

ASIAN

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EDITED BY THE JAPAN MEDICAL ASSOCIATION

Special Edition

The 16th General Assembly of the Japan Medical Congress

*The 3rd Congress of the Confederation of Medical Associations in
Asia and Oceania*

*List of International Medical Meetings to be held in Japan
(1963 - 1966)*

———— *March, 1963* ————



Invigorative and Detoxicative

GURONSAN

- *Detoxicative the poisoning substances of the living body.*
- *For invigorating metabolic functions of the living body.*
- *For improvement of liver functioning.*



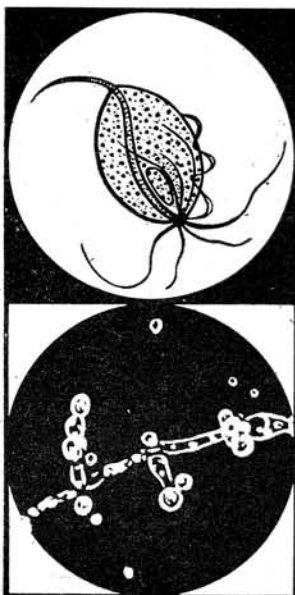
Chugai Pharmaceutical Co., Ltd.
Honcho, Nihonbashi, Chuo-ku, Tokyo, Japan

❖ *Trichomoniasis* ❖ *Candidiasis*
 ❖ *Mixed infections due to Trichomonas and Candida*

TRICHOMYCIN



TRICHOMONIASIS + CANDIDIASIS



Trichomycin is a new antibiotic produced by *Streptomyces hachijensis*, and being different from other antibiotics it selectively affects protozoa and fungi such as *Trichomonas*, *Ameba*, *Candida* and *Trichophyton*, &c. Thus, excellent therapeutic effects can be obtained with Trichomycin in vaginal trichomoniasis or vaginal candidiasis, and, moreover, mixed infections due to *Trichomonas* and *Candida* can effectively be treated with Trichomycin.

Trichomycin does not affect Döderlein's bacillus which plays a very important role in purification of vagina. Trichomycin is free from any side effect and can be administered safely; it has so far been proved to cause no resistant strains.

VAGINAL TABLETS: *Trichomonas vaginalis* vaginitis, *Candida albicans* vaginitis, mixed infections due to these organisms, leucorrhœa, pruritus vulvæ.

ORAL TABLETS: Disseminated, pulmonary and other internal candidiasis, *Trichomonas vaginalis* vaginitis, *Candida albicans* vaginitis, urinary trichomoniasis and candidiasis, mixed infections due to *Trichomonas* and *Candida*, and amebiasis.

OINTMENT: Dermatomycoses, cutaneous candidiasis and pruritus vulvæ.

