to have

abnormal chest X-ray findings suggestive of tuberculosis. The mass miniature X-ray machine is capable of examining a hundred persons an hour and serves as a very effective sieve to pick up suspects for bacteriological investigation. We have specific drugs to treat, render non-infectious and cure the cases of tuberculosis discovered. And in BCG we have a very effective and reliable vaccine to protect the susceptible members of our community.

Important Discovery

One other important discovery is of special interest to us in Malaya: hospitalisation is not necessary to treat every case of tuberculosis. Treatment is in most cases as effective when given on an out-patient basis, where the patient attends the hospital once a week or even once a month to collect the drugs for consumption at home and takes them regularly for the necessary period of time. What is even more important: the vast majority of cases discovered need not stop work while they are having treatment. If and when leave from work is advised it need rarely exceed three months, six weeks being ample in most cases.

Public Health Approach

The accent in our control campaign is on the protection of healthy people. It consists of an organized national effort to reduce the risk to healthy individuals of contracting tuberculosis. The approach is a public health one directed to the community rather than to the individual patient. The individual patient is important only because rendering him non-infectious prevents him from infecting the community. Protection of the health of the community is the primary aim.

The Control Campaign in Malaya

The Control Campaign in Malaya consists of a three-pronged attack on tuberculosis in this order of priority:

1. **The Training Programme** to train the different categories of technical personnel required for the country-wide control campaign.
2. **B.C.G. Vaccination Drive** to protect the susceptible members of our population against tuberculous infection.
3. **Case-finding Drive** using mass miniature chest X-ray units followed by bacteriological examination of sputum of suspects to discover the infectious cases in the community so that they may be rendered non-infectious with treatment.

Organization to implement the Control Campaign

As stated previously a separate Division of Tuberculosis was set up in the Ministry of Health in 1961. The Senior Tuberculosis Specialist was appointed to head this Division and charged with the responsibility of planning, organizing and directing the National Tuberculosis Control Campaign.

A National Tuberculosis Centre was set up in Kuala Lumpur to serve as the headquarters of the national tuberculosis service. This Centre is responsible for the co-ordination and technical direction of the National Control Campaign. It also undertakes the training of all categories of technical personnel required for the country-wide control campaign. Initially this training was conducted with the assistance of technical experts provided by Australia under Colombo Plan Aid and by the World Health Organization. Since the beginning of this year (1964) the training is conducted almost entirely by our own local officers.

Although the National Tuberculosis Centre started functioning in 1961 it is still in the process of development and will be fully developed and fully operational at the end of 1964.

Also in the stage of development are State Tuberculosis Centres which were set up in 1961 in Alor Star, Ipoh, Johore Bahru, Kota Bharu, Kuantan, Kuala Trengganu, Malacca, Penang and Seremban. These Centres, most of which are already developed, serve as the headquarters of the state tuberculosis service and are responsible for the implementation of the control campaign in their respective states. Each State Centre is modelled on the National Centre in Kuala Lumpur having the same range of activities and provided with similar facilities but on a smaller scale relative to the individual needs of the State Centre.

In order to implement the campaign at the district and rural level 20 District Tuberculosis Clinics are being developed in 20 of the 42 district hospitals in the country. In making the choice of the 20 district hospitals where fully equipped and adequately staffed District Tuberculosis Clinics are being set up, many factors have been taken into consideration,

the more important ones being population density of the area served, accessibility, geographical location and the load of tuberculosis work borne by the hospitals in past years.

The following 20 District Hospitals were chosen: Kangar (Perlis), Sungei Patani (Kedah), Bukit Mertajam (Penang), Taiping, Kuala Kangsar, Tapah, Telok Anson, Tanjong Malim (Perak), Kuala Kubu Bharu, Klang, Kajang (Selangor), Kuala Pilah (Negri Sembilan), Segamat, Muar, Batu Pahat, Kluang (Johore), Mentakab, Kuala Lipis (Pahang), Kuala Dungun (Trengganu) and Kuala Krai (Kelantan).

Most of these district tuberculosis clinics have been almost completely developed; the others will be fully developed and operational by 1965.

In addition to these district tuberculosis clinics, all the other district hospitals in the country together with all the Maternity and Child Health Clinics and Rural Health Units are participating actively in the implementation of the control campaign.

Cost of the Control Campaign

$6.7 million were allocated to the National Tuberculosis Control Project as capital expenditure under the National Second Five-Year Development Plan to provide new buildings and equipment. $3.87 million have already been spent and the remaining $2.83 million will be fully utilized by the end of 1965 when the National Control Campaign will be fully developed.

In so far as annually recurrent expenditure for the Control Project is concerned $223,553 were allocated in 1961, $1,379,466 in 1962, $1,505,163 in 1963 and $1,769,928 have been allocated for 1964.

This special allocation for the Project is intended to augment the expenditure normally borne by the states for tuberculosis prevention and treatment out of their total annual allocation. A study of the expenditure incurred by the nation prior to the commencement of the National Tuberculosis Control Project reveals that tuberculosis alone consumed almost one-tenth of the total annual Health Budget of nearly $100 million.

The special allocation for the Tuberculosis Control Project does not therefore represent the total annually recurrent expenditure incurred by the country for the prevention and treatment of tuberculosis. It is intended merely to supplement it, to meet particularly the additional financial commitments occasioned by the special requirements of the control project.

Duration and Aim of the Campaign

The National Tuberculosis Control Campaign launched in 1961 will be fully developed by 1965. By then all the components of the campaign will be operating with optimum speed and efficiency. Only by 1965, therefore, will the campaign become fully operational. Within five years of that date, that is by 1970, a definite decline may be expected in the incidence of tuberculosis in the country. By 1975, that is 10 years after the campaign got into full swing and 5 years after a drop is noticed in the prevalence of the disease the target of the control campaign will be achieved — elimination of tuberculosis as a public health problem. Cases of tuberculosis will still continue to occur, but they will be few and far between, occasional sporadic cases easily controlled, presenting little or no danger to the health of the community.

When that stage is reached the ultimate aim of the current campaign will have been achieved but that does not mean that we could then afford to put away our weapons and call off the fight. Constant vigilance will be necessary to keep the enemy safely contained and effectively controlled. The cost to the country of this careful and constant vigilance will be only a tiny fraction of the vast sums of money that have been and are being spent to prosecute the control campaign. Handsome dividends are in store for us for the wise investment we are making to ensure freedom from the tuberculosis menace to ourselves, to our children and to our children's children.

THE TRAINING PROGRAMME

The Training Programme constitutes the first prong of the National Tuberculosis Control Campaign. It commands the highest priority in the control campaign. The reason for this is pretty obvious. No technical project, however sound it may be in principle, can possibly succeed unless technically skilled persons are available in adequate numbers to operate the various technical components comprising the project. Malaya, like other developing countries, does not have enough skilled technicians. There is a grave shortage of technically trained medical personnel at all

levels from doctors and nurses to technicians and other para-medical staff.

Role of the National Tuberculosis Centre

Since June, 1961, a vigorous training programme has been under way for all categories of technical personnel required for the nation-wide tuberculosis control campaign: doctors, nurses, laboratory technicians, mass X-ray operators, X-ray technologists, home-visitors, record clerks, etc. The training is conducted in the National Tuberculosis Centre in Kuala Lumpur. The training courses which are conducted by the senior staff at the Centre have been organised and evolved with the assistance of technical experts. Since the beginning of this year, (1964) the training courses are conducted almost entirely by our own local officers.

The National Centre in Kuala Lumpur is, apart from its many other functions, also actively engaged in providing a tuberculosis service to the State of Selangor and functions as a model State Centre. In so doing it affords ample and ideal demonstration facilities for the training of all categories of technical personnel required for the country-wide control campaign. Here the latest methods of tuberculosis control are tried, appraised and appropriately modified to suit local conditions before they are introduced for country-wide application.

The training programme at the Centre caters for two categories of personnel. One: in-service training for technical personnel who are already in the service and able to engage in tuberculosis control duties in their particular spheres of activity in addition to their normal duties. In this category are doctors, nurses, hospital assistants, health visitors, radiographers, record clerks, statistical clerks, etc. The other category comprises technical personnel specially recruited for the campaign who work exclusively in tuberculosis institutions like assistant nurses, mass X-ray operators, laboratory assistants, etc.

The development of the control campaign throughout the country is carefully geared to the training programme. As more and more trained technical personnel become available the scope and extent of the control campaign is stepped up in all the different parts of the country.

Nursing Staff

The most important training course conducted for nursing personnel is the *Training Course in Tuberculosis Control Methods for Supervisory Nursing Staff*. It is the most important course for two reasons. Firstly, it forms the basis for all training courses arranged for all categories of nursing personnel — orientation courses for senior nursing officers like Matrons and Sister Tutors, practical training courses for auxiliary nursing personnel like Assistant Nurses, training in Tuberculosis Nursing and Control Methods as an integral part of the prescribed course for Student Nurses in their third year at the School of Nursing, training in the public health aspects of Tuberculosis Control for Public Health Nurses attending the Health Visitors Training Course. Secondly, supervisory nursing staff (Nursing Sisters, Health Sisters, Staff Nurses and Health Nurses) constitute the backbone of the BCG vaccination drive, examination of Contacts, tuberculosis nursing, chest clinic practice, home-visiting and follow-up and after-care of patients — the vital components of the tuberculosis control campaign. On their return to their home stations after completing the training course at the Centre, they not only participate actively in the control campaign but also undertake the training of their junior colleagues in new techniques and procedures they have learnt, thus broadening both the concept and scope of the National Tuberculosis Control Campaign.

This course extends over a period of four weeks. The first week is taken up with instruction in the classroom and consists of 11 Lectures, 10 Lecture-Demonstrations and 4 Demonstrations. During the subsequent three weeks the trainees are posted for practical training to the different sections of the tuberculosis control department of the National Tuberculosis Centre — Contact Clinic, School BCG Vaccination Service, Maternity Hospital Vaccination Service for Newborns, Medico-social Service including home-visiting, health education, policing of defaulters and after-care of discharged patients. These postings for practical instruction are made on a rotation basis so that not more than three are attached to any one section at a time. The trainees are able thus to participate actively in all the duties undertaken by the section to which they are posted under the close personal supervision of the officer-in-charge of the Section. During this three-week period of practical training emphasis is placed on active participation as a responsible member of the staff.

Student Nurses in their third year at the School of Nursing attend the full course of four weeks and in addition spend two weeks

doing tuberculosis nursing in the wards and out-patients department of the Centre. At the end of their 6 weeks' training they are given a written examination.

Assistant Nurses undergo an 8 weeks' training course. Their curriculum is essentially the same as the main course except that it is considerably simplified. The theoretical component is substantially curtailed and the scope of practical training correspondingly increased. On their return to their home stations after completing the course, they are posted to tuberculosis institutions to engage exclusively in tuberculosis nursing and control duties.

Public Health Nurses attending the Health Visitors Training Course spend one week at the Centre as part of their prescribed course of training. This week coincides with the first week of the Main Course which consists of Lectures, Lecture-Demonstrations and Demonstrations which they attend together with the nursing supervisory staff.

Senior nursing officers (State Matrons, Health Matrons, Hospital Matrons, Sister Tutors, Male Tutors and Midwifery Tutors) attend Orientation Courses on Tuberculosis Control of three days' duration. The main purpose of these orientation courses is to broaden the concept of the National Tuberculosis Control Campaign at all levels of the Nursing Service and to ensure enlightened supervision and co-ordination of effort.

Since 1961 as many as 589 Nursing personnel comprising all grades and categories of nurses have completed courses of instruction at the Centre (37 Matrons, 9 Nursing Tutors, 74 Nursing Sisters, 185 Staff Nurses, 162 Assistant Nurses, 58 Student Nurses, 16 Health Visitors, 23 Hospital Assistants, 25 Public Health Inspectors).

All these nursing personnel are now actively engaged in prosecuting the control campaign particularly the BCG vaccination drive throughout the country.

Mass X-ray Operators constitute another important category of technical personnel receiving training in the National Tuberculosis Centre in Kuala Lumpur. They are trained to operate the Mass Miniature X-ray Units deployed all over the country in the case-finding drive. Young lads in their late teens or early twenties fresh from school with a Lower Certificate of Education are recruited for training as Mass X-ray Operators. After an introductory course in Public Relations and orientation in the different aspects of the tuberculosis control project to provide them with an enlightened approach to their particular field of operation they are put through an intensive three-month practical course in mass X-ray techniques and procedures. They learn how to operate the mass miniature X-ray units, how to take care of these very expensive pieces of equipment by learning the basic elements of X-ray technology and electricity. They are also taught how to process films in the dark room, how to record particulars of persons to be X-rayed correctly on the X-ray cards, and also all the technical, clerical and ethical procedures connected with the whole mass X-ray operation. Then they are posted to the field to operate the mass X-ray units under the supervision of senior operators and radiographers. When they have attained proficiency in this work — usually after 3 to 6 months in the field — they are ready to take independent charge of mobile or static X-ray units. Three mass X-ray operators are assigned to each mobile unit and two to each static unit.

This novel yet simple approach to the problem of finding suitable personnel in adequate numbers to operate mass miniature X-ray units is proving to be very successful and extremely effective. When the scheme was first mooted considerable opposition was encountered from some quarters particularly in view of their youth and the highly technical and hazardous nature of the work expected of them. It was suggested that only trained radiographers were qualified to operate X-ray units. With the country-wide shortage of radiographers which was expected to continue for some years the prospect of launching a mass chest X-ray drive to implement the Tuberculosis Control Plan did not at all look bright. Radiographers take three years to qualify which meant that the case-finding drive using mass X-ray units would have to be postponed for at least three years. This delay the Division of Tuberculosis was not prepared to accept.

It stands to the credit of all the parties who had grave misgivings about the proposed scheme, that good sense prevailed in the end and the scheme was allowed to proceed as planned. When the scope of the work expected of Mass X-ray Operators was properly clarified and its limitations carefully defined, opposition to the scheme soon vanished. The modern mass miniature X-ray unit is extremely easy and safe to operate. With its built-in safety and automatic devices the whole operation of X-ray taking is a simple push-button procedure requiring no special

technical training or knowledge. All that is necessary is to posture the subject and then press the button. The intricate electronic automatic devices built into the unit compute exposure time and X-ray dosage more accurately than a human being. The whole mass X-ray procedure is a repetitive type of work which would not appeal to a qualified radiographer who is trained to exercise judgement and initiative in the many intricate techniques of medical radiography of which the operation of mass miniature X-ray units forms but a small and uninteresting part. Assigned whole time to mass miniature chest radiography such a highly qualified and skilful technologist as a radiographer would find the work tiresome and even frustrating. So it was agreed to give the scheme we had in mind a try. The results have been most gratifying and the experiment has been a resounding success.

Since 1962 when this training started, 50 mass X-ray operators have been trained and are operating the mobile and static mass X-ray units deployed all over the country in the Case-finding Drive. Another 50 will be recruited for training in 1964.

Laboratory Assistants

Also receiving training at the Centre are Laboratory Assistants, a very important category of technicians, whose work is vital to the successful prosecution of the control campaign, for the diagnosis of infectious tuberculosis can be made only by them in the laboratory. The X-ray does not make a diagnosis of tuberculosis. It merely picks up the suspects who might or might not be cases of tuberculosis. It is in the laboratory that the diagnosis is made after the examination of sputum specimens and it is in the laboratory that progress of patients under treatment and their eventual cure is assessed and established. The laboratory with its technicians working in it constitute the sheet anchor of the National Tuberculosis Control Campaign.

Training of laboratory assistants forms an important and integral part of the Training Programme at the Centre. Unfortunately this particular vocation does not appear to attract enough applicants for reasons which are pretty obvious. It is not pleasant work examining sputum specimens all day and furthermore the qualifications required of prospective candidates command more attractive and lucrative jobs in other fields. Little wonder that when 20 candidates were offered employment only 13 accepted. Of these, three left during the training course for better jobs and most of the remaining ten after they have been trained as laboratory assistants are constantly keeping a watchful eye for openings in other fields and vocations. It is a problem which is actively engaging the attention of the Division of Tuberculosis and a solution is being sought. Of the ten that have been trained, six have been posted to State and District Tuberculosis Clinics which were in urgent need of this essential laboratory service.

There are now in all 40 vacant posts for laboratory assistants in the Division of Tuberculosis and it is proposed to recruit them this year (1964) in two batches of 20 each for training. After they have been trained they will be posted to laboratories in state tuberculosis centres and district tuberculosis clinics. How many we shall be able to retain after training them is open to speculation. If the exodus continues an alternative scheme will have to be considered.

Other technical personnel

Also receiving training in the Centre are doctors, social welfare assistants and paramedical personnel required for the control campaign.

THE B.C.G. VACCINATION DRIVE

The BCG Vaccination Drive constitutes the second prong of the attack that is being mounted against tuberculosis in Malaya. It was launched almost simultaneously with the training programme and has kept pace with it, being steadily extended as more and more trained personnel become available.