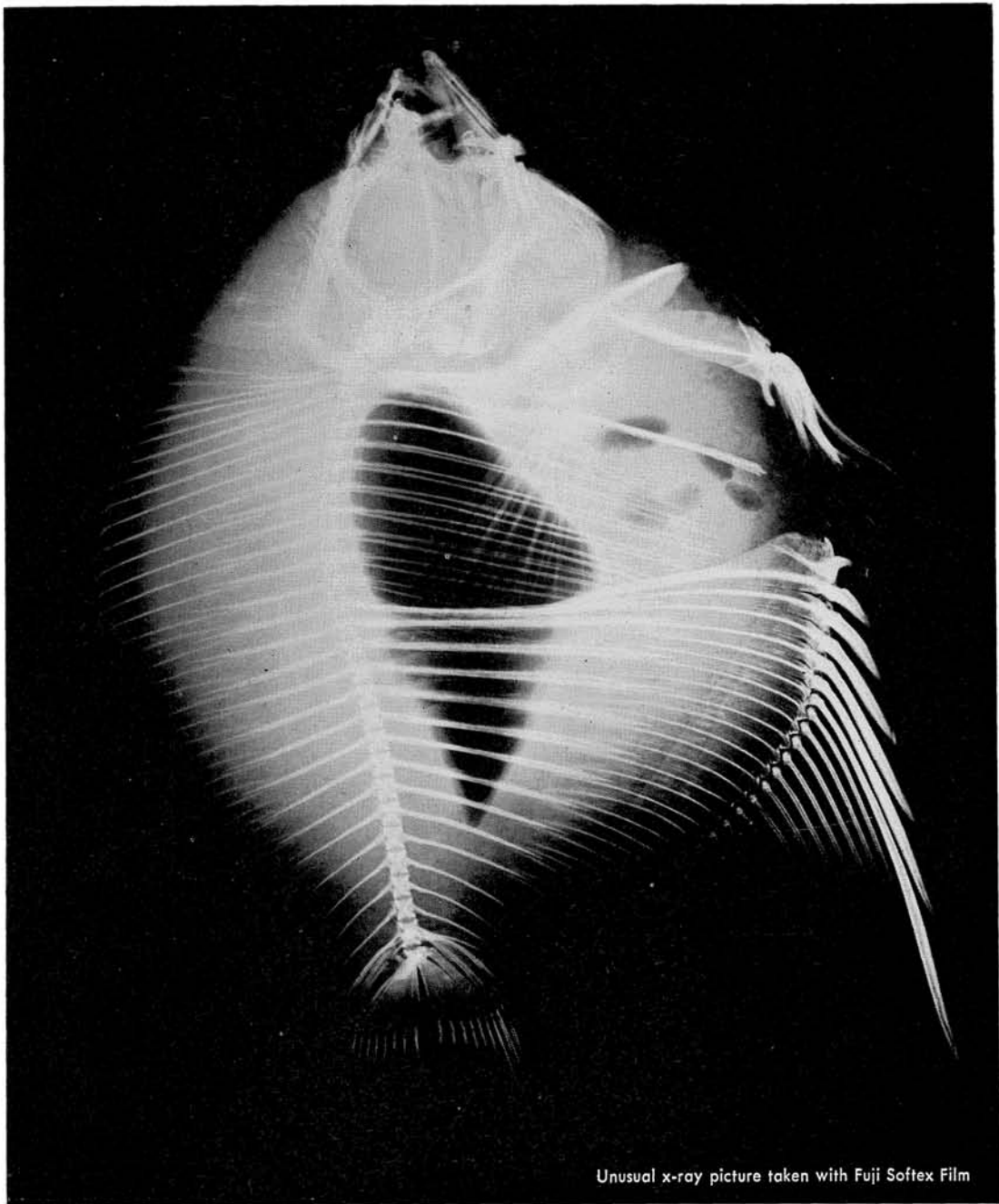
* Trichomycin is exclusively represented in more than 47 countries abroad.

** Further information and literature will be sent on request.

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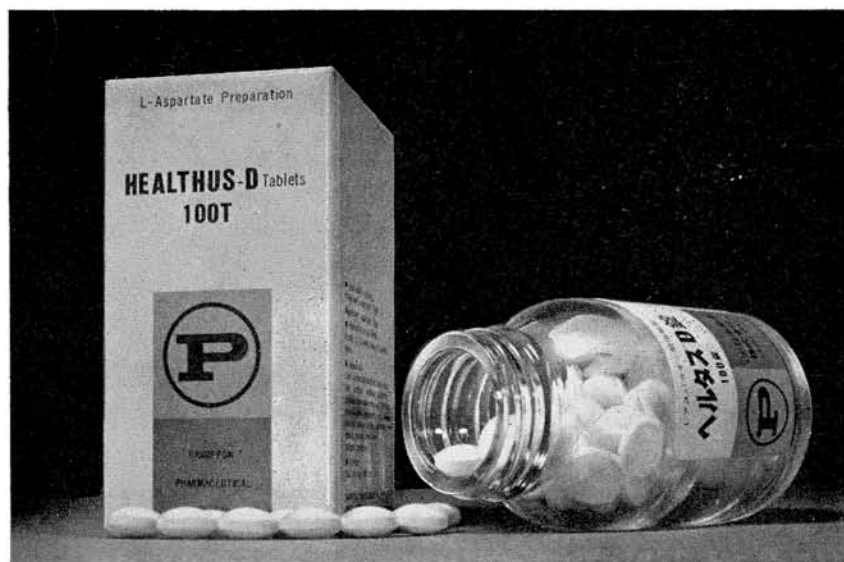
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Potassium L-aspartate.....75mg

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Healthus-D smoothes the TCA cycle.

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Healthus-D is of importance in the
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the muscular contraction power.

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Gestational toxicosis

Hyperammonemia

Hypokaliemia

Heart diseases



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- ★ Extremely well tolerated.

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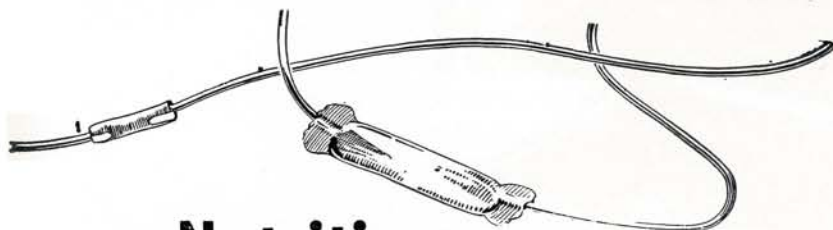


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Essential Amino Acids Injection in the Formula Recommended by
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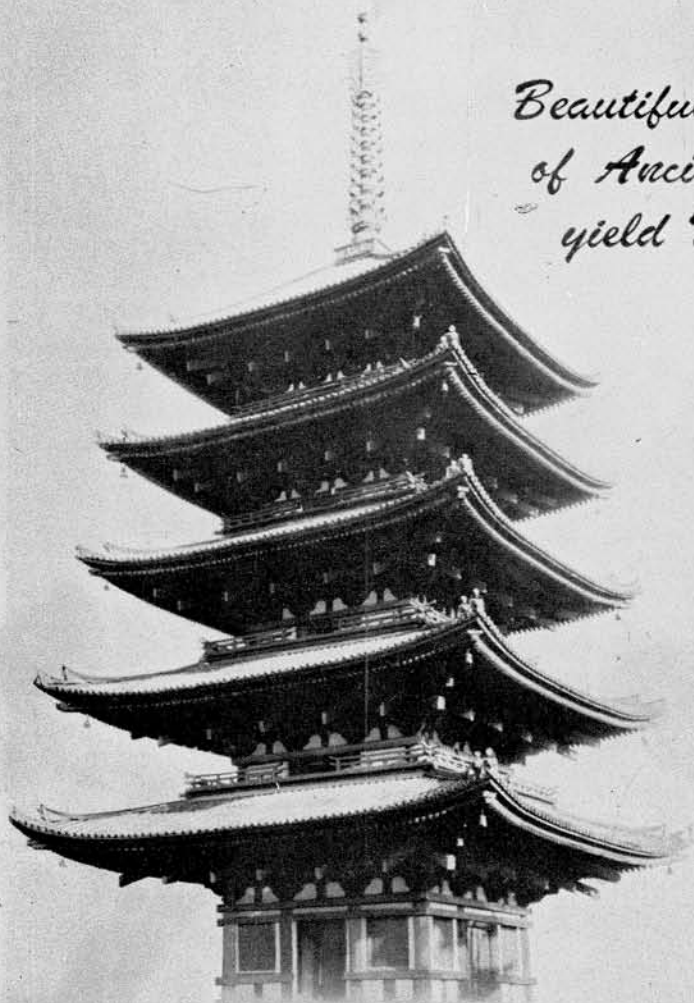
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**Conclusive Agent
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INDICATIONS

1. Respiratory tract infections due to Staphylococcus, Pneumococcus, Streptococcus viridans and Hemophilus influenzae.
2. Urinary tract infections due to Staphylococcus, Escherichia coli, Pseudomonas aeruginosa and Proteus vulgaris.
3. Gonococcal infections.
4. Suppurative infections due to Staphylococcus, Pneumococcus and Pseudomonas aeruginosa.
5. Bacillary dysentery.
6. Acute diarrhea.
7. Adnexitis.
8. Pulmonary tuberculosis, urinary tuberculosis, genital tuberculosis, and tuberculosis of the lymphnode.
9. Prophylactic use for post-operative infections.
10. Weil's disease.

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**Type
PCD-4**



The most important single step in the clinical inspection of blood samples is the cell count.

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A well-designed dark field illumination system produces a sharp optical contrast between the cells and their background. This exceptionally clear image means high visibility and high accuracy for the photoelectric counter.

The Shimadzu Photoelectric Automatic Blood Cell Counter will save you manpower, time, and money.