Protection given by BCG

BCG vaccination is a most valuable weapon in the fight against tuberculosis. Its efficacy as a protective vaccine providing protection against tuberculosis infection and disease has been established beyond doubt. A successful vaccination reduces the chances of infection developing into tuberculous disease by about 80%; or to put it another way: of every five cases of tuberculosis appearing in unvaccinated persons, four could have been prevented by vaccination. It has also been shown that if tuberculosis does develop in vaccinated persons it tends to run a mild course, responds readily to treatment and rarely proves fatal. In infants and children BCG affords absolute protection against the fulminating fatal forms of tuberculosis — tuber-

culous meningitis and miliary tuberculosis, which occur with alarming frequency in Malaya. No case of tuberculous meningitis or miliary tuberculosis has yet been reported in a child who has been successfully vaccinated with BCG.

Selection of Groups for Vaccination

As the infection rate amongst the child population in Malaya is extremely high (25% at the age of 5, 50% at 10 years and 75% at 15 years of age) protection with BCG should be given at the youngest age group possible, preferably at birth. BCG is, therefore, offered to all babies born in hospitals, infants and children attending Maternity and Child Health Clinics, primary school children, contacts of known cases and hospital workers who have not yet been naturally infected. In other words all those who are living and working "at risk" are offered BCG. Since at the age of 15 years three out of four are already naturally infected and are, therefore, too late for protective vaccination, the BCG drive in the country is directed mainly at the child population. The aim is to provide protection with BCG, before the child can be naturally infected with virulent tuberculosis, so that when he is subsequently challenged with tuberculous infection he is able to resist it.

Priorities

As only readily accessible groups of the child population can be covered and different states vary in their ability to cover even the accessible groups the following priorities have been established in the BCG drive:

1. All babies born in hospitals are automatically vaccinated 12 hours after birth by nurses in maternity hospitals and wards. All these nursing personnel have received training at the National Tuberculosis Centre.

2. Most of the larger Maternity and Child Health Clinics are able to vaccinate non-reactors to Tuberculin (not yet naturally infected) amongst the infants and children attending the clinics. Infants under three months of age are given BCG without a preliminary tuberculin test and because only one attendance is necessary, most of the smaller clinics are able to undertake this. The coverage of infants and pre-school children in Maternity and Child Health Clinics will increase considerably in coming years as Malaya is now engaged in an extensive Rural Development Programme which envisages in the Health Sector the provision of essential Maternity and Child Health Services for the entire rural population. Each rural district of about 50,000 inhabitants will be served by a Rural Health Unit consisting of a Main Health Centre, 4 Sub-Health Centres and 25 Midwife's Clinics. Each midwife's clinic will serve a population of 2,000 persons and a Sub-Health Centre 10,000 persons. Most of the Health Centres and a considerable number of midwife's clinics have been built, equipped and adequately staffed. All nursing personnel posted to these centres and clinics receive training at the National Tuberculosis Centre in Kuala Lumpur in tuberculin testing and BCG vaccination techniques, and are able to undertake BCG vaccination of infants and pre-school children in addition to their other duties at these centres and clinics. The coverage of infants in the rural areas will, therefore, increase substantially and rapidly with the development and expansion of the rural health services.

3. Contacts of known cases and hospital workers who are tuberculin negative (not yet naturally infected) enjoy equal priority with newborn babies in the BCG programme, contacts of infectious cases claiming the highest priority.

4. Primary school children come next on the priority list. In this group primary school *entrants* are accorded priority over *leavers* and the children in the other classes are offered BCG only after all entrants and leavers have been covered before the school year is out. The extent of cover varies from state to state but every effort is made to cover at least the primary school entrants.

Progress and Targets

Since the control campaign started in June 1961, more than half a million infants and children have been vaccinated. Of these more than 200,000 were newborn babies. 85,196 were vaccinated in 1961, 164,024 in 1962, 213,573 in 1963. The target for 1964 is 350,000 children and when the control campaign is fully developed in 1965 it is hoped to get an annual coverage of half a million children.

THE CASE-FINDING DRIVE

The Case-finding Drive which constitutes the third prong of the attack on tuberculosis

was a much later development than the other two — the Training Programme and the BCG Vaccination Drive — which became operational in June 1961 almost simultaneously with the official launching of the control campaign. It was only two years later, that it was possible to institute the case-finding drive on a mass country-wide scale.

Launching of Mass Case-finding Drive

The case-finding drive on a mass scale was officially launched by the Minister of Health on Monday, 4th March 1963. To mark the occasion a procession was held in Kuala Lumpur of 8 mass miniature X-ray units and 11 vehicles with all the departments and units of the National Tuberculosis Centre participating. The purpose of the procession was to display the mobile and static X-ray units which would be going into operation, give the widest publicity to the campaign through the media of the Information Services, the Press, the Malayan Film Unit and Radio Malaya to make every Malayan fully aware of the attack which was being mounted on a national scale to eliminate the menace of tuberculosis from his midst and at the same time to impress upon him the important part he was expected to play in the achievement of this object.

The institution of the Mass Case-finding Drive opened a new and important phase in the development of the National Tuberculosis Control Campaign. The first phase — the Training Programme and the BCG Vaccination Drive — had already been developed and was being firmly consolidated. With the advent of this second phase — the Case-finding Drive — for the first time since the National Control Campaign was launched in June 1961, all three components of the campaign started operating together in the all-out attack on tuberculosis.

Not that no case-finding was carried out before 1963. For one whole year prior to March 1963, two mobile and two static mass X-ray units were operating on a pilot or demonstration footing, serving as an exercise to provide training for mass X-ray operators and experience for tuberculosis control personnel in the many and varied technical aspects of the case-finding procedure. Although more than 150,000 persons were X-rayed during the course of this exercise it was nevertheless merely a pilot or demonstration project.

During 1963, 9 Mobile and 9 Static X-ray Units were operating in the country. In 1964 another 6 Units (two mobile and four static) will be put into operation to provide a complete cover of the whole of Malaya. Except for Penang all the states in Malaya are being covered with mobile X-ray units since March, 1963. Penang will be served with a mobile unit commencing April, 1964.

Static units are operating in Kangar, Alor Star, Taiping, Kuala Lumpur (two units), Seremban, Malacca, Johore Bharu and Kota Bharu. During 1964 static units will be installed in Penang, Ipoh, Kuantan and Kuala Trengganu.

Selection of groups for Mass X-ray

Only selected groups of the adult population are X-rayed in the Case-finding Drive:

1. **High Prevalence Group** (those likely to produce a high yield of cases).
   - (i) Out-patients and in-patients of all hospitals.
   - (ii) Patients referred by private medical practitioners.
   - (iii) Contacts of known cases of tuberculosis.
   - (iv) Self-referred persons: any member of the general public who wishes to have a chest X-ray usually on account of symptoms.
2. **Danger Group** (those who if suffering from tuberculosis constitute a danger to the community as their work brings them into frequent and prolonged contact with the general public, particularly children).
   - (i) Food handlers (hawkers, cooks, waiters, coffee-shop and eating-house assistants, stall-holders, etc.) barbers, hairdressers, trishaw riders, bus conductors, etc.
   - (ii) All school teachers and school children over 15 years of age.
   - (iii) Domestic servants, baby amahs, etc.
3. **Other Groups**
   - (i) Hospital staff and their families.
   - (ii) Government Servants and their families.
   - (iii) Members of the Armed Forces and their families.

There are of course other ways of covering the adult population of a country, particularly the conventional way which is to divide the

country into areas and then proceed to X-ray the whole adult population area by area until the whole country is covered and repeat the survey every three or four years. In Malaya we have decided to cover only selected groups of the population for two very good reasons. Firstly, we wish to derive the utmost benefit in our campaign with the limited resources available. Secondly, we must endeavour to keep the number of persons requiring further investigation after X-ray at a level which is comfortably within the capacity of our district hospitals to handle efficiently. Even when the available resources improve in quantity and quality it is not the intention to alter this policy but rather to increase the coverage of the selected groups. Experience has shown that it is infinitely more rewarding to focus the case-finding effort on selected groups of the population than dissipate it by attempting to cover the whole eligible population on a geographical basis. Specificity in approach concentrating on the groups that matter seems more logical and sensible than indiscriminate cover in an all-embracing operation.

The Mass X-ray Unit

The primary aim of the case-finding drive is to find the infectious cases of tuberculosis in the community and render them non-infectious with treatment. The mass miniature X-ray machine is a most valuable electro-mechanical device for the quick detection of such cases but it has its limitations which, regretfully enough, are not often fully recognized. It is capable of X-raying up to 100 persons an hour, provided the X-ray cards carrying particulars of the persons to be X-rayed are completed in bulk before hand. When the cards are completed individually before each X-ray examination the time taken for the whole X-ray procedure is hardly two minutes from the moment one presents oneself for examination. In actual practice a combination of these two methods usually obtains and provided a continuous flow of examinees is assured, each X-ray unit can comfortably X-ray 500 persons in one working day. In order to make its operation worthwhile each unit should X-ray at least 20,000 persons a year, though 40,000 a year is more like the figure which could be achieved without much difficulty.

X-ray does not provide diagnosis — importance of bacteriology

What of the limitations of the X-ray machine? Although it is a very valuable tool in the mass case-finding operation it does not by itself actually detect infectious cases of tuberculosis. The X-ray does not provide a diagnosis. All it does and all it is capable of doing is to separate the examinees into two distinct groups: a large group comprising (according to our experience in Malaya) about 95% of those examined who are free from an X-ray abnormality of the lungs and a small group of about 5% whose X-rays reveal a lung abnormality. The later group, the so called "X-ray abnormals" require further investigations in a chest clinic of which sputum examination is the most important before a diagnosis of infectious tuberculosis can be made. The mass X-ray operation merely picks up "X-ray abnormals" for further investigation. It does not and cannot make a diagnosis. The diagnosis is made only after further examination in a chest clinic, particularly bacteriological examination in the clinic laboratory.

Investigation of X-ray abnormals

A lung abnormality picked up by the X-ray does not, therefore, automatically mean tuberculosis as is erroneously believed by many people. There are many conditions, some serious, others quite harmless, which produce shadows in the X-ray film which are quite indistinguishable from those produced by tuberculosis. The chances of the abnormality being tuberculosis, according to our experience in Malaya, are roughly even. About one half of the "X-ray abnormals", therefore, are found to be suffering from active tuberculosis. Of these nearly half have infectious tuberculosis.

In case-finding it is not the taking of an X-ray which is important but the subsequent investigation in the chest clinic of the X-ray abnormals discovered to determine which amongst them are cases of tuberculosis. This involves the hospital concerned in extra work. Also the hospital must be within easy reach of the persons recalled for further investigation. For these two very important reasons our case-finding drive operates in and near our hospitals and is geared to keep within the capacity of the hospital to cope efficiently with the extra load.

The success of the case-finding drive is measured not by the number of persons X-rayed but by the speed, efficiency and completeness of the investigation of "X-ray abnormals" discovered. The taking of an X-ray merely initiates the case-finding drive. It is the subsequent investigation of *all* the X-ray abnormals discovered which brings the whole

operation to a successful conclusion. This important point needs particular emphasis because it is often over-looked in the understandable desire and over-eagerness to X-ray as many people as possible as if the taking of an X-ray in itself is the paramount aim of case-finding. The case-finding drive initiated by the X-ray operation concludes successfully only when each and every X-ray abnormality discovered has been fully investigated and dealt with as is appropriate to the particular case.

Deployment of Mass X-ray Units

Apart from the static X-ray units which operate continuously throughout the year in the hospitals in which they have been installed, the mobile units are so deployed that they make regular monthly visits to all the hospitals in the country for two to six consecutive days depending upon the size of the hospital and the population to be served in the area. When circumstances require that a special group should be X-rayed outside the hospital a special mobile unit is usually assigned for this purpose so as not to interfere with the scheduled programme of the unit operating in that area. The special groups served under this scheme are usually Government Servants, members of the Armed Forces, licensees of Municipalities and other Local Authorities, factories, etc. provided, of course, they fall within the selected groups of the population listed on page 247.

The mobile units are deployed from and controlled by the National Tuberculosis Centre in Kuala Lumpur. They operate for eleven months in the year, returning to the Centre in December for servicing when the mass X-ray operators and drivers of the vans take their annual vacation.

Central Processing and Reading of X-rays

All the rolls of exposed X-ray film are processed in the National Centre to ensure that the diagnostic quality of the X-ray picture is maintained at a uniform optimal standard. Every roll is scrupulously inspected for flaws due to poor technique or reduced efficiency of the equipment and steps are immediately taken by our technologists to rectify them.

The X-ray films are subjected to dual independent reading by experienced doctors in the Centre. The X-rays which show abnormalities are returned forthwith to the hospital responsible for further investigation of the "X-ray abnormals" discovered. At the same time letters are despatched to the "X-ray abnormals" themselves requesting them to report to the hospital concerned for this investigation. The Medical Officer in the hospital who undertakes this investigation is obliged to complete and return to the National Centre monthly returns of the progress he has made in carrying out the investigation. Constant reminders are sent to defaulters who fail to turn up for investigation and in extreme cases home visits are made to urge their attendance.

No News is Good News

To avoid tedious secretarial work and to save considerable expense, no reports are made on normal X-rays nor are the persons concerned notified by letter. No news should be regarded as good news.

The result of the chest X-ray examination when an abnormality is discovered, or of any subsequent investigation at the chest clinic is treated as strictly confidential. It is disclosed only to the individual concerned, never to any other person however closely he may be related to or associated with him, except with his express permission.

Progress and Targets

Since the case-finding drive was instituted initially as an exercise and later on a mass scale, more than 400,000 persons have been X-rayed by our mass miniature units and about 20,000 persons have been found to haxe X-ray abnormalities suggestive of tuberculosis. Not all these 20,000 "X-ray abnormals" have been investigated to find out how many of them have active tuberculosis and how many are in an infectious state. The response to recall for further investigation has been far from satisfactory. A substantial number — almost a quarter of them — have not turned up at all despite several reminders. Of those who have responded to recall, almost one quarter have failed to complete the full investigation required, having defaulted after one or two attendances at our chest clinics. There are many reasons for this lack of cooperation. The most important is just plain apathy which is a pretty universal human failing not at all peculiar to Malaya. The experience of other countries is much the same. Human beings all over the world will not submit readily and whole-heartedly to a full and complete medical examination if they feel well and are free from distressing symptoms. Early tuberculosis is notorious for the remarkably few and quite inconsequential symptoms, if any, it produces

and the slow and insidious way in which it develops. Another important reason is fear — fear of detection, fear of losing one's job, fear of social ostracism, fear of prolonged hospitalisation, fear of being a hopeless case. We know that most of these fears are quite groundless but there is little we can do to dispel them until education, particularly health education, and the healthy growth of the sense of civic responsibility are able in their own time to bring about enlightenment and proper understanding.

This sad lack of response to recall is causing some considerable concern and many methods are being devised and tried to reduce the defaulter rate. The most effective method we have found is direct personal approach by visiting the home of the defaulter and urging him to attend at the chest clinic for further investigation. Unfortunately this method cannot be applied with equal effectiveness throughout the country owing to shortage of trained personnel. Nor is there any prospect of our being able to do so in the near future as we are not likely for some considerable time to have enough home visitors to chase every defaulter. We have, therefore, established certain priorities in chasing defaulters. Only those with gross X-ray abnormalities who if tuberculous are very likely to be infectious are visited in their homes to ferret them out for further investigation. They represent about a quarter of those found with X-ray abnormalities. Home visits are also made to infectous cases who default in their treatment. In this way the limited number of trained personnel available are used to best advantage by concentrating on those defaulters who pose a real danger to the community.

About 7,000 new cases of tuberculosis have been discovered in the case-finding drive and put on active treatment. Almost all those with gross X-ray abnormalities have been rounded up for investigation and all the infectious cases discovered placed under close and careful supervision to ensure that they take their treatment regularly. Thus by a judicious use of the limited number of trained personnel available the utmost benefit is derived in protecting the health of the community.

It must be remembered, however, that a normal chest X-ray is no guarantee that a person will continue for the rest of his life to be free from tuberculosis. Our ultimate aim is to make every adult over the age of 15 years subscribe voluntarily to the inviolable rule of having a chest X-ray once a year. Only then will the case-finding drive achieve a complete cover of the eligible population of the country. Although this is an ideal no country in the world has succeeded in attaining, it must, nevertheless, constitute the ultimate aim of the case-finding drive in Malaya.

Public Support and Cooperation Most Essential

The success of the campaign will depend on the cooperation and support of the general public, for this is really their problem. If they are determined to rid this country of this curable and preventable disease and so protect the health of those who are still uninfected, they *must* come forward for a regular chest X-ray and they *must* report for further investigation when requested to do so and they *must* also persist with treatment when they are found to be suffering from tuberculosis.