Specifications

1. Optical System
Illumination: Dark field system
Magnification of counted image: 20 times
Magnification of eye-piece: 10 times
2. Electronic System
Photomultiplier: Hamamatsu TV-R106 or
RCA 1P28
Counting tube: 4NIXIE Indicating Tubes
3. Light Source: Tungsten lamp 6V, 5A
4. Dimensions: width 500 mm, length
420 mm, height 460 mm

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Neoplasm Palliative

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A New Antibiotic, Chromomycin A₃, for Injection

CHARACTERISTICS

- Acts selectively on tumor cells and exerts a marked cell destroying effect.
- High affinity towards lymphatic tissue so that an effect is obtained in lymphnode metastasis.
- Marked improvement of subjective and objective symptoms.
- Superior life-prolonging effect.
- Minimal side effects. Low leucopenic effect makes possible long-term treatment.

INDICATIONS

Alleviation of subjective and objective symptoms in malignant chorioepithelioma and Hodgkin's disease.

Cancer (Gastric cancer, uterine cancer, pulmonary cancer, rectal cancer, breast cancer, esophageal cancer, liver cancer, skin cancer, carcinogenous peritonitis, Grawitz's tumor, ovarian cancer, perineal cancer).

Sarcoma (Reticulosarcoma, lymphosarcoma).

ADMINISTRATION AND DOSAGE

The standard method of administration and dosage is as follows.

Intravenous injection.

Dissolve 0.5 mg. of Chromomycin in 10 ml of sterile water for injection or glucose solution, inject intravenously once daily or at suitable intervals. Avoid leakage from vein as induration or necrosis may be produced at the site of injection. A single course of treatment usually consists of a total of 30 mg. May be repeated as indicated. May also be administered intra-arterially, directly into the pleural space, into the tumor or by local infusion.

CASE REPORT

Effect of Toyomycin in a Case of Cancer of The Transverse Colon

Katsuya, Yoshinaga and Masanobu Akagi
Department of Surgery,
Kumamoto University Medical School

The concomitant use of antitumor agents together with surgery is quite new but in view of the rise in therapeutic rate, this method has come to be widely used. This method is based on the effect of the antitumor agent against the "floating cancer cell".

Various antitumor agents have been used together with surgery since 1958 in the department and quite satisfactory results have been obtained.

In the present study, Toyomycin was used in a case of giant tumor of colon and the results are presented here.

● **Case 1:** O. M. 37 year old male

Clinical Diagnosis: Tumor of the Abdominal Wall.

Present Illness:

In July of 1960, a painful tumor of unknown cause developed in the left epigastric region. The tumor gradually increased in size. Localized peritonitis had been diagnosed elsewhere and a small quantity of purulent fluid removed by incision on Sept. 22. The tumor, however, continued to enlarge despite the incision and from Oct. 1, intestinal content began to be excreted from the site of incision and no trend for healing was noted for 2 months. The patient was referred to the Surgery Department on Dec. 15 and immediately admitted under the tentative diagnosis of malignant tumor of the abdominal wall.



Fig. 1 cancer of colon
(when admitted) ▶

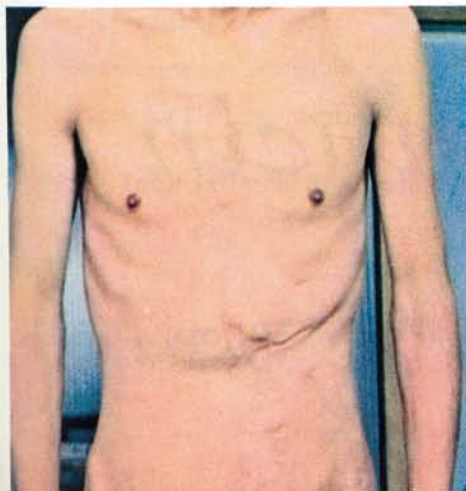
Toyomycin

Course:

The patient was somewhat emaciated, and there was severe pain in the tumor region and there was a complaint of inability to sleep, anorexia and general lassitude.

Chart 1 shows the tumor in situ. A large goose-egg sized tumor was present in the left upper quadrant. A fecal fistula could be seen in the central part of the tumor and moist, elevated granulating tissue surrounded the fistula. Routine tests revealed moderate anemia, leucocytosis and 14% positive B. S. P.

Other tests were negative. Treatment with Toyomycin was started on Dec. 25 in a daily dose of 1,000 mcg. intravenously and given for 30 days. There was a slight decrease in the pain and some localization of the tumor at one time, but the tumor again began to grow larger. From Jan. 7, Prednisolone was given concomitantly in a dose of 50 mg a day intramuscularly whereupon, the pain almost completely disappeared and there was no further increase in size of the tumor. As the tumor was larger compared to the time of admittance, radiotherapy was then tried but there was no response. After a total of



▲ Fig. 3 isolated tumor

◀ Fig. 2 after healing

30,000 mcg of Toyomycin surgery was carried out on Jan. 25 and the tumor totally removed. (Chart 3).

The postoperative course was uneventful and the patient was discharged on May 10. There has been no sign of reoccurrence after 1 year 10 months and the patient is living a normal life.

Chart 2 shows the patient after the operation.

SUMMARY

Toyomycin was given for a total of 30,000 mcg in a case of malignant tumor of the colon. The tumor was then removed surgically. There has been no sign of recurrence or metastasis after 1 year 10 months. Side effects were not observed. It is believed that treatment by surgery together with an antitumor agent is the method of choice at the present time.

Toyomycin

TAKEDA CHEMICAL INDUSTRIES, LTD. OSAKA·JAPAN

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List of International Medical Meetings to be held in Japan, 1963-1966

(compiled by J. M. A.)

**The 3rd Congress of The Confederation of Medical Associations
in Asia and Oceania**

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The 16th General Assembly of The Japan Medical Congress



—MANDRAKE—



*Place of Opening ceremony
(The Osaka Municipal Central Gymnasium)*



Flower of Mandrake

Flower of mandrake has been adopted as a symbol for the 16th General Assembly of Japan Medical Congress and its design has been made by Kenkichi Tomimoto (awarded Order for Culture Merits for 1961)

Seishu Hanaoka (1760-1835) came from Kii province, and conceived a prescription for general anesthetic called Tsūsensan or Mafutsusan.

The prescription was as follows.

Datura	8
Angelicae daburicae Radix	2
Cnidii Rizoma	2
Angelicae sinensis Radix	2
Arisaematis Rizoma	1

About 2 gram of Tsūsensan is boiled in about 360 cc of water to make about 820 cc of solution and is given to patients, who indulge themselves into state of narcosis one or two hours afterward but recover from it 5-6 hours afterward.

Historical recording tells that Seishu Hanaoka succeeded for the first time in excision of mammary cancer under state of general narcosis due to Tsūsensan on October 13th, 1805.

This had taken place about 40 years before a great operation was performed under state of narcosis resulting from ether or chloroform. In 1951 Seishu Hanaoka was enshrined in Hall of Fame in Chicago as "one of physicians with distinguished merits who contributed much to the human being of the world". Mrs. Hanaoka assisted him in his research, and making herself an object for experiment became blind. A Japanese picture portraying his lady lying in bed with closed eyes and Seishu himself and his mother looking at her is kept in Hall of Fame. This was dedicated by Japanese Surgical Society.



Welcome Message

The Medical sciences, benefiting from the progress made in the related sciences and inspired by their own humane traditions, continue to advance.

The 16th General Assembly of the Japan Medical Congress marks one more stage in the long history of Japanese Medicine.

The many foreign scholars who have been invited will be asked to speak about medical progress in their respective countries.

I hope, by the cooperation of all participants, the Congress will provide a good opportunity for the introduction to the world of the most up-to-date aspect of medical science, and I also hope the Congress will make a successful synthesis of the fruits achieved in the various fields of medical science during the past four years.

I am sure such an exchange of information by some of the most eminent proponents of medical science cannot fail to be of great benefit to humanity, and it gives me great pleasure to welcome all those who are to take part.

A handwritten signature in cursive script that reads "Arao Imamura".

ARAO IMAMURA M.D.

President

*The 16th General Assembly of
The Japan Medical Congress*

Outline of The 16th General Assembly of The Japan Medical Congress *April 1-5, 1963, Osaka*

The 16th General Assembly of the Japan Medical Congress will be held on April 1st, 2nd and 5th, 1963 and general meeting of each member society on 3rd and 4th, in Osaka respectively.

Ever since its first General Assembly in 1902, it has taken place every four years and is the largest in scale of the national meetings of medical sciences.

The Medical Congress to-day is consisted of 53 member societies all of which will participate in the next General Assembly and organize lectures, symposia on 150 subjects of special interest in various field of medicine.

At the coming scientific meeting, differing more or less from that of the last General Assembly of the Japan Medical Congress, special lectures by guest speakers (22 subjects), lectures at the General Assembly by Japanese specialists (58 subjects) and symposia (71 subjects) are scheduled.