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AN INVESTIGATION INTO THE COMMON CAUSES OF BLOODSTAIN DETERIORATION

PART I

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Pioneer work by investigators like Landsteiner Jansky and Moss in the early years of this century led to the discovery of blood groups. Since then many workers have increased the number of blood agglutinogens, such as, M N S and P agglutinogens, Rh, factors, etc., which can be identified and differentiated by blood grouping techniques.

The blood group specialist is, in most cases, concerned with blood group determinations, for blood transfusion work, when he is solely interested in establishing the compatibility or otherwise of the donor and recipient bloods. The forensic scientist, on the other hand, deals with dried bloodstains, with a view to determine their source in cases of assault, rape or homicide and almost invariably confines himself to the determination of A B O groups.

One of the authors (S.S.), in the course of work, carried out in the Penang Laboratory has come across bloodstained exhibits which often do not give conclusive results as to their origin and blood groups. Such cases are commonly reported as being inconclusive due to putrefaction or deterioration of the bloodstains although there is no departmental information sheet on the causes and possibly the extent of decomposition caused by interfering substances. It was with a view to find out the common substances or conditions that cause the decomposition, and therefore, are responsible for the inconclusive results, that this investigation was undertaken. In all thirty-four conditions have been investigated and the findings are summarised.

Method of Securing Blood Samples

The supervisor of the Blood Bank, Penang was informed in advance of the type of blood required and when a donor of that group arrived at the bank he would notify one of us (O.Y.H.K.) who would arm himself with two vials each containing a few crystals of anticoagulant[1] (mixture of ammonium and potassium oxalates). After the supervisor had finished taking his sample; the delivery tube just before disconnection, was allowed to drip into our vials. The vials were then stoppered and the anticoagulant uniformly dispersed by a few gentle inversions of the vials. The agglutinin and agglutinogen titres of each sample of blood were next determined and the blood then used to stain filter papers as indicated below:—

1. **Stain Air Dried** — The filter paper was stained with blood and allowed to dry in an air-conditioned room.

2. **Stain Sun Dried** — The filter paper was stained as above and the wet stain placed in bright sunlight for about three hours (Temp. 96°F.).

3. **Bacteria and Blood** — Some Staphylococcus pyogenes, obtained from I.M.R. were suspended in deionised water. This suspension was employed to wet the filter paper which was allowed to stand for about five minutes. Then the bacteria impregnated paper (still wet) was stained with blood and kept moist for 24 hours by placing in a dessicator containing water.

4. **Polluted Blood** — Fresh blood was innoculated with the above bacteria, incubated at 37°C. for 24 hours and then used to stain filter paper.

5. **Stain Oven Dried** — Wet stain dried in an oven at 100°C. for ca. 3 hours.

6. **Moist Paper Stained** — Paper moistened with tap water and the wet area stained with blood. The blood gave two types of absorptions — one darker (more intense) than the other. Dried in air-conditioned room.

7. **Bloodstain Washed** — Stained filter paper, air dried and then held in running water (from tap) for approximately one minute.

8. **Perspiration and Bloodstain** — Paper stained with perspiration, dried and then blood stained. Dried in room.

9. **Blood and Soap Solution** — A 1% w/v solution was employed to wet a piece of filter paper. This was dried and then stained with blood.

10. **Bloodstain and FAB Solution** — Paper stained with 1% w/v FAB solution, dried and then stained with blood.

11. **Bloodstain Ironed** — Paper stained with blood, air dried and after ca. 2 hours ironed with an electric iron for approximately one minute.

12. **Bloodstain and Coconut Oil** — The paper was stained with coconut oil, allowed to dry and then counter stained with blood.

13. **Bloodstain and Vaseline Hair Cream** — The filter papers were smeared with vaseline, which was allowed to partially dry and then stained with blood.

14. **Bloodstain and Hydrogen Peroxide (10 vols.)** — Filter paper stained with blood and then hydrogen peroxide added drop by drop onto the wet stain. Gas was evolved and the wet stained area was foam covered. When the hydrogen peroxide was dropped onto a clean (free of blood) area of the filter paper no foam was formed.

15. **Bloodstain and Loam** — The filter paper was stained with blood and the wet area was covered with wet loam (taken from a flower bed) for 24 hours.

16. **Bloodstain and Clean Sand** — Same as above except that clean dry sand from the store was employed.

17. **Bloodstain and Starch** — The filter paper was soaked in starch solution (1% w/v), dried and then stained with blood.

18. **Bloodstain and Tannic Acid** — The filter paper was soaked in an aqueous solution of tannic acid (1% w/v) allowed to dry and then stained with blood.

19. **Bloodstain and Dilute Acetic Acid** — The filter paper was soaked in dilute acetic acid (1% w/v), allowed to partially dry and then stained with blood.

20. **Bloodstain and Perfume** — Paper stained with "PATRA" perfume dried and then counter stained with blood.

21. **Dettol** — A 1% v/v solution was prepared. (a) Filter paper was wetted with dettol dilution, allowed to dry and then stained with blood. (b) A filter paper was stained with blood, allowed to stand for about 10 minutes and then the stained area wetted with dettol dilution.

22. **Carbolic Soap** — A 1% w/v solution was made. (a) A filter paper was wetted with the solution, allowed to dry and then stained with blood. (b) Stained a filter paper with blood, allowed it to stand for about 10 minutes and then wetted the stained area with the carbolic soap solution.

23. **Alcohol** — The filter paper was stained with blood, allowed to stand for 10 minutes and then wetted with 95% ethanol.

24. **Heat** — Two filter papers were stained with blood, allowed to stand at room temperature for about 5 minutes and then; (a) one paper was maintained at 150°C. for one hour, and (b) the other paper was placed for 5 minutes in an air oven previously adjusted to 200°C.

25. **Formalin** — A filter paper was stained with blood, allowed to stand for 10 minutes, and then wetted with formalin solution (20%).

26. **Betel Nut** — Gopal, the gardner was made to chew some betel nut and then discharge the coloured sputum onto a filter paper. After the spittle had dried it was counter stained with blood.

27. **Saliva** — A few filter papers were wetted with Gopal's saliva, allowed to dry and then blood stained. (Gopal's blood group is O).

28. **Rust** — Some iron filings were sprinkled onto filter papers, moistened with water and then allowed to rust. The paper with

rust covered filings was next stained with blood.

29. **Dessication** — Filter papers were stained with blood and kept in dessicators (containing concentrated H_2SO_4) for two days.

30. **Dilute Acetic Acid** — Filter papers were stained with blood, allowed to semi-dry (5 minutes) and then wetted all over with dilute acetic acid (1% v/v).

31. **Bacteria Coli** — (a) Bacteria impregnated papers were moistened with blood and kept moist for 48 hours. (b) Blood samples were innoculated with B. coli, incubated at 37°C. for 48 hours and then used to stain filter papers.

32. **Staphylococcus pyogenes** — Staining as in the case of B. coli conditions (a) and (b).

33. **Incubated Blood** — Samples of blood were incubated for 48 hours at 37°C. and then used to stain filter papers.

34. **Hæmolysed Blood** — Samples of blood were allowed to stand at room temperature (ca. 30°C.) until completely hæmolysed (no cells visible under the miscroscope) and then used to stain filter papers.

PLAN ADOPTED

Grouping tests and whenever possible simultaneous agglutinin titre determinations on the bloodstains from each group of blood were carried out at the following times:

(a) bloodstains one or two days' old.

(b) bloodstains two weeks' old.

(c) bloodstains two months' old.

Precipitin tests at dilutions of 1:1000; 1:2000 and 1:4000 were also performed on the two month old stains.

A further lot of four blood samples, one per blood group, were secured. These were used to study conditions not previously studied and also modifications of a few of the conditions already investigated with the first four samples of blood. The latter modified conditions were investigated to determine whether or not such modifications would accentuate their adverse effects.

METHOD USED

Each sample of fresh blood, unless otherwise stated, was separated by centrifugation into: (a) cells and (b) plasma. The cells were washed until clear (three times) with physiological saline and then a suspension (2%) in normal saline made. A typical agglutinogen and agglutinin titre determination is described below:

Agglutinogen titre of Group A Blood — O-sera and anti A-sera of known titres were each titrated with physiological saline as in the case of saliva grouping[2]. To the diluted sera an A-cell suspension (2% cells of blood to be grouped) was added and the Dreyer tubes incubated at 37°C. for one hour after which agglutination readings were taken with the aid of a microscope.

Agglutinin titre of Group A Blood — Two volumes (0.008 cc) of the plasma from this blood was diluted with physiological saline and then to one row of dilutions a known A-cell suspension was added and to the adjacent row a similar B-cell suspension added. The tubes were again incubated and read after one hour.

Agglutinin titre of Group A Bloodstain — (cf. Grouping of Saliva Stains[2]). About one sq. cm. of stained filter paper was cut into very small pieces, transferred into an extraction tube and extracted with the minimum quantity of physiological saline. The extract, free from suspended matter, was diluted in two rows of Dreyer tubes. To these known A-cell and B-cell suspensions (2%) in physiological saline were added and the tubes incubated at 37°C. for one hour after which the agglutination readings were taken.

Agglutinogen titre of Group A Bloodstains — (cf. Grouping of Saliva Stains[2]). Bloodstained paper was extracted with O-sera, the extract diluted with physiological saline and then mixed with A-cell and B-cell suspensions. Agglutination readings were then recorded. In each set of experiments a blank was performed by extracting clean filter paper and then treating the extract in a manner similar to extracts from bloodstained areas.

Precipitin Test — The bloodstain on each filter paper was extracted (overnight) with

physiological saline and then the clear extract diluted further with saline solution to yield dilutions of 1 in 1000, 1 in 2000 and 1 in 4000. These dilutions were then carefully layered, in three separate Dreyer tubes, onto some human anti-sera and the common surface observed three times for signs of opacity. The observations were spaced at ten minute intervals. A blank, extracting unstained filter paper was performed in each case.

DISCUSSION

In the section of "Method Used" only a very brief outline is given since a detailed account of essentially the same method has already been given elsewhere[2].

The possible contaminants chosen for study in the case of the first four blood samples were those that would commonly be expected to be present on articles stained with blood.

Before the precipitin reaction was performed, the human anti-sera was tested with known dilutions (v/v) of human sera and found to have an activity of 1 in 6000. The stain extracts were then diluted according to the foam test method[5], taking good care to use tubes of the same dimensions and the same pipette for preparing the different dilutions. It was found that with the exception of group A blood, even stain number 1, the reference standard did not give a precipitin positive reaction at the maximum dilution of 1 in 4000. The contaminants that produced adverse effects were heat (100°C.), tannic acid, acetic acid, heat (electric iron), perspiration, etc. These contaminants must have altered the nature of the serum proteins.

For all grouping work in this project fresh A and B cells were always taken from the same two persons so as to ensure that the "quality" of the cells was maintained at the same level throughout the project. Also the agglutination readings have been taken by the same person with the aid of the same microscope, thereby maintaining a uniform consistency in readings.

In experiments on agglutinin titre determinations, the blank was performed by diluting with physiological saline anti-A and anti-B sera of high agglutinin titres and then adding the A and B cells respectively. This determined the maximum agglutinating ability of the cells employed. Any agglutinations, with saline extracts from bloodstains, short of those shown by the blank may therefore be alluded to either the presence of agglutinins of low titres in the stains or the absorption of agglutinins by the filter paper carrying the stains. Since stain No. 1 in each type of blood has been taken as reference for comparison purposes, it may be logically inferred that any adverse deviations from stain No. 1 were due to decreases in agglutinin titres only.

In the extractions with sera, a standardised procedure was adopted with a view to minimise irregularities in results due to differences in stained areas and sera volumes used for extraction. Stains of one sq. cm. area were extracted with two drops of sera delivered from the same micro pipette. This procedure yielded approximately the same volume of sera in excess.

The results of stain number 1 in each case have been taken as a reference standard for all the other stains obtained from the same sample of blood. Therefore, a comparative study of the results obtained for other stains with those of stain number 1 would indicate any decrease in the agglutinogen/agglutinin potency.

Grouping tests on the first two samples of blood (groups A and B) were carried out with O-sera as the extraction medium and the results obtained in each case were found to conform to theory. With the third sample of blood (group AB), however, the stain extractions showed different degrees of cell agglutination although the fresh blood yielded equal titres for both the A and B agglutinogens and the O-sera used had equal agglutinin titre. Fresh stains of the same blood were again extracted, now employing anti-A and anti-B sera and this time the extracts agglutinated to the same extent with both A and B cells respectively, thus indicating the uneven removal[3] of agglutinins from the O-sera. A further sample (fourth) of blood (Group AB) was also found to "misbehave" with O-sera and yet comply with theoretical requirements when anti-A and anti-B sera were substituted

instead. As a result of the above it has been decided to replace the conventional O-sera (used in the Penang Laboratory since the commencement of blood group work) with anti-A and anti-B sera for all group determinations of bloodstained exhibits. Extractions with diluted anti-A and anti-B sera have been found to yield more conclusive results than those obtained by the use of undiluted sera.

No attempt at differentiation of the subgroups of groups A and AB has been made in this piece of work.

One Day Old Stains — A comparison of the results derived from the twenty, one-day old stains, of each blood group was made and a summary of the conditions/contaminants lowering agglutinogen and agglutinin titres is given:—

Agglutinogens

BLOOD GROUP AB

Stain No.	Active Agent	Agglutinogen affected
2	Sun dried	A and B; A = B
5	Heat at 100°C.	A
7	Stain washed	B
11	Heat (electric iron)	A and B; A > B
15	Loam	A and B; A > B
16	Clean Sand	A and B; A > B
17	Starch	A and B; A = B
18	Tannic Acid	A and B; B > A
19	Acetic Acid	A and B; A = B
20	Perfume	A and B; A > B

BLOOD GROUP B

Stain No.	Active Agent	Agglutinogen affected
5	Heat 100°C.	B
6	Wet paper stained	B
11	Heat (electric iron)	B
18	Tannic Acid	B

BLOOD GROUP A

Stain No.	Active Agent	Agglutinogen affected
3	Bacteria	A
5	Heat 100°C.	A
6	Wet paper stained	A
11	Heat (electric iron)	A
15	Loam	A
17	Starch	A

From the above it could be reasonably deduced that bloods belonging to group AB are most prone to agglutinogen deterioration whereas group B and group A bloods suffer almost equal degrees of agglutinogen inactivation.

Agglutinins

BLOOD GROUP O

Stain No.	Active Agent	Agglutinin affected
2	Sun dried	a and b; a > b
3	Bacteria	a and b; a > b
4	Polluted blood	a and b; a > b
5	Heat (100°C.)	a and b; a > b
6	Wet paper stained	a and b; a > b
7	Stain washed	a and b; a = b
8	Perspiration	a and b; a > b
9	Soap	a and b; a > b
10	Synthetic Detergent	b
11	Heat (electric iron)	a and b; a = b
12	Coconut oil	b

13	Vaseline	b
14	Hydrogen Peroxide	b
15	Loam	a and b: a > b
16	Clean Sand	a and b: a > b
18	Tannic acid	a
19	Acetic Acid	a and b: a = b
20	Perfume	a

BLOOD GROUP B

Stain No.	Active Agent	Agglutinin affected
3	Bacteria	a
5	Heat (100°C.)	a

BLOOD GROUP A

Stain No.	Active Agent	Agglutinin affected
2	Sun dried	b
3	Bacteria	b
4	Polluted blood	b
5	Heat (100°C.)	b
6	Wet paper stained	b
7	Stain washed	b
8	Perspiration	b
9	Soap	b
10	Synthetic Detergent	b
11	Heat (electric iron)	b
12	Coconut oil	b
13	Vaseline Hair Cream	b
14	Hydrogen Peroxide	b
15	Loam	b
16	Clean Sand	b
17	Starch	b
18	Tannic acid	b
19	Acetic Acid	b
20	Perfume	b

A survey of the above summary of agglutinin results indicates that bloods of group O and group A are most susceptible and blood of group B least susceptible to changes in their agglutinin potencies.

Effect of Age — Time does not have any significant effect on the agglutinogens although it does lower the titres of the agglutinins as indicated in the summary given below:—

Agglutinogens

BLOOD GROUP AB

no effect

BLOOD GROUP B

Stain No.	Condition	Agglutinogen affected
4	Polluted Blood	b
11	Heat (electric iron)	b
12	Coconut Oil	b
13	Vaseline Hair Cream	b
14	Hydrogen Peroxide	b
16	Clean sand	b

BLOOD GROUP A

no effect

Agglutinins

BLOOD GROUP O

Stain No.	Condition	Agglutinins affected
1	Air dried	a
2	Sun dried	a
3	Bacteria	a and b; a > b
5	Heat (100°C.)	b
6	Wet paper stained	a and b; a > b
7	Stain washed	a and b; a = b
8	Perspiration	a and b; b > a
9	Soap	a and b; a > b
10	Synthetic Detergent	a
11	Heat (electric iron)	a and b; a > b
12	Coconut Oil	a and b; b > a
13	Vaseline Hair Cream	a and b; b > a
14	Hydrogen Peroxide	a and b; a > b
15	Loam	a and b; a = b
16	Clean sand	a
17	Starch	a and b; a > b
18	Tannic Acid	a and b; a > b
19	Acetic Acid	a
20	Perfume	a and b; b > a

BLOOD GROUP B

Stain No.	Condition	Agglutinins affected
1	Air dried	a
2	Sun dried	a
4	Polluted Blood	a
5	Heat (100°C.)	a
6	Wet paper stained	a
7	Stain washed	a
8	Perspiration	a
9	Soap	a
10	Synthetic Detergent	a
11	Heat (electric iron)	a
12	Coconut Oil	a
13	Vaseline Hair Cream	a
14	Hydrogen Peroxide	a
15	Loam	a
16	Clean Sand	a
17	Starch	a
18	Tannic Acid	a
19	Acetic Acid	a
20	Perfume	a

BLOOD GROUP A

Stain No.	Condition	Agglutinins affected
5	Heat (100°C.)	b
8	Perspiration	b
10	Synthetic Detergent	b
11	Heat (electric iron)	b
12	Coconut Oil	b
13	Vaseline Hair Cream	b
15	Loam	b
18	Tannic Acid	b

From the above summary it is obvious that agglutinin a is more prone to deterioration with time than agglutinin b.

Two Weeks' Old Stains — With the second lot of blood samples a few additional conditions (Nos. 21 – 34) were investigated when the stains were two weeks old. This period was considered suitable since it was felt that is was the minimum time that lapsed before bloodstained exhibits are either made available, especially from distant and remote areas, or are normally examined, unless special circum-

stances warrant immediate attention. Further by the end of the two week period, both the contaminant and to some extent, the time factor would have already exercised their adverse effects, if any. Given below is a summary of the results:—

Agglutinogens

BLOOD GROUP AB

Stain No.	Active Agent	Agglutinogen affected
23	Alcohol	a and b: a = b
24a	Heat 150°C.	a and b: a > b
24b	Heat 200°C.	a and b: a ≦ b
25	Formalin	a and b: a > b
28	Rust	b
32b	Bacteria	a and b: a > b

BLOOD GROUP B

Stain No.	Active Agent	Agglutinogen affected
24a	Heat 150°C.	b
24b	Heat 200°C.	b
25	Formalin	b
34	Hæmolysed Blood	b

BLOOD GROUP A

Stain No.	Active Agent	Agglutinogen affected
24a	Heat 150°C.	a
24b	Heat 200°C.	a
25	Formalin	a
28	Rust	a
29	Dessication	a
30	Acetic acid	a
32b	Bacteria	a

Agglutinins

BLOOD GROUP O

Stain No.	Active Agent	Agglutinin affected
21b	Dettol	a and b: a = b
22a	Carbolic soap	a and b: a > b
22b	Carbolic soap	a and b: a = b
23	Alcohol	a and b: a = b
24a	Heat 150°C.	a and b: a = b
24b	Heat 200°C.	a and b: a = b
25	Formalin	a and b: a = b
26	Betel Nut	a and b: a = b
28	Rust	a
30	Acetic acid	a and b: a = b
31a	Bacteria	a and b: a > b
31b	Bacteria	a and b: a = b
32a	Bacteria	a and b: a = b
32b	Bacteria	b
33	Incubated Blood	a
34	Hæmolysed Blood	a and b: a = b

BLOOD GROUP B

Stain No.	Active Agent	Agglutinin affected
21a	Dettol	a
21b	Dettol	a
22a	Carbolic soap	a
22b	Carbolic soap	a
23	Alcohol	a
24a	Heat 150°C.	a

24b	Heat 200°C.	a
25	Formalin	a
26	Betel Nut	a
27	Saliva	a
28	Rust	a
29	Dessication	a
30	Acetic Acid	a
31a	Bacteria	a
31b	Bacteria	a
32a	Bacteria	a
32b	Bacteria	a

Agglutinins

BLOOD GROUP A

Stain No.	Active Agent	Agglutinin affected
22a	Carbolic soap	b
22b	Carbolic soap	b
24a	Heat 150°C.	b
24b	Heat 200°C.	b
25	Formalin	b
26	Betel Nut	b
28	Rust	b
31a	Bacteria	b
32a	Bacteria	b
34	Hæmolysed Blood	b

The above results endorse the previous finding that agglutinin a is more unstable than agglutinin b.

In the assessment of potencies, (listed in the above summaries) only those conditions or contaminants which have produced a difference of one place or more in the agglutination titre, relative to that of the reference standard, have been considered to be deactivating.

The conditions/contaminants investigated may be classified under the following four headings:—

I. **Those exercising Marked Influence** — These are conditions/contaminants that deactivate either the agglutinogens or the agglutinins in everyone of the three possible bloods in each set of four studied.

II. **Those exercising Intermediate Influence** — These are conditions that lower the activity of either agglutinogens or agglutinins in at least two bloods out of the four of each set.

III. **Those exercising Slight Influence** — Under this category are included the conditions that effect either agglutinogen or agglutinin potency in only one blood out of a set.

IV. **Those exercising Doubtful Influence** — This class includes all those conditions in which apparent reduction of agglutinogen/agglutinin potency may be due to employing for extraction purposes areas of filter papers stained to different degrees.

MARKED INFLUENCE ON:

Agglutinogen		Agglutinin	
Heat 100°C.		Bacteria	
Heat (electric iron)		Heat 100°C.	
Heat 150°C.	T F	Carbolic soap	
Heat 200°C.		Heat 150°C.	
Formalin		Heat 200°C.	
		Formalin	T F
		Betel Nut	
		Rust	
		Bacteria	

INTERMEDIATE INFLUENCE ON:

Agglutinogen		**Agglutinin**	
Tannic Acid		Sun dried	
Starch		Polluted Blood	
Rust	T F	Perspiration	
Bacteria		Soap	
		Synthetic Detergent	
		Heat (electric iron)	
		Coconut Oil	
		Vaseline Hair Cream	
		Hydrogen Peroxide	
		Tannic Acid	
		Acetic Acid	
		Perfume	
		Alcohol	
		Dettol	T F
		Bacteria	
		Hæmolysed blood	

SLIGHT INFLUENCE ON:

Agglutinogen		**Agglutinin**
Sun dried		Starch
Dilute Acetic Acid		Dessication — T F
Bacteria		Incubated blood
Perfume		
Dessication		
Alcohol	T F	
Hæmolysed Blood		

DOUBTFUL INFLUENCE ON:

Agglutinogen	**Agglutinin**
Moist Paper Stained	Moist paper stained
Stain washed	Stain washed
Clean sand	Loam
Loam	Clean sand

Those conditions with "TF" against them have had a two week time factor incorporated in them, as distinct from the others without this factor.

In this project, an attempt has been made to study the effect of one condition/contaminant at a time and it has been shown that only a limited few out of the 34 conditions investigated have a marked effect and consequently render grouping tests inconclusive.

In practice however, two or more factors, such as sweat, starch, oil, heat, bacterial decay, rust, folding up of clothing with wet or semi-dry bloodstains, etc., are present on the great majority of bloodstained exhibits and their collective effect is at times so great that the more susceptible agglutinins (especially with the more abundant group O bloods) are completely destroyed and the less susceptible agglutinogens (if present) may be inactivated to such degrees that no sound conclusions are possible from a comparison of the clumpings (with A and B-cells) of their sera and saline extracts with clumpings of the blank (non-stained material) extracts.

It has been our experience on blood group determination work carried out on very dirty

exhibits, such as clothing from labourers, old felt hats and songkoks, turbans, etc., that the added A and B cells break up during the 37°C. incubation period, especially at the higher concentrations of sera extracts and also in the saline extracts where no dilution is carried out during normal routine grouping. The possible causes for this phenomenon are:—

(a) attack by bacteria that were initially present in a dormant state but have been nourished and consequently activated during the period of sera extraction (approximately 16 hours), and

(b) contamination of sera or physiological saline extracts with the "salts" which are normally present in sweat[4] (sodium chloride, urea, lactic acid, etc.) thus changing the "salt" concentration in the saline which now is no longer isotonic with the red cell contents. The resulting hypertonic solution would cause the cells to shrink, while attack by the bacteria would help to lyse the cell wall, the cell contents oozing out into the suspending fluid.

RECOMMENDATIONS

In all offences of a criminal nature, the bloodstained weapons or instruments, the clothing of both the injured person and the perpetrator, etc., should immediately be taken into custody by the investigating officer. He should then ensure that all the bloodstains are dry and in the case of articles of clothing, he would be well advised to air them inside a well ventilated room for a period of at least three hours, before folding them for packing purposes. The dried bloodstained articles should then be forwarded to the Department of Chemistry, without unnecessary delay. Soon after being received it would be advisable for the Chemist concerned to examine the bloodstains for their origin, blood group, etc., as per request by the investigating officer.

In the opinion of one of us (S.S.) if the above suggestions are closely adhered to at each stage, then inconclusive results of analysis would be reduced to a bare minimum and consequently the possibility of guilt or innocence of a person held in connection with a crime more readily established.

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REFERENCES

1. Wall "Practical Blood Grouping Methods," Charles C Thomas, Springfield, Illinois, 1952. p.16.
2. Surinder Singh and Ow Yong Heng Khuan "Medical Journal of Malaya, 1961, XV, 222.
3. Schiff and Boyd "Blood Grouping Technic," Interscience Publishers, New York, 1942, p.71.
4. Best and Taylor "The Physiological Basis of Medical Practice" Bailliere, Tindall & Cox, London, 1950, p.723.
5. Roche Lynch. The Analyst, 1928, LIII 5.

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AN INVESTIGATION INTO THE COMMON CAUSES OF BLOODSTAIN DETERIORATION

PART II

Effect of Perspiration and Saliva on Blood Grouping

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In Part II[1] of this work it was noticed that even sweat from a person whose blood belonged to group O had an intermediate effect on the blood agglutinins. Since some sweat is invariably present on bloodstained clothing, it was felt that the effect should be further investigated.

In this investigation, the co-operation of eight staff members, two from each blood group, was sought. Each person was supplied with 4 clean filter papers which had previously been marked with his name and respective blood group. The individuals were told to dab their bodies twice each day, once when they got to office in the morning and once again when they returned to work after lunch. They were to use the same filter paper for four days, i.e., each paper had received eight dabbing sessions before it was put away and a second one used instead. Despite the numerous dabbings, some of the filter papers when returned were noticed to be relatively clean. This was due to the fact that some of those selected to supply the sweat apparently did not perspire. To ensure that the filter papers contained group specific substances before they were counterstained with blood, four persons out of the above eight, each belonging to a different blood group were made to spit into different beakers. The saliva was then used to stain clean filter papers.

When the sweat-stained and saliva-stained filter papers were ready, four samples of oxalated blood, each belonging to a different group were obtained from the Blood Bank at Penang. These bloods were employed to stain filter papers as indicated below:—

	Filter Papers	Nos. of Bloodstained Filter Papers
Group A Blood	Clean	1
	Group A sweat-stained	2 and 3
	Group B „ „	4 and 5
	Group O „ „	6 and 7
	Group AB „ „	8 and 9
	Group A Saliva-stained	10
	Group B „ „	11
	Group O „ „	12
	Group AB „ „	13

Therefore each sample of blood stained thirteen filter papers in all. These were dried in an air-conditioned room and then kept aside for a two-week period, after which their agglutinin and agglutinogen titres were determined as described and Part II[1] and the Grouping of Saliva[2]. The results obtained are given in Tables I to VII.

DISCUSSION

In this piece of work precautions similar to those described in Part II[1] of the work were taken so as to minimise irregularities, and thereby maintain consistency of results.

The suppliers of sweat and saliva have previously been shown to be secretors[2].

The results obtained are best discussed under the two sub-headings:—

(a) Saline Extract.

(b) Anti-sera Extract.

(a) **Saline Extract** (Tables I - III) — These results provide an indication of the agglutinin

concentrations in each stain. Since the sweat and saliva of secretors are known to contain group specific substances[3], we would expect these to remove all or at least in part the corresponding agglutinins from the bloodstains by agglutination. In cases where this takes place, the saline extracts of the bloodstains would show a titre less than that for the corresponding control (bloodstain No. 1).

The above hypothesis is well substantiated by the results obtained (Tables I – III). The more complete removal of agglutinins by saliva may be alluded to the saliva containing a higher concentration of agglutinogens than sweat.

Although sweats from group O individuals are free from group specific substances, yet they do reduce the titres of the blood agglutinins (Tables I – III). The adverse effect is most probably due to the deteriorative effects of sweat on agglutinins. Therefore sweat from secretors would have a two fold effect on bloods, viz., deteriorative and agglutinin removal. The latter effect would only take place if the blood contains an agglutinin capable of reacting with the group specific substance of the sweat.

(b) **Anti-sera Extracts** (Tables IV – VI) — Extraction of bloodstains with the corresponding anti-sera and then a study of its agglutination abilities (with known cells) at different dilutions provides a basis for a comparative study of the agglutinogen concentrations that were originally present in the bloodstains. As a typical example let us consider:—

(1) filter papers stained with the sweat of a group A secretor, and

(2) filter papers stained with the saliva of a secretor whose blood belonged to group A.

(1) **Sweat-stained Filter Papers** — If the sweat-stained filter paper was counter-stained with blood of group A (taken from a different person), then according to theory, the blood-stained area should contain an agglutinogen concentration higher than that which would be present in the bloodstain on a clean piece of filter paper. This would be indicated by a decrease, relative to control (stain 1) in the agglutinations of the anti-A sera extracts.

A similar sweat-stained paper when counter-stained with blood belonging to group B should not show any decrease in its agglutinations with known B cells, although the anti-A sera extract would exhibit reduced/increased agglutination (relative to control) depending upon the relative concentrations of agglutinogen A and blood agglutinin a present.

If, however, the sweat-stained paper was counter-stained with blood from a group AB individual, then the absorption of anti-A sera would increase resulting in a decrease of the extract agglutinations with known A cells. Agglutinations of the anti-B extract would correspond to those of the control.

Sweat from a group A secretor counter-stained with group O blood would only show changes in the saline extracts, unless the concentration of any group specific substances present is in excess of the corresponding blood agglutinin present.

(2) **Saliva** — The same sort of results would be expected from filter papers first stained with the saliva of a group A secretor and then counter-stained with blood.

From the agglutination results of sera extracts (Tables IV – VI) it will be noticed that those bloodstains containing saliva (Nos. 10 – 13) comply with the above theoretical requirements while the others (Nos. 2 – 9) containing sweat behave as though they were non-contaminated bloodstains (No. 1) or contaminated by substances that do not have agglutinin/agglutinogen reactions.

This apparent non compliance especially for sera extracts may be explained on the following basis:—

(a) That the bloodstain was on an area free of sweat.

(b) That the concentration of group specific substances in sweat is small and these in most cases have already been removed by agglutination at the time of counter-staining with blood.

Results — A summary of these cases where the results of grouping were inconclusive or the determined blood group differed from the group of the blood used to stain the filter papers is given in the following page:—

GROUP A BLOOD USED FOR STAINING

Stain No.	Contaminant	Sera Extract Result	Saline Extract Result
10	Saliva A	inconclusive	A
11	Saliva B	inconclusive	AB
13	Saliva AB	inconclusive	AB

GROUP B BLOOD USED FOR STAINING

Stain No.	Contaminant	Sera Extract Result	Saline Extract Result
2	Sweat A	B	AB
3	Sweat A	B	AB
8	Sweat AB	B	AB
10	Saliva A	inconclusive	AB
11	Saliva B	inconclusive	B
13	Saliva AB	inconclusive	AB

GROUP AB BLOOD USED FOR STAINING

Stain No.	Contaminant	Sera Extract Result
2	Sweat A	inconclusive
3	Sweat A	inconclusive
6	Sweat O	inconclusive
9	Sweat AB	inconclusive
10	Saliva A	inconclusive
11	Saliva B	inconclusive
12	Saliva O	inconclusive
13	Saliva AB	inconclusive

GROUP O BLOOD USED FOR STAINING

Stain No.	Contaminant	Saline Extract Result
3	Sweat A	A
5	Sweat B	B
8	Sweat AB	B
10	Saliva A	A
11	Saliva B	B
13	Saliva AB	AB

The results in the "blood group indicated" column (Tables IV - VI) were obtained by comparing the agglutinations of sera extracts from bloodstained areas with those of aera extracts from areas free of blood (Table VII). Where the difference in the agglutinations of the two corresponding sera extracts was greater than three places a definite group was reported. In all other cases the results were recorded as being inconclusive.

Grouping tests on saliva stains 10, 11 and 13 counter-stained with blood belonging to group A were in each case inconclusive. Stain number 10 and the corresponding blank showed a complete removal of agglutinin a and therefore, no conclusions were possible. The inconclusiveness in the case of stain number 11 was due to the saliva agglutinogen B being present in excess of the blood agglutinin b. This excess showed up in the anti-B sera extract while the deficient blood agglutinin b did not show up in the saline extract. From the blank for stain number 11, it may be inferred that the agglutinogen B in the saliva had a concentration either equal to or slightly greater than the concentration of the agglutinin b in the sera used for extractions. Again the case of stain Number 13, the B agglutinogen in the saliva of an AB secretor was

in excess of the blood agglutinin b and therefore, the latter did not show up in the saline extract. The B-cell agglutinations being in excess of A-cell agglutinations may be explained on the basis that at the time of stain extraction the B agglutinogen concentration was less than the A agglutinogen concentration, since the former agglutinogen had already been partly removed by the naturally occurring blood agglutinin b, while the saliva agglutinogen A, instead had a further addition from the blood.

With the blood of group B, the bloodstains containing sweat unlike those containing saliva, gave definite (clear cut) yet contradictory results for the sera and saline extracts. To account for these stain numbers 2 and 3 must have had a concentration of agglutinogen A (from sweat) just in excess of the concentration of the blood agglutinin a. This resulted in the complete removal (by agglutination) of the latter, hence its absence in the saline extract. The balance of the slight excess agglutinogen A did not exert any significant effect on the agglutinations of the anti-A sera extracts with known A cells. The results obtained for stain number 8 could be explained along the same lines.

In a similar manner, all the remaining anomalous results could be explained.

From the above one could therefore reasonably conclude that in the case of bloodstains contaminated with the sweats of secretors, the results of grouping tests obtained would depend upon the relative titres of the agglutinogens of the contaminating fluids and the corresponding blood agglutinins.

Invariably with saliva which has a higher concentration of group specific substances, the grouping tests tend to show the presence of the excess agglutinogen.

Since with group AB and O bloodstains only sera and saline extracts respectively were made and their agglutinations observed, it may be argued that the erroneous/inconclusive results with these two bloods may not have arisen if the sera and saline extracts had been studied in each of these cases, just as is usually done when grouping bloodstains of unkonwn group.

In the opinion of one of us (S.S.) this extra labour would not have yielded any results better than those already obtained, for the following reasons:—

Group AB bloodstains — The stains of this blood when contaminated with stains of agglutinogen containing fluids would only alter the agglutinogen concentration of the bloodstain and therefore the saline extracts would not be expected to show any signs of agglutination with known A and B cells.

Group O bloodstains — Sera extracts from these stains would either yield no decisive agglutination results if agglutinogen concentration in the contaminant is equal to the corresponding agglutinin concentration in the blood (unless the contamination is by fluid from a group AB secretor), or if the agglutinogen concentration is in excess of that of the corresponding agglutinin, then the sera extract would only help to confirm the wrong results already indicated by the saline extract agglutinations.

From the summary given on page 264 it is obvious that the contaminant — saliva — has a marked effect! and in almost all cases yields inconclusive results. It can, however, be responsible for erroneous results, if during grouping, the saliva contaminated bloodstain were extracted with sera and saline and for the blank instead an area free of saliva had been unintentionally selected. Then stains such as number 11 (Tables I and IV) and numbers 10 and 13 (Tables II and V) would confidently have been reported as belonging to group AB although they actually belonged to groups A and B respectively. Thus even with the method of double check (sera extract with saline extract) erroneous results are possible.

The forensic scientist should, therefore, be extra careful when examining bloodstained articles in which the probability of contamination by fluids (saliva and seminal fluid) containing a relatively high concentration of group specific substances is present. Such articles would include handkerchiefs, materials used for gagging purposes, bloodstained clothing in cases of rape, etc.

RESULTS OF GROUPING TESTS CARRIED OUT ON FILTER PAPERS STAINED WITH BLOOD OF GROUPS A, B & O STAINS EXTRACTED WITH PHYSIOLOGICAL SALINE

			TABLE I — GROUP A BLOODSTAINS									TABLE II — GROUP B BLOODSTAINS									TABLE III — GROUP O BLOODSTAINS								
Filter Paper Number	Blood Group of Secretor	Blood Cells	1	1/2	1/4	1/8	1/16	1/32	1/64	1/128	Blood Group Indicated	1	1/2	1/4	1/8	1/16	1/32	1/64	1/128	Blood Group Indicated	1	1/2	1/4	1/8	1/16	1/32	1/64	1/128	Blood Group Indicated
1	Nil	A	—	—	—	—	—	—	—	—	A	+	±	—	—	—	—	—	—	B	+	+	+	+	+	+	±	—	O
		B	+	±	+	+	+	±				—	—	—	—	—	—	—	—		+	+	+	+	—	—	—	—	
2	A	A	—	—	—	—	—	—	—	—	A	—	—	—	—	—	—	—	—	AB	+	—	±	—	—	—	—	—	O
		B	+	+	+	+	+	±				—	—	—	—	—	—	—	—		+	±							
3	A	A	—	—	—	—	—	—	—	—	A	—	—	—	—	—	—	—	—	AB	—	—	—	—	—	—	—	—	A
		B	+	+	+	+	+	+	—	—		—	—	—	—	—	—	—	—		+	—	—	—	—	—	—	—	
4	B	A	—	—	—	—	—	—	—	—	A	+	—	—	—	—	—	—	—	B	+	+	+	+	+	+	+	—	O
		B	—	+	—	—	—	—	—	—		—	—	—	—	—	—	—	—		+	—	—	—	—	—	—	—	
5	B	A	—	—	—	—	—	—	—	—	A	+	±	—	—	—	—	—	—	B	+	+	+	—	—	—	—	—	B
		B	+	±	—	—	—	—	—	—		—	—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	
6	O	A	—	—	—	—	—	—	—	—	A	+	—	—	—	—	—	—	—	B	+	+	+	—	—	—	—	—	O
		B	+	+	+	+	—	—	—	—		—	—	—	—	—	—	—	—		+	+	—	—	—	—	—	—	
7	O	A	—	—	—	—	—	—	—	—	A	+	—	—	—	—	—	—	—	B	+	+	+	+	+	+	+	—	O
		B	+	+	+	+	+	—	—	—		—	—	—	—	—	—	—	—		+	+	+	+	—	—	—	—	
8	AB	A	—	—	—	—	—	—	—	—	A	—	—	—	—	—	—	—	—	AB	+	±	—	—	—	—	—	—	B
		B	±	+	—	—	—	—	—	—		—	—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	
9	AB	A	—	—	—	—	—	—	—	—	A	+	—	—	—	—	—	—	—	B	+	+	+	+	+	—	—	—	O
		B	+	+	+	+	—	—	—	—		—	—	—	—	—	—	—	—		+	+	+	—	—	—	—	—	
10	A	A	—	—	—	—	—	—	—	—	A	—	—	—	—	—	—	—	—	AB	—	—	—	—	—	—	—	—	A
		B	+	+	+	+	+	+	—	—		—	—	—	—	—	—	—	—		+	+	+	±	—	—	—	—	
11	B	A	—	—	—	—	—	—	—	—	AB	+	—	—	—	—	—	—	—	B	+	+	+	+	+	—	—	—	B
		B	—	—	—	—	—	—	—	—		—	—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	
12	0	A	—	—	—	—	—	—	—	—	A	+	—	—	—	—	—	—	—	B	+	+	+	+	+	+	+	—	O
		B	+	+	+	—	+	—	—	—		—	—	—	—	—	—	—	—		+	+	+	+	—	—	—	—	
13	AB	A	—	—	—	—	—	—	—	—	AB	—	—	—	—	—	—	—	—	AB	—	—	—	—	—	—	—	—	AB
		B	—	—	—	—	—	—	—	—		—	—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	
Anti - A Anti - B	Nil	A	+	+	+	+	+	+	+	+		+	+	+	+	—	+	+	+		+	+	+	+	+	+	+	+	
		B	+	+	+	+	+	+	+	+		+	+	+	+	—	+	+	+		+	+	+	+	+	+	+	+	

\+ indicates agglutination.
± indicates beginnings of agglutination.
— indicates no agglutination.

RESULTS OF GROUPING TESTS CARRIED OUT ON FILTER PAPERS STAINED WITH BLOOD OF GROUPS A, B & AB AND ON THE BLANKS STAINS EXTRACTED WITH 1 IN 2 DILUTION OF ANTI-A AND ANTI-B SERA

TABLE IV											TABLE V										TABLE VI										TABLE VII								
			GROUP A BLOODSTAINS								GROUP B BLOODSTAINS										GROUP AB BLOODSTAINS										BLANKS								
Filter Paper Number	Blood Group of Secretor	Blood Cells	1/2	1/4	1/8	1/16	1/32	1/64	1/128	1/256	Blood Group Indicated	1/2	1/4	1/8	1/16	1/32	1/64	1/128	1/256	Blood Group Indicated	1/2	1/4	1/8	1/16	1/32	1/64	1/128	1/256	Blood Group Indicated	Sweat/Saliva Stained Area	1/2	1/4	1/8	1/16	1/32	1/64	1/128	1/256
1	Nil	A	+	−	−	−	−	−	−	−	A	+	+	+	+	+	+	+	+	B	+	−	−	+	−	−	−	−	AB	Nil	+	+	+	+	+	+	+	+
		B	+	−	−	−	−	+	+	+		+	−	−	−	−	−	−	−		+	+	−	+	−	−	−	−			+	+	+	+	+	+	+	+
2	A	A	+	+	−	−	−	−	−	−	A	+	+	+	−	−	−	−	−	B	+	−	−	−	−	−	−	−	Inconclusive	Sweat	+	+	+	+	+	+	−	−
		B	+	−	−	+	+	+	+	+		+	+	−	−	−	−	−	−		+	−	−	−	−	−	−	−			+	+	+	+	+	+	+	+
3	A	A	−	−	−	−	−	−	−	−	A	+	+	+	−	−	−	−	−	B	+	−	+	−	−	−	−	−	Inconclusive	Sweat	+	+	+	+	+	+	−	−
		B	+	−	−	+	+	+	+	+		+	+	−	−	−	−	−	−		+	−	−	−	−	−	−	−			+	+	+	+	+	+	+	+
4	B	A	+	+	−	−	−	−	−	−	A	+	−	−	−	−	−	−	−	B	+	+	+	−	−	−	−	−	AB	Sweat	+	+	+	+	+	+	+	+
		B	+	+	+	+	+	+	+	−		−	−	−	+	+	+	+	+		+	+	+	−	−	−	−	−			+	+	+	+	+	+	+	+
5	B	A	+	+	−	−	−	−	−	−	A	+	−	−	−	−	−	−	+	B	+	+	+	+	−	−	−	−	AB	Sweat	+	+	+	+	+	+	+	+
		B	+	+	+	+	+	+	+	+		−	−	−	−	−	−	−	−		+	+	+	+	−	−	−	−			+	+	+	+	+	+	+	+
6	O	A	+	+	−	−	−	−	−	−	A	−	−	−	−	−	−	−	−	B	+	+	+	+	+	−	−	−	Inconclusive	Sweat	+	+	+	+	+	+	+	+
		B	+	+	+	+	+	+	+	+		−	−	−	−	−	−	−	−		+	+	+	+	−	−	−	−			+	+	+	+	+	+	+	+
7	O	A	−	+	−	−	−	−	−	−	A	−	−	−	−	−	−	−	−	B	+	+	+	−	−	−	−	−	AB	Sweat	+	+	+	+	+	+	+	+
		B	+	+	+	+	+	+	+	+		−	−	+	−	−	−	−	−		+	+	+	−	−	−	−	−			+	+	+	+	+	+	+	+
8	AB	A	+	+	−	−	−	−	−	−	A	+	−	−	−	−	−	−	−	B	+	−	+	−	−	−	−	−	AB	Sweat	+	+	+	+	+	+	+	+
		B	+	+	+	+	+	+	+	+		+	+	−	−	−	−	−	−		+	+	+	−	−	−	−	−			+	+	+	+	+	+	+	+
9	AB	A	+	+	−	−	−	−	−	−	A	+	−	−	−	−	−	+	+	B	+	+	+	+	+	−	−	−	Inconclusive	Sweat	+	+	+	+	+	+	+	−
		B	+	+	+	+	+	+	+	+		−	−	−	−	−	−	−	−		+	+	+	+	+	−	−	−			+	+	+	+	+	+	+	+
10	A	A	−	−	−	−	−	−	−	−	Inconclusive	−	−	−	−	−	−	−	−	Inconclusive	−	−	−	−	−	−	−	−	Inconclusive	Saliva	−	−	−	−	−	−	−	−
		B	+	+	+	+	+	+	+	+		−	−	−	−	−	−	−	−		+	+	+	+	+	−	−	−			+	+	+	+	+	+	+	+
11	B	A	+	+	+	+	−	−	−	−	Inconclusive	+	+	+	+	+	+	−	−	Inconclusive	+	−	+	+	+	−	−	−	Inconclusive	Saliva	+	+	+	+	+	−	−	+
		B	+	−	−	−	−	−	−	−		−	−	−	−	−	−	−	−		−	−	−	−	−	−	−	−			−	−	−	−	−	−	−	−
12	O	A	+	+	+	−	−	−	−	−	A	+	+	+	+	+	−	−	−	B	+	−	+	+	+	−	−	−	Inconclusive	Saliva	+	+	+	+	+	+	+	−
		B	+	+	+	−	−	−	−	−		−	−	−	−	−	−	−	−		+	+	+	+	+	−	−	−			+	+	+	+	+	+	+	+
13	AB	A	−	−	−	−	−	−	−	−	Inconclusive	+	−	−	−	−	−	−	−	Inconclusive	−	−	−	−	−	−	−	−	Inconclusive	Saliva	−	−	−	−	−	−	−	−
		B	+	−	−	−	−	−	−	−		−	−	−	−	−	−	−	−		+	+	−	−	−	−	−	−			−	−	−	−	−	−	−	−

+ indicates agglutination.

± indicates beginnings of agglutination.

— indicates no agglutination.

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REFERENCES

1. Surinder Singh and Ow Yong Heng Khuan "An investigation into the common causes of bloodstain deterioration Part I" in the press.
2. Surinder Singh and Ow Yong Heng Khuan, Medical Journal of Malaya, 1961, XV, 222.
3. Alexander S. Wiener "Blood Groups and Blood Transfusion," George Banta Publishing Co., Menasha, Wiscosin 1935, p.148.

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SERUM IRON AND IRON BINDING CAPACITY IN MALAYANS.

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Studies on serum iron levels have been carried out in Malaya by Tasker (1955) in non anaemic patients and in nutritional anaemias. Serum iron has not been estimated in other types of anaemia and all studies carried out by Tasker applied to adults only. No work has been carried out in children and iron binding capacity has not been estimated in any condition.

The study described in this paper was done with the following purpose: (a) to establish not only the serum iron but also the serum iron binding capacity in normal healthy Malayans, (b) to study the serum iron and iron binding capacity in anaemias in children in order to evaluate the incidence of iron deficiency (c) to study the serum iron and iron binding capacity in Malayan new borns in order to see how in general, babies in this country start their life as regards the amount of iron in their blood. (d) to study the iron and iron binding capacity in other conditions, in order to check whether the results we obtained agree with those already reported by others.

METHODS AND MATERIALS

Methods. Since iron is such an ubiquitous element and its amount in serum so small, it became absolutely necessary that, in order to prevent extraneous contamination during the determination of serum iron and iron binding capacity all glassware should be entirely iron free. Therefore, all syringes and glassware were washed with either hot or boiling 5 N HCL, followed by a generous rinsing with all glass distilled water. After drying, such glassware were set aside only for iron determinations. Only all glass distilled water was used to rinse glassware and to make up reagent solutions.

Serum iron estimation was carried out by the method of Ramsay (1954), in which acetate-buffered-dipyridyl solution was added to serum followed by the reducing agent sodium sulphite. Proteins were removed by heating in a boiling water bath and centrifugation. The resultant pink ferrous-dipyridl complex was measured spectro-photometrically at 520 mu and the amount of serum iron was calculated and expressed as μg/100 ml.

Unsaturated iron binding capacity was performed by the method of Ressler and Zak (1958). The procedure involved a known amount of ferrous ions being added to a buffered solution of serum in order to saturate the binding capacity of the iron binding protein (siderophilin). The excess amount of ferrous ions that did not combine with the siderophilin was then determined photometrically after reacting the mixture with dipyridl. By subtracting this observed amount from the known amount of ferrous ions put in at the beginning of the experiment, the amount that had combined with the siderophilin may calculated. This constitutes the unsaturated iron-binding capacity and was expressed as μg/100 ml. (μg per cent). The total iron binding capacity is the sum of serum iron and unsaturated iron binding capacity.

Material. All blood samples were taken in the morning at about 10 a.m. to minimize the influence of diurnal variation (Heilmeyer and Plötner 1937, Hemmeler 1944, Höyer 1944, Waldenström 1946, Hamilton et al 1950, Paterson et al. 1952, Howard 1953, Antila 1962).

The material for study consisted of four different groups, a group of normal healthy individuals, a group of anaemic children, a group of newborns and their mothers, and a group of miscellaneous diseases.

The normal healthy non anaemic person were obtained from the blood bank. Only blood donors who came for the first time were considered for study. This group also consisted of laboratory workers and administrative personnel. They were all adults, ranging in age between 17 and 50 years.

The group of anaemic children were obtained from the paediatric wards of the General Hospital, Kuala Lumpur. They were of the middle and lower income groups and they ranged in age between 3 months to 7 years. These children were taken at random the only criterion being that the haemoglobin level was below 8 g %.

Newborns and their mothers were obtained from the labour room in the General Hospital. The mothers were in parturition and the blood was taken only in the morning from them. Blood specimens of the newborns were taken from the cord and were divided into two groups, those born in the morning and those born in the afternoon or evening.

RESULTS

A total of 324 persons have been examined for serum iron and serum iron binding capacity. The results in 136 normal healthy persons, 131 males and 5 females, are presented in table 1. The males are divided into six racial groups, 48 Chinese, 39 Malays, 29 Indians, 4 Sikhs, 6 Eurasians, and 5 Europeans. The number of females examined was too small for consideration. They are put into one group.

A total of 97 anaemic children were examined. Four of them were excluded because it was found that they were under iron treatment when the blood was obtained. The results in the remaining 93 are found in table 2. They are grouped according to haematological diagnosis. A group of miscellaneous diseases in these children consists of a number of diseases (too few of each for proper evaluation.)

The cord bloods of 32 newborns and peripheral blood of 36 mothers were studied. 26 of the mothers were examined before delivery, 10 after delivery. In 6 mothers the babies blood was not examined. Of 2 babies the mother was not examined. 24 of the babies were born in the morning, only 2 were born in the afternoon. In 6 the time of delivery was not recorded. The results are presented in table 3.

The findings in 23 miscellaneous diseases obtained from different wards of the General Hospital are not presented in a table because the number of each disease is considered too small to be of much importance.

DISCUSSION

Normal persons. In the three representative ethnic groups in Malaya, the Chinese, Malays, and Indians, the mean for serum iron was found to be respectively 123 ug per cent, 112 μg per cent and 114 μg per cent. These are within the normal range when compared with those reported by many other workers from different countries. In Europeans, the mean for serum iron in men reported in 37 papers from 8 different countries (compiled by Viljo Antilo 1962), ranged between 100 and 146 μg per cent. In Asia, Agarwal and Misra (1955) found a mean of 117 μg per cent for 50 men in India, and Sakakura found a mean of serum iron of 121 μg per cent in 20 male Japanese. In Malaya, Tasker (1955) examined patients with haemoglobin levels of higher than 14 g % and found a mean serum iron of 145 μg per cent. They were in fact not normal and healthy individuals, since they came to the hospital for one or other complaint.

Of the three representative groups in our series, the Chinese had the highest mean serum iron level, the Malays and Indians had about the same mean serum iron. There is some indication that the Europeans had the highest mean serum iron, while the Sikhs had the lowest of the whole series examined. However, the numbers of persons examined in these last mentioned groups were too small to warrant any conclusion.

As regards the total iron binding capacity, the values obtained by us, 318 μg per cent in Chinese, 325 μg per cent in Malays and 326 μg per cent in Indians are again within the normal range when compared with those reported in 12 papers from European countries, which ranged between 253 μg per cent and 348 μg per cent (Antila (1962).

Anaemic children. A total of 97 anaemic children with haemoglobin below 8 g % were examined. Four were excluded because they were on iron treatment when the blood was taken. The mean serum iron in the rest, a total of 93 children, was 76 μg per cent with a range of 10 μg per cent to 363 μg per cent.

TABLE I
Serum Iron and Total Iron Binding Capacity (TIBC)
in Normal Healthy Malayans

Race	Sex	No. exam.	Serum Iron µg%			TIBC µg%		
			Mean	Range	SD	Mean	Range	SD
Chinese	M	48	123	45-240	49.1	318	217-508	78.6
Malay	M	39	112	67-228	37.1	325	218-392	68.8
Indian	M	29	114	60-180	32.4	326	270-441	59.5
Sikh	M	4	89	40-120	37.3	410	293-502	105.1
Eurasian	M	6	128	94-202	45.7	341	258-484	88.9
European	M	5	132	64-177	46.3	299	233-344	45.4
Total	M	131	117	40-240	41.6	324	217-508	62.3
Total	F	5	105	75-160	32.4	334	284-368	36.4

TABLE II
Serum Iron and Total Iron Binding Capacity (TIBC)
in Anaemia Children

Haematological Classification	No. and Race	Serum iron µg%			T.IBC µg%		
		Mean	Range	S.D.	Mean	Range	S.D.
Iron def. anaemia	49 (M9 C10 I30)	40	10-131	24.0	364	250-509	74.2
Megalobl. anaemia	15 (M3 C1 I10 P1)	62	20-154	39.4	367	184-439	76.1
Haemolytic anaemia	9 (M2 C6 A1)	174	56-343	101.4	266	164-351	68.1
Malaria	6 (M4 C1 I1)	145	40-363	92.1	318	243-512	100.4
Leukaemia	3 (all C)	107	60-144	43.0	372	336-360	43.8
Miscellaneous	11 (M3 C7 I1)	125	31-471	129.5	298	144-516	133.5
Total	93	76	10-363	77.7	345	106-516	88.8

M = Malay
C = Chinese
I = Indian
P = Pakistani

TABLE III

Serum iron and Total Iron Binding Capacity (TIBC) in Newborns and their Mothers

	time blood taken	No. and Race	Serum iron μg%			TIBC μg%		
			Mean	Range	S.D.	Mean	Range	S.D.
Newborns	at birth in morning	24 (M1 C16 15 E2)	171	40-308	67.3	306	121-483	82.5
	at birth in afternoon and night	2 (I2)	165		—	274		45.3
	Unknown	6 (C3 13)	106	60-200	58.5	336	204-471	109.7
	Total	32 (M1 C19 I10 E)	158	40-308	67.6	310	121-483	85.5
Mothers	Before delivery in the morning	26 (M2 C18 I6)	96	28-210	53.2	452	339-566	58.2
	After delivery in the morning	10 (C7 I3)	64	20-140	38.6	368	149-495	101.2
	Total	36 (M2 C25 I9) 36	87	20-210	51.2	431	149-566	79.2

M = Malays
C = Chinese
I = Indians
E = European

49 of them had serum iron levels below 50 μg per cent. That is, more than half had very low serum iron levels. In the group classified as iron deficiency anaemia from haematological studies, the mean serum iron was 40 μg per cent and the mean total iron binding capacity 364 μg per cent. That the mean serum iron of the whole series is not as low as expected with such a large proportion of the children having a low serum iron level is due to the increase of serum iron in 15 cases of haemolytic anaemia, 9 congenital and 6 due to malaria. If these haemolytic cases were taken apart, the mean serum iron of the rest would have been 62 μg per cent and the total iron binding capacity 356 μg per cent, giving a low mean saturation of 17%. These fiindings clearly demonstrate the importance of iron deficiency in anaemias in children in this country. It could be argued, that many of the children admitted to the hospital must have had one or other infection accounting for a low serum iron level. However, it can be seen from the unsaturated iron binding capacity, that most of them had a high unsaturated iron binding capacity, except the group of haemolytic anaemias. Only 2 of those with low serum iron level had an unsaturated iron binding capacity below 200 μg per cent, while the mean unsaturated iron binding capacity from the whole non haemolytic group is 294 μg per cent. This indicates that the low serum iron in the majority in this group was indeed due to iron deficiency, since in infection, a low unsaturated iron binding capacity is usually found. We have also been able to further check it by clinical and haematological means, for instance by studying the haematological picture and response to iron treatment. From studies of the faeces for hookworms eggs in these children (Lie-Injo Luan Eng and Virik, Lie Kian Joe 1963) it has to be concluded that hookworm is of no importance in the causation of anaemia in children below 2 years in Kuala Lumpur and environment. In children between 2 years and 7 years, there may be some influence, but in general, it was considered not to be great. The major cause for iron deficiency must therefore be sought in nutrition. Whether this is due to actual deficiency of iron in the daily diet of the Malayan child or to the factor of poor absorption due to the presence of substances taken at the same time in their daily food, which precipitate the iron present in the diet, such as phytic acid, can not be said from this study. However, Thomson (1960) and Sedky (1962) found that the iron in the daily diet in Malayans in general, is below standard requirements.

The four cases who were found to be on iron treatment when the blood sample was taken, were excluded from the list in order not to obscure the findings. Two of these cases were found to have extremely high serum iron levels of 600 μg per cent and 450 μg per cent.

In anaemia with megaloblastic changes of the bone marrow, the mean serum iron was 62 μg per cent in 15 children examined. It must be mentioned however, that the megaloblastic changes were mostly of the intermediate type and that they were in addition to an iron deficiency state in 12 of the cases. The unsaturated binding capacity was also high in this series, indicating an iron deficiency state.

In haemolytic anaemias most of them congenital (6 thalassaemia major, 2 Hb E-thalassaemia and 1 auto-immune haemolytic anaemia) the serum iron was definitely increased with a low unsaturated iron binding capacity. The same can be said of the cases of malaria which is essentially haemolytic in nature. As can be seen from the range of serum iron values, not all of them showed a high serum iron level. This probably depended upon the haemolytic activity at the time the blood was obtained.

Newborns and their mothers. The mean serum iron in newborns was 158 μg% (see table 3) that is, higher than the mean in normal male adults. This is in agreement with those reported by others (Hagbergh 1953, Sturgeon 1954). It is also higher than the mean serum iron in the mothers in whom a mean serum iron level of 87 μg per cent was found. A proportion of the babies we examined, had a very low serum iron level at birth. No comparison can be made between the serum iron levels of babies born in the morning with those born in the afternoon since only two samples were from babies known to be born in the afternoon. The mean total iron binding capacity in our newborn babies was normal. In the mothers the mean serum iron was found to be lower than in normal males

Blood samples taken before delivery gave a higher mean serum level than in those taken after delivery. The total iron binding capacity was relatively high. The findings in this group are difficult to evaluate, since the factor of iron treatment is unreliable. Most of the mothers visited antenatal clinics and were given ferrous iron tablets prior to delivery, but very often this was not recorded. The mothers, when asked whether they had had iron treatment, usually did not know, since they did not know whether the tablets they received from the clinics or from their private doctors or which they bought from the dispensary at the advice of friends, did contain iron. It was because of these difficulties that we stopped studying this group further. The mode of transmission of iron from mother to her foetus is still obscure. Our number of estimations is too small to warrant conclusions. However, it would be interesting to study this problem further, in more detail and on the basis of more reliable data as regards iron treatment prior to delivery.

Miscellaneous diseases. A total of 23 patients with different diseases were examined. In general the results of our study in this group agreed with those reported by others. For instance in three cases of infectious hepatitis, two were found to have much increased serum iron level and the mean in this group was 176 ug per cent. In 5 cases of leukaemia the serum iron level was found to be within normal limits and the mean was 104 ug per cent. The mean total iron binding capacity was also normal. In two cases of autoimmune haemolytic anaemia and 2 cases of haemoglobinopathy in adults, the serum iron was much increased. The same can be said of cases of malaria in adults. The unsaturated iron binding capacity in these haemolytic conditions accordingly was relatively low. We did not find an increase of serum iron in 9 cases of thalassaemia trait carriers, but the number examined is too small included 3 females, 2 examined soon after delivery. The number of each disease examined in this miscellaneous group was too small to enable a proper evaluation of the findings.

Conclusion. Several useful data were obtained from this study. The normal values for serum iron and iron binding capacity in Malayan males were established and were found to be similar to those reported by other workers. The importance of iron deficiency in anaemias in children was clearly demonstrated and an idea as regards the serum iron and iron binding capacity at the start of life in new borns was obtained.

SUMMARY

A study of serum iron and iron binding capacity was carried out (a) in normal healthy Malayans, (b) in anaemic children admitted to the General Hospital, (c) in newborns and their mothers and (d) in a group of miscellaneous diseases.

The mean serum iron in the normal healthy males was as follows: 123 μg per cent in 48; Chinese, 112 μg per cent in 39 Malays, 114 μg per cent in 29 Indians 89 μg per cent in 4; Sikhs 128 μg per cent in Eurasians and 132, μg per cent in 5 Europeans. The total iron binding capacity in the same groups were as follows: 318 μg per cent in Chinese, 325 μg per cent in Malays, 326 μg per cent in Indians 410 μg per cent in Sikhs, 341 μg per cent in 6 Eurasians and 299 μg per cent in Europeans.

The findings in anaemic children clearly demonstrate the importance of iron deficiency in the causation of anaemia in children in Kuala Lumpur and environment, which was most probably due to a deficient diet. The mean serum iron in this group is 76 μg per cent with a range of 10 μg per cent to 363 μg per cent, 49 of these falling below 50 μg per cent. According to haematological classification 49 had iron deficiency anaemia, without megaloblasts in the bone marrow, 15 had megaloblastic changes of the bone marrow, 12 of whom in association with iron deficiency.

In congenital haemolytic anaemia and malaria in these children, the serum iron was high with a low unsaturated iron binding capacity.

In 32 newborns the serum iron was found to be generally high, higher than those of the mothers. A number of the newborns had a relatively very low serum iron level.

A group of 23 miscellaneous diseases gave results in general in agreement with those reported by others but the number of cases examined of each disease was too small to warrant conclusions.

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REFERENCES

Agarwal, S. C. and Misra, S. S. (1955). Indian J. Med. Res. **43**: 403.

Antila, V. Thesis, Helsinki 1962.

Hagbergh, B. (1953). Acta paediat. Supp. 9.

Hamilton, L. D., Gubler, C. J., Cartwright, G. E. and Winstrobe, M.M. (1950). Proc. Soc. exp. Biol. N. Y. **75**, 65.

Heilmeyer, L. and Plötner, K. Das Serum eisen. und die Eisenmangelkrankheit, Jena 1937.

Hemmeler, G. (1944). Helv. Med. Acta. **11**, 201.

Howard, R. B. (1953). J. Lab. Clin. Med. **42**, 816.

Höyer, K. (1944). Acta Med. Scand. **119**, 562.

Lie-Injo Luan Eng and Virik, H. K. will be published.

Lie Kian Joe, W.H.O. Expert Committee on Helminthiasis, August 1963. W.H.O./Helminth/30.

Paterson, C. J. S., Marrak, D. and Wiggins, H. S. (1952). Clin. Sci. **11**, 417.

Ramsay, W. N. M. (1954). Biochem. J. **57**, XVII.

Ressler, N. and Zak, B. (1958). Am. J. Chin Path. **30**, 87.

Sakakura, J. (1940). Tokyo Igakkwai, Zassi. **54**, 225.

Sedky, A. (1962). Rep. Series on Malayan Food Commodities. No. 1.

Sturgeon, P. (1954). Pediatrics **13**, 107.

Tasker, P. W. G. (1955). Trans. Roy. Soc. Trop. Med. and Hyg. **49**, 478.

Thomson, F. A. (1960). Bull. 10 Inst. Med. Res. Fed. Malaya.

Waldenström, J. Acta Med. Scan. 1946, **170**, 252.

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"REPORT ON THE OUTBREAK OF CHOLERA IN MALACCA, 1963 BY THE COMMITTEE OF ENQUIRY APPOINTED BY THE CABINET"

A CRITICAL REVIEW

By PROFESSOR A. A. SANDOSHAM

During May and June 1963 there was an epidemic outbreak of cholera in Malacca. The criticisms levelled at Government for the handling of the epidemic led to the setting up of a Committee of Enquiry whose report is now on sale (Government Printers, Kuala Lumpur. $2.50) It contains much material of great interest and practical importance to the medical profession but as Government Reports do not readily find their way to the shelves of medical men the Editor feels a brief review would be desirable.

Although early records are meagre, there is evidence that the earliest outbreaks of cholera in Malaya probably occured in Malacca in the sixteenth century. Cholera was recognized in the Durian Daun Hospital in Malacca in 1828-30, in Langkat village in Selangor in the late seventies, and in Pudu Goal, Kuala Lumpur in 1895. Significant outbreaks have been recorded in the present century, notably in 1910-11, 1914, 1918, 1927 and 1945-46.

The causative organism in the recent epidemic has been shown to be the El Tor type of cholera vibrio, which was first found among the Mecca pilgrims at El Tor (hence the name) in the Sinai Peninsula in 1905. Though identical in appearance and biochemical and other properties with the true cholera vibrio the Mecca pilgrims did not exhibit the usual clinical symptoms of cholera and the type was regarded as non-pathogenic. In 1937, however, a type of cholera vibrio similar to the El Tor variety was responsible for an outbreak in a village in Celebes in which the worst cases developed collapse and died in a few hours. However, many cases were relatively mild and the tendency to epidemic spread was not so great as in true cholera. An outbreak of El Tor cholera occured in Singapore in 1943 and there was a major diffusion by the seaways of South-East Asia and the Western Pacific in 1961-63. Professor H. B. Maitland of the Institute for Medical Research found the Malacca strains were different from the typical Vibrio El Tor in that they did not haemolyse the red blood cells of the sheep and goat, resembling in this respect the West New Guinea strains. The speculation is that the infection was conveyed to Malacca by sea routes from W. New Guinea through Java and Sumatra. The occurrence of cholera was recognized in Sumatra in 1962 and it is conceivable that the organism may have crossed the narrow Straits of Malacca through the many small craft plying between Malacca and the Sumatran coast without medical inspection of the crew.

The epidemic of cholera in Malacca appeared towards the close of a severe drought when the monthly rainfall dropped to between 1 and 2 inches instead of 7 and 14. Cholera is essentially a water-borne infection. About three-quarters of the Malacca town's water supply comes from the lower reaches of the Malacca river, the Bukit Sebukor supply. When the intake was placed some seven miles upstream it was thought to be beyond the reach of the tides but during the 1963 drought the level of water in the river fell to an unprecedented low level and at high tides sea water was carried upstream beyond the intake. This brackish water was highly polluted by (1) the effluents of the septic tanks of the Malacca General Hospital entering the river about one mile below, (2) the washing of sanitary buckets of the Municipality in the river about five miles below the intake and (3) the sewage from numerous riverside latrines entering directly into the river. Such contaminated water was passing into the Bukit Sebukor filtration plant during the weeks preceeding the epidemic. There are indications that chlorination may have failed for several hours during critical periods in the early stages of the epidemic and there were defects in the filtration process. Thus, there appears to be some circumstantial evidence to implicate the Bukit Sebukor water supply in the epidemic. There are 626 wells within the municipal area of Malacca but 599 of them are private wells mostly within houses. Although there was ample opportunity for pollution the grouping of cases does not appear to incriminate any of the communal wells.

The Enquiry brought to light other anomalies in the public health methods of the

town. It is pointed out that the main method of sewage disposal in Malacca town and suburbs is still the "bucket system". The latrine buckets are emptied daily and the nightsoil collected for disposal by trenching outside the town. The method, primitive at the best of times, is seen at its near worst in the back-to-back houses of the older parts of the town where the difficulties of maintaining reasonable hygienic standards are well nigh insuperable.

On the subject of measures taken (or not taken) to deal with the epidemic the Committee of Enquiry points out that cholera caused by Vibrio El Tor may be very severe or relatively mild. The less severe cases may be confused clinically with gastro-enteritis, food poisoning or bacilliary dysentery, all of which are commonly seen in Malayan hospitals throughout the year. The initial diagnosis may hence be hesitant especially in a non-endemic area like Malacca where cholera has not been reported for the past 133 years. During the last few days of April 1963 the doctors of Malacca were seeing patients who had a severe type of diarrhoea. On the morning of May 4 the condition of two of the patients led the physician to suspect the possibility of cholera and request a bacteriological confirmation. The laboratory in Malacca was not geared for cholera work and suitable culture media for the primary isolation of the vibrio were not immediately available. The final isolation was complete on the morning of May 9 in Malacca and confirmed by the Institute for Medical Research, Kuala Lumpur the following morning. Despite the difficulties and deficiencies the response of the Medical Unit was prompt and efficient.

Plans for cholera control were prepared by the Ministry of Health in 1961 when the presence of the El Tor cholera was first reported in the Malaysian region. One of the recommendations was that "every outbreak of gastro-enteritis as reported in the newspapers should be investigated by the health officer of the area". Malacca had no Health Officer and it was not till May 11 when a Health Officer had arrived from Kuala Lumpur that epidemiological investigations began and control measures were instituted. The decline of the epidemic began six days after the start of these control measures. However, mass inoculations only began on May 15 on receipt of vaccine supplies from abroad. It had been decided in 1961 that 900,000 doses should be ready for immediate use and stocked at the Institute for Medical Research. The vaccine stock available, however, was 217,000 ml.: 21,850 ml. being bottled and ready for immediate use; the rest held in bulk concentrate available only after it had been diluted, bottled and tested, a procedure which takes several days. This would have been sufficient to commence mass inoculation by giving the intradermal dosage of 0.2 ml. As there was some doubt about the effectiveness of a single intradermal inoculation and the amount available was too small to start a mass campaign giving 1 ml. doses subcutaneously there was a delay. This delay in commencing mass inoculation caused considerable anxiety among the population. The Committee maintains that it is no part of its task to assess the value of cholera inoculation in the control of the Malacca epidemic, nor does it consider itself competent to do so. The Committee has been able, however, to discuss the question with experts on El Tor cholera whose view it is that an undue emphasis is placed on the role of cholera inoculation in an epidemic situation. The primary drive in cholera control must always be directed to the water supplies, to sanitation and environmental hygiene, and to the discovery and elimination of foci of infection.

The most important part of the Report is the section on "Recommendations to prevent a recurrence of cholera" and the Committee has very wisely taken a broad approach, extending its recommendations to a wider geographical field than Malacca and reviewing the problem of interepidemic planning for the management of a possible recurrence.

In regard to water supplies the Committee recommends:

> that the Malacca supply should conform with internationally recognised bacteriological and chemical standards or with standards approved by the Ministries of Health and of Public Works; and
>
> that the Ministries of Health and Public Works should consider how best these standards might be made general throughout the country;
>
> that the development and control of the water supplies of Malacca State should be vested in single authority;
>
> that the Malacca Municipal supplies should be placed in the meantime under the technical surveillance of the Public Works Department; and
>
> that the bye-laws on water supply, sitting of meters, storage tanks, water con-

nections, etc., in force in the areas supplied by the Public Works Department be made immediately applicable to the Malacca Municipality.

The Committee views that the water supplies of the Federation should be controlled and developed as a public utility by State Water Boards co-ordinated and advised by a Federal Authority in Kuala Lumpur but recognising the constitutional and financial implications recommends that Government should examine or re-examine the case for a future development on these lines.

The Committee, recognising the potential contribution of wells to the epidemic, but realising that tradition dies hard, that old Malacca residents often prefer well water to the piped supply, and that radical measures to close the wells would be premature and socially unacceptable recommends:

that the Health Authorities should institute a regular inspection of all wells, condemning by closing down those which are open to gross pollution and taking such steps as may be practical to improve the rest;

that well-owners should be instructed by pamphlet or precept in the methods of sterilising their well water;

that all wells should be regularly chlorinated by the health authorities during periods of drought;

that a bacteriological examination of the water for cholera should be made whenever resources permit and the wells appear to be implicated in the spread of any acute gastro-intestinal infection; and

that where appropriate the necessary legal powers to enforce the above should be provided under the various health laws.

In regard to the Health Services of Malacca the Committee recommends:

that at least one of the vacant posts in the Malacca Health Department should be filled as soon as a qualified health officer is available;

that, as an interim measure in the absence of a suitably qualified health officer, a doctor should be diverted from the hospital branch to public health; and

that the Ministry should review the case for the restoration of the post of Senior Health Officer, Malacca, by up-grading one of the two posts for Health Officers;

that the Municipal Authorities should review the working of the "bucket" system of sanitation in relation to the irregularities disclosed;

that the Ministries of Health, of Interior and Public Works should lay down minimum standards for constructing public sewage disposal schemes and for their day-to-day maintenance;

that a modern system of sewage disposal should stand high in the Municipal plans for future development;

that back-lanes should be constructed to divide the back-to-back "long-houses" as is enforced in other Town Councils and Municipal areas;

that the Government should introduce legislation to control the development of villages, towns and cities in Malaysia according to modern town planning concepts, which should be binding at all levels of administration and from which no deviation should be allowed if such action would lead to infringement of health rules or cause a hazard to the health of the community. (Malaysia being a young country early action along these lines will pay handsome dividends in health for the future, without causing much hardship to the present generation. The haphazard development of Malacca without the aid of modern town planning should be a lesson for the rest of Malaysia, especially for local authorities.); and

that on an outbreak of cholera on an epidemic scale, all the resources of the Health Departments of Local Authorities should come under the control of the Ministry of Health and that provisions to this effect should be incorporated in future Federal health legislation;

that the sewage purification system be re-examined by the Public Works Department and that any defects discovered be corrected;

that the Ministry of Health should review the existing balance between the preventive and curative branches of the Medical Department and investigate the causes of the shortage of qualified health officers;

that the Ministry should review the possible means of strengthening the health services;

(i) by encouraging medical officers to take the Diploma of Public Health;

(ii) by restoring payment in relation to present-day values, of an allowance formerly made to time-scale health officers holding the Diploma of Public Health;

(iii) by according to the Diploma of Public Health the same status as other higher qualifications in the selection to superscale posts;

(iv) by reserving all unfilled super-scale posts in the Health Branch for health officers holding the Diploma of Public Health;

(v) by creating more senior health posts, i.e., the posts of Senior Medical Officers of Health be raised from four to ten in Malaya;

(vi) by recruiting qualified health officers on contract;

that the Ministry should consider whether in the absence of qualified health officers, medical officers should be temporarily diverted to public health in States where the need is pressing; and

that an Epidemiological and Medical Statistics Section be established in the Ministry of Health with properly qualified staff appointed to reorganise at all levels the certification and collection of medical and health statistics.

In regard to Inter-epidemic planning the Committee recommends that the

Ministry of Health be advised to revise the 1961 detailed plans of action in the light of experience from the Malacca outbreak. Such plans should, as hitherto, be circulated to staff at all levels of the administration and should include specific instructions to every category of officers on all measures necessary to help direct and conduct a campaign against cholera;

that the Ministry of Health should re-assess in the light of experience gained in the Malacca outbreak, the quantity of cholera vaccine to be held in the country for immediate use;

that in an epidemic situation cholera vaccine should be supplied by the Ministry of Health free of charge and without delay to all medical authorities within the epidemic area who are competent to use it; and

that stocks of cholera vaccine should always be computed in millilitres (ml.).

The Committee, considering the possibility that cholera may recur in this country and especially in Malacca, recommends

that the Ministry of Health should advise State Medical and Health Authorities on the timing and regional scope of any re-inoculation campaign the Ministry may consider necessary; and

that a Health Officer in the Ministry should be designated to review this for action from time to time.

The Committee, having reviewed the difficulties of the Malacca Pathological Laboratory in the early period of the epidemic and the heavy pressure under which the laboratory was working recommends

that a public health laboratory should be established in each major State;

that the Ministry of Health should review the possibility of co-ordination and standardisation of methods in the laboratories of the country able to undertake the diagnosis of cholera;

that the Ministry of Health should consider the possible formation of a mobile cholera investigation unit free from the responsibilities of control, equipped with a travelling laboratory, able to move to any suspected area at short notice and charged with the special — if not necessarily the only — task of defining the source and spread of infection and of giving technical support to the control authorities; and

that the Ministry should consider the case for a request to the World Health Organisation for an expert cholera team, the team to be posted to Malaya during the period of El Tor endemicity for the purpose of training Malaysian personnel and of supporting the local control authorities.

In view of the difficulties experienced in the Malacca Hospital in regard to beds, mattresses, etc. recommends

that the Ministry of Health should review the stocks of drugs and equipment to be kept in State medical stores in the light of experience during the Malacca epidemic.

The Committee, having reviewed the problems involved, and the criticisms of, the appli-

cation of quarantine in the Malacca cholera outbreak, and the heavy pressure on the quarantined wards in the Malacca General Hospital, recommends

> that State Medical and Health Authorities should receive ministerial guidance on the timing, scope, legislative procedure and application of quarantine restrictions;
>
> that the Malaysian Government should review the possibilities of shortening the delays between the decision to impose quarantine restrictions and the receipt of the relevant Gazette Notifications by the authorities concerned;
>
> that consideration be given to bring about a National Public Health Act in which is consolidated all the existing Federal, State and subsidiary legislation, thus satisfying, inter alia, the whole legal process pertaining to quarantine;
>
> that the Ministry of Health should re-consider the case for the construction of isolation hospitals for infectious diseases in Malacca, Selangor, Penang and other towns where there are international air and/or sea ports:
>
> that the health inspection of small craft calling at all ports, big and small, be strictly enforced with the co-operation of the Customs, Marine and Immigration Departments. These four departments should be represented on the Port Commission or Port Committees of every port in Malaysia, if the port health work is to be efficiently carried out. The Ministry of Health should ensure that health officers supervise the ports in their Districts; and
>
> that the Ministry of Health should consider the establishment of quarantine anchorage at ports handling international traffic and trade.

On the subject of Coordination of effort the Committee has this to say: "An epidemic of cholera is a social emergency, to be met with all the resources of Government. The Ministry of Health directs the effort and bears the main responsibility: other ministries and departments, and the people themselves, have a lesser but important role. The impact of control measures will be maximal when all agencies are kept well-informed on the situation, when they know what is expected of them, when their efforts are co-ordinated. Quick decisions have to be made, difficulties to be speedily resolved. The Committee has ample evidence of the energy with which the Malacca epidemics was tackled and of the will of the Municipal Authorities, the Police, the medical profession and other bodies to co-operate. It is less assured that these bodies were always clearly informed or their efforts well co-ordinated. Meetings were called, but not with the frequency or regularity which an epidemic situation demands." The Committee therefore recommends:

> that the Chief Medical and Health Officer of any State declared to be cholera-infected should convene without delay a State Co-ordinating Cholera Control Committee representing all Government departments, the local authority, voluntary organisations and other bodies involved, the Committee to meet daily during the epidemic to review events, to smooth out difficulties, and to co-ordinate control activities.

The Committee expresses the hope that its recommendations and sugegstions, if seriously considered and implemented, will contribute to the prevention of further epidemics and help to ensure that the country is fully prepared should cholera again appear in epidemic form.

Government statement on the report shows that the suggestions will indeed be considered seriously. Government has assured that departmental enquiries will be held as a matter of priority into the irregularities referred to in the report and "appropriate action against the officers concerned will, where necessary, be taken." The Government statement further shows that it regards most of the recommendations as sound and accepts them and that in fact some are already under implementation. This is most reassuring and the medical profession as well as the public owe a debt of gratitude to the excellent work of the Committee of Enquiry under the Chairmanship of Senator Khaw Kai Boh. In addition to Mr. Ng Chong Chee, the Committee included two distinguished medical men, namely Dr. John W. Field, a former Director of the Institute for Medical Research and Dr. Haji Megat Khas, a private practitioner.

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PRESIDENT'S SPEECH, MALAYAN MEDICAL ASSOCIATION at the Annual Dinner & Dance on 28.3.64

Dato Dr. R. SATHIAH

It is my pleasure and privilege on being installed as the President of the M.M.A., to welcome on behalf of the Council and members Their Highnesses the Sultan and Tengku Ampuan of Selangor, The Hon'ble the Deputy Prime Minister Tun Abdul Razak Al-Haj and Toh Puan Raha, the Hon'ble the Menteri Besar of Selangor Dato' Abu Bakar bin Baginda and Datin Zeharani, who have found time to join us this evening in spite of their multifarious duties.

I also have the pleasure of voicing on behalf of the members of our Association our appreciation and sincere congratulations to Dr. Abdullah and the members of his Council for a year of considerable activity and progress. If the incoming Council can emulate them in a small measure, we shall have cause to be satisfied.

This might be a suitable opportunity to disabuse the mind of the public of some of the misconceptions that seem to have found currency regarding the objects and policy of the M.M.A. We are not a Trade Union fighting for the rights of Government doctors or private medical practitioners. (*We are a professional body of medical men trying to maintain the high ideals of our noble calling and to serve the public.*) We try to do this in many ways. We organize frequent clinical meetings and talks on medical subjects, especially by distinguished specialists passing through this country to help bring our doctors up-to-date; we publish a medical journal encouraging doctors to investigate their cases thoroughly and write up their findings for the benefit of others. We have an Ethical Committee which tries to maintain a code of professional behaviour in keeping with the ideals of a noble profession.

We are aware of the shortage of doctors in the country and the difficulty the Government is experiencing in staffing the rural areas. We wish to assure the Government that we are most willing to co-operate with the Ministry of Health so far as an Association like ours can do. We cannot of course, force our members to work for the Government or set up practice in this place or that according to the needs of the country any more than Associations of Engineers or Lawyers can do. *Members have their individual right of choice, and in a democratic country the freedom of the individual has to be respected.* We can proudly say our country believes strongly in the freedom of the individual.

It has been hinted in the press that we are opposed to the recruitment of doctors from abroad. That is far from the truth. Of course, we would like to be assured, like any right thinking persons, that the doctors brought in have the necessary basic qualifications and do not lower the standards of medical practice in this country. Some members of the public and press, and I am sorry to say, it includes even a medically qualified person at the Ministerial level, are under the misapprehension that the Malayan Medical Association decides who is adequately qualified and who is not, and that our Association is preventing many doctors from entering the country. This is not true. To decide who is worthy of being placed on the Medical Register is the responsibility of a statutory body, the Medical Council of the country in whose deliberations the M.M.A. has no direct say. And yet, because an influx of doctors may affect the medical practice of some of our members, some of the uninitiated have jumped to the conclusion that our Association must of necessity be opposed to inviting Foreign doctors. The Association has been aware of the growing needs of a rapidly expanding population for properly qualified men and has long agitated for the establishment of a Medical School in Kuala Lumpur. We have also sought to gain public opinion in favour of providing better working conditions for our doctors in hospitals and rural clinics to prevent the mass exodus of men from the Public Services. At last, we are happy to find an enlightened Government which is beginning to implement some of our recommendations. It is indeed

a pleasure to see the Faculty of Medicine taking concrete shape in the Pantai Valley. The new proposals for salary structure for doctors and increase in superscale posts should go some way towards challenging the unpopularity of the Government service.

However, the M.M.A. feels strongly that unless the State and Local governments take on some of the administrative and financial responsibilities for the Health Services of the nation, there cannot be provided sufficient health coverage for every person in the country. The State Governments spend very little on the Health Services although under the Constitution they are jointly responsible with the Federal Government for these services. Despite the fact that the central government is spending record sums on the Health Services the people's needs are still not fully accommodated. The Minister of Health, in laying down annual priorities for the achievement of schemes should mobilise the State and Local governments to come to his aid. The enormity of the task of providing satisfactory Health Services cannot afford to be underestimated and success would be that much closer with the right kind of co-operation and support from the regional authorities.

Ladies and Gentlemen, it has been said that an after dinner speech should resemble a lady's dress, it should be short enough to be interesting and long enough to cover all the important points! I think I have covered these, which are to congratulate my predecessor, to assure you that the M.M.A. exists to help its members professionally and be of service to the public to which end it will co-operate whole-heartedly with the Government, and last but not least to thank you one and all, especially Their Highnesses and the distinguished guests who have graced the function this evening.

THE MEDICAL COUNCIL OF SINGAPORE
THE PENAL CASES COMMITTEE

The Penal Cases Committee of the Council on the 17th January and 28th March, 1963, inquired into four complaints of issuing sick certificates by four private medical practitioners in Singapore. Mr. Yeoh Ghim Seng, B.B.M., President of the Council, was in the Chair.

The four private medical practitioners concerned were as follows:—

(a) Dr. Elapulli Anantanarayana SHANKAR,
Shankar Dispensary,
351 Serangoon Road,
Singapore 8.

(b) Dr. Anent KULKARNI,
Kulkarni Dispensary,
367 Upper East Coast Road,
Singapore 15.

(c) Dr. Mohamed GAUS Mahyudin,
City Dispensary & Store,
132 Serangoon Road,
Singapore 8.

(d) Dr. Jack FLINTER,
Ong Dispensary,
306 Joo Chiat Road,
Singapore 15.

The determinations of the Committee were as follows:—

(i) Inquiries be held by the Medical Council of Singapore in accordance with regulation 15(c) of the Medical Registration Regulations, 1955 in respect of Doctors E. A. Shankar, A. Kulkarni and M. Gaus.

(ii) A letter be sent to Dr. J. Flinter, informing him that the charge against him in respect of issuing a false medical certificate had been withdrawn. However, he should be warned that by allowing the dispenser to handle the signed blank certificates, he had abused the privilege of the medical profession and that such practice should be discontinued forthwith. The Council accepted the decision of the Committee and directed the Registrar to send a letter to Dr. J. Flinter accordingly.

INQUIRIES OF THE MEDICAL COUNCIL OF SINGAPORE

The Council held three inquiries on the 5th September and 17th October, 1963. Mr. Yeoh Ghim Seng, B.B.M., President of the Council, was in the Chair. Mr. H. L. Wee of Messrs. C. J. Koh & Co. and Mr. N. N. Leicester of Messrs. Leicester & Chen were appointed as Legal Assessor and Solicitor to the Council respectively.

Inquiry No. 1 — Dr. Elapulli Anantanarayana SHANKAR

Dr. E. A. Shankar was charged as follows:—

> "That you on the 29th day of October, 1962, at about 9.50 a.m., at Shankar Dispensary, 351 Serangoon Road, Singapore, did issue to one Wong Chai Fook of 15-C Tanglin Halt Road, Singapore, a medical certificate stating he was suffering from diarrhœa and required rest for one day which certificate you knew to be false, and that in relation to the facts alleged you have been guilty of infamous conduct in a professional respect."

The President, after the Council's deliberation in camera, announced that Dr. Shankar was found guilty of the offence charged but that in view of the circumstances then, he directed that a letter of warning be sent to Dr. Shankar.

Dr. E. A. Shankar was present and was represented by Mr. M. Karthigesu, Solicitor.

Inquiry No. 2 — Dr. Anent KULKARNI

Dr. A. Kulkarni was charged as follows:—

> "That you on the 6th day of November, 1962, at about 9.10 a.m., at Kulkarni Dispensary, 367 Upper East Coast Road, Singapore, did issue to one R. Bupendra of 794-9 Pasir Panjang Road,

Singapore, a certificate stating he was under treatment for influenza and was unfit for work for two days, which certificate you knew to be false, and that in relation to the facts alleged you have been guilty of infamous conduct in a professional respect."

The Council having deliberated in camera, the President announced that by reason of the conviction which had been proved against him, the Council had directed the Registrar to erase Dr. Anent Kulkarni's name from the Register.

Dr. A. Kulkarni was present and represented by Mr. L. A. J. Smith, Solicitor.

Inquiry No. 3 — Dr. Mohamed GAUS Mahyudin

Dr. M. Gaus was charged as follows:—

"That you on the 5th day of November, 1962, at about 8.40 a.m., at City Dispensary and Store, 132 Serangoon Road, Singapore, did issue to one R. Bupendra of 794-9 Pasir Panjang Road, Singapore, a certificate stating that he was suffering from influenza and recommended 2 days sick leave with effect from 5.11.62 which certificate you knew to be false, and that in relation to the facts alleged you have been guilty of infamous conduct in a professional respects."

The Council, after hearing and considering the evidence, determined that Dr. M. Gaus was not guilty as charged. However, Dr. M. Gaus was given an admonition through its President to advise and warn him not to continue with the practice of leaving behind signed blank medical certificates.

Dr. M. Gaus was present and was represented by Dato Syed Esa Almenoar, Solicitor.

BOOK REVIEW

Medical Embryology by Jan Langman, M.D.Ph.D., published by Bailliere, Tindall & Cox, 1963, pp 335, 70s.

Professor of Anatomy, McGill University

The author states that his purpose in publishing this book was to present a concise account of human embryologic development which would incorporate recent advances in the field and correlate this knowledge with the clinical sciences. His book is intended primarily for medical students. The first part is devoted to general development and the second half to the embryologic development of each system. There is an interesting discussion on the etiologies of congenital malformations which includes some of the recent knowledge of the chromosomes and their abnormalities. Each of the chapters which concern the development of the various systems incorporates some information pertaining to the more important and frequently encountered congenital malformations.

The information presented is limited almost entirely to descriptive anatomy. There is practically no mention of theoretical aspects of embryology nor is any historical perspective presented. In the few places in the book where mention is made of etiologic factors of congenital malformations the statements are so general and brief as to be of almost no value to the student. A bibliography of varying length is appended to each chapter. This feature is often neglected in our textbooks for medical students in the basic sciences. However, most of the articles listed are 20 to 30 years old and pertain to aspects of embryology which are only casually mentioned in the text. The book lacks a glossary which would be of great help. However, a number of the definitions in the text are incorrect or so brief and vague that they are misleading. Though there are a number of line drawings throughout the book which are well conceived a number of the structures briefly described in the text are not accompanied by illustrations.

The book is very readable but compares in depth with publications by various drug houses. The discussions are too general and too brief. The book might be of value for a busy practising physician and surgeon or even the medical student as a summary and a review of the subject but I do not think that it would serve the purposes of a basic textbook for medical students or a good reference work for doctors.

RAY SELBY, M.D.

THE MICROSCOPICAL DIAGNOSIS OF HUMAN MALARIA (PART I) 2nd EDITION.

(Studies from the Institute for Medical Research, Federation of Malaya No. 30).
FIELD J. W., SANDOSHAM, A. A. and YAP LONG FONG (1963).
The Economy Printers Ltd., Kuala Lumpur. Price $12.

The second edition of what must be regarded as a standard reference book in this aspect of malarial work will be welcomed not only by malariologists but by those clinical pathologists and physicians who have an interest in this field of medicine.

Although the first edition was published shortly after the discovery of the exo-erythrocytic phase in human malaria and many new techniques have been evolved since then, for all practical purposes the positive diagnosis of the infection depends on the finding and identification of the parasites in blood films.

Dr. Field and Prof. Sandosham have admirably condensed their wide experience of this subject in the new volume and, in addition, have increased the scope to include Plasmodium ovale.

The clinical aspects have not been neglected and the descriptive chapters at the beginning of the book together with the clinical notes on appropriate colour plates will be appreciated.

Yap Long Fong, who was responsible for the illustrations in the first edition, has produced a new set of illustrations for this edition of his usual high standard. In addition the volume has been enhanced by the inclusion of a series of colour plates.

The authors are to be congratulated in producing a study in the best traditions of the Institute for Medical Research, Kuala Lumpur.

OBITUARY

JOHN WILLIAM SCHARFF.

B.A., M.B., B.A.O., B.Ch., D.P.H., M.D., D.T.M.&H. (London).

Dr J. W. Scharff, former Chief Health Officer, Singapore, died in London on 21st March, 1964, after an illness of 3 months. With his death Malaya has lost one of her greatest malariologists as well as one of her most outstanding health officers, who, more than any other anti-malarial worker, after Sir Malcolm Watson, was responsible for the country's freedom from malaria.

Born in Dublin in May, 1895, he was the son of Dr. R. F. Scharff, B.Sc., Ph.D., F.Z.S., Curator of Dublin Museum, and Alice Hutton After his early schooling in County Wicklow, he graduated in biology in 1912 and, a year later, entered Trinity College, Dublin. His medical studies were, however, interrupted by the 1914–18 War when he served as Surgeon Probationer from 1915–17 in the Royal Naval Reserve and took part in the famous Battle of Jutland. Graduating B.A., M.B., B.A.O., B.Ch., in 1918, he became Assistant Pathology lecturer in T.C.D., and in July of the following year he qualified D.P.H., M.D., D.T.M.&H. (London); a month later, he came to Malaya to work in a rubber estate in Malacca as assistant to Dr. Malcolm Rattery.

In 1920, he entered the Colonial Medical Service as Asst. Health Officer to Dr. Gilbert Brooke under whom he served for 5 years until 1925, when he returned home to marry Kathleen Esther Burn, the daughter of Sir Joseph and Lady Burn. From then on until the fall of Singapore in February, 1942, Dr. Scharff devoted himself to the eradication of malaria and the improvement of health in the states to which he was posted, viz. Malacca, Trengganu, Penang and Singapore where, in addition to his substantive post as C.H.O. of the whole island, he lectured in Public Health in the K.E. VII College of Medicine. During the thirties he organised anti-malarial courses under the auspices of the League of Nations, which became so internationally famous that Health Officers and Malariologists from all parts of the world came to attend. To keep abreast of the latest developments in malarial prevention and public health, he travelled extensively to various parts of the United States, Europe and the Far East, and he also wrote on many aspects of his field of study and made many original contributions to medical and health journals.

During 1942 — 1945, Dr. Scharff served in the R.A.M.C. with the rank of colonel, returning to Malaya in 1945 as Malaria Adviser to the Army. In 1946 he retired from the Colonial Medical Service and returned to the United Kingdom where he worked on Soil Fertility until 1959 when he made his second return to Malaya to serve as Medical Officer of Health in Cameron Highland which post he relinquished in early 1962.

Completely dedicated to his work, Dr. Scharff was, par excellence, the ideal Health Officer who did not spare himself in order to achieve his main object — the betterment of people's health; and this he achieved aided by his boundless energy, unflagging enthusiasm and complete understanding of his work. He was interested in all aspects of health work but his forte was the prevention of malaria, the problem which called forth from his fertile mind many original ideas and inventions.

Everyone conversant with the history of public health in Malaya will readily agree that the present good health of this country is due, in no small measure, to the untiring efforts of Dr. Scharff and the wonderful tradition of public health which, by precept and example, he has handed down to his successors, subordinates and students.

In the course of his work, he came across all strata of the populace and everyone, who had the good fortune to know him, benefitted by his encouragement, kindness and generosity. No one genuinely in need of help, who approached him, was ever turned away; and if there was one trait which endeared him to all who worked under him, it was his happy knack of remembering the names of all his subordinates, whether health officers, inspectors, overseers, or the lowliest of labourers in whose welfare he took the keenest interest.

Dr. Scharff was eminent in that long list of Colonial servants whose lives and work have so enriched Malaysia, and to his widow and seven children, we extend our deepest sympathy.

O.E.K.

NOTICES

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS

The Victorian State Committee is arranging a Course of Instruction in Post-graduate Surgery to be held in Melbourne from the 27th July to 2nd October, 1964.

The Course will be full-time. During the mornings, entrants will, with some restrictions, be able to attend and observe the work in the various clinical schools. Tuition will begin each week day at 2.00 p.m. and at 4.00 p.m. on each day there will be a session on Clinical Surgery. The Course has been so arranged to enable anyone who wishes to do so, to take off 12 weeks prior to the Final Examination for the F.R.A.C.S. in October, to prepare for this examination. In addition, the Clinical instruction is being conducted in the late afternoons, in order that Senior Resident Medical Officers may be able to attend this part of the Course.

The fees for the Course are:—

(A) Full time ... 20 gns.

(B) Clinical only ... 10 gns.

Entries for the Course close on 13th July, 1964. Candidates when entering must forward a remittance for the fee, viz. 20 gns. or 10 gns. as the case may be. Candidates resident in New Zealand, or elsewhere, should remit by bank draft drawn on Melbourne in favour of The Royal Australasian College of Surgeons, and payable in Australian currency.

RORY WILLIS,
Honorary Secretary,
Victorian State Committee.

Spring St.,
MELBOURNE: C.1.

25th May, 1964.

The Editor.

Dear Sir,

May I, as President of the Royal Medico-Psychological Association, ask you to bring to the attention of your readers the Sixth International Congress of Psychotherapy, which is being sponsored by the Association at the request of the International Federation of Medical Psychoterapy. The Congress is being held in *London from 24ht – 29th August, 1964.* At this Congress, the whole progress of psychotherapy in the last twenty-five years will be reviewed, with special reference to the diverse fields of activity in which psychotherapeutic techniques have recently developed.

Many psychotherapists of international standing are reading papers at this Congress and delegates are expected from all over the world. The Congress has not only a stimulating professional programme, but is balance by a variety of social events.

Registration forms and full details can be obtained from the Organising Secretary, 11 Whitehall Court, London, S.W.1.

Yours faithfully,

Desmond Curran.

President

Royal Medico Psychological Association

NUTRITION SOCIETY

FORTHCOMING MEETINGS

Friday, July 17, 1964.

The Society is holding, in Reading, a symposium on "The effect of soils, fertilizers and environment on yield and nutrient content of plants."

SUBSCRIPTIONS

Members who have not paid their subscription for 1964 are asked to send them immediately to the Hon. Treasurer, Dr. A. E. Bender, Research Department, Farley's Infant Food, Ltd., Galleymead Road, Colnbrook, Bucks. The subscription is £1 10s. for those who wish to receive only the Proceedings and £3 for those who wish to receive the British Journal of Nutrition as well.

It would greatly assist administration if members would pay their subscription by Banker's Order and the Hon. Treasurer will send the necessary form to any member on application.

Members are reminded that they may qualify for United Kingdom income tax relief in respect of their subscriptions. Full particulars may be obtained from any tax office. The effect of the relief for a member paying tax at 7s. 9d. will be to reduce the actual cost of his subscription to 18s. 5d. if he takes only the Proceedings, and £1 16s. 10d. if he takes the Journal as well.

DATIN LADY THOMSON, M.R.C.S., L.R.C.P.,
Hon. Overseas Correspondent in Malaya,
Institute for Medical Research,
Kuala Lumpur.

Symposium on "The effect of soils, fertilisers and environment on the yield and nutrient content of plants"

To be held at the University of Reading on 17th July, 1964.

Chairman: Professor R. G. Baskett

1. Ultimate limits of crop production — Professor J. N. Black
2. Limits imposed by rainfall and irrigation — Dr. Olivier
3. .. .. .. soil (a) inorganic — Dr. C. Bould
4. .. .. (b) organic — Professor A. H. Bunting
5. Composition in relation to agronomical practice. — Dr. R. Waite

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THE NUTRITION SOCIETY

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Saturday, September 12, 1964

The Scottish Group of the Society is holding in Dundee a symposium on "Major elements in Nutrition."

XVIII General Assembly of the World Medical Association

The XVIII General Assembly of the World Medical Association will take place in Helsinki from 13th to 19th June, 1964. It will include a scientific session on factors affecting the development of children. The Finnish Medical Association will act as host. Registration forms may be obtained from the Finnish Committee on Arrangements, c/o Finnish Medical Association, Ruoholahdenkatu, 4, Helsinki.

DATIN LADY THOMSON, M.R.C.S., L.R.C.P.,
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