Lists of subjects and lecturers, chairmen, have been decided after repeated discussions at the Program Compiling Committee organized at the headquarters and composed of chairman and 23 members, and the emphasis of selection are not only placed upon subject of cancer, but also those of adult diseases and viral diseases, but cover a wider field of medical treatment in general.

The total number of attendance is expected to exceed 30,000 or even 35,000. Besides the scientific meetings at some 17 halls, there will be a scientific exhibit and medical motion pictures and color-television. The firms will have a large scale technical exhibit. Several social events will be arranged for participants.

Doctors and medical scientists from abroad are cordially invited for the purpose of exchange of medical informations as well as of mutual understanding and friendliness. On the other hand, in order that the General Assembly may not become a feast to physicians only, but secure a general public correct understanding of advanced contemporary medicine as well as a knowledge of historical course of development of the medicine, many events such as lectures, motion pictures and exhibitions are scheduled.

Moreover, it will promote a significance of the General Assembly of the Japan Medical Congress and be becoming to a grand festivity of the Japanese medicine that the General Assembly will posthumously honor Kōan Ogata (born in Osaka) and Seishu Hanaoka (born in Wakayama), two Japanese pioneers in Western medical science, on this occasion. (the year of 1963 is the 100th death anniversary of Kōan)

The fact that a MANDRAKE flower, using which Seishu is believed to have succeeded in an operation by applying a general anesthetic for the first time in world medical history, is adopted as symbol of the General Assembly, stands for the foregoing reason.

Officers:

- President : Arao Imamura (Director of Center for Adult Disease, Osaka; former President of Osaka University and Emeritus Professor of Internal Medicine, Osaka University)
- Vice President : Yoshio Ozawa (Director of Osaka Workmen's Accidental Hospital; Emeritus Professor of Surgery, Osaka University)
- Secretary General : Imasato Dônomae (Director of Osaka Prefectural Hospital; Emeritus Professor of Internal Medicine, Osaka University)

Office :

The Memorial Hall of Osaka University Medical School,
33, Jyoan-cho, Kitaku, Osaka

Fees :

Membership Fee	1,000 yen
Family of Membership Fee	500 yen

Program

- I. Opening ceremony: 9.00 a.m. Monday, April 1, 1963
The Osaka Municipal Central Gymnasium, Osaka
(Osaka-shi Chûo Tai-ikukan)
- II. Scientific Meetings
- | | |
|--|-------------|
| 1) Special lectures by guest speakers | 22 subjects |
| 2) Lectures at the General Assembly
by Japanese Specialists | 58 subjects |
| 3) Symposia | 71 subjects |
- III. Exhibits
- 1) Scientific Exhibit (April 1—April 5)
 - 2) Technical Exhibit (April 1—April 5)
 - 3) Motion Pictures and Color-Television (April 1—April 5)
- IV. Evening Forum (April 3, 4, 7:00 p.m.)
- V. Memorial Lecture Meeting of the 100th Anniversary of Kôan [March 31, afternoon, Mainichi Kokusai Salon (R')]
- VI. Closing ceremony: 4.00 p.m. Friday, April 5, 1963
The Festival Hall, Osaka

Scientific Exhibit

Main Place: Furitsu Taiikukaikan (S)

(Near western side of Osaka Baseball Ground 200 meters to the southwest of Nanba station of the subway)

April 1st - 5th

- A: 1st floor, Main building, Furitsu Taiikukaikan**
Exhibit of recent progress in malignant tumors, virology, biochemistry, cardiovascular diseases, diseases of the cerebro-nervous system, endocrinology, allergy, infection and surgical operation as well as of matters related to trauma due to traffic accidents, hospital and medical electronics.
- B: Annex, Furitsu Taiikukaikan (S) (to the south of main building)**
(is located next to the place for TV.)
Exhibit of the newest equipments for operation room and anesthesiology, artificial respiration and blood transfusion.
- C: 2nd floor, Furitsu Taiikukaikan**
Exhibit of systematic classification of antibiotics, neurotropic agents and metabolites such as internal secretion and vitamin as well as of application of these agents.
Recent statistics of medicine, medical care and population problem.
- D: Kokusai Mihon-ichi Kaikan (In the International Hotel Osaka (T), Uchihon-cho, Osaka)**
Exhibit chiefly intended for demonstration related to medical electronics.

Technical Exhibit

April 1st – 5th

The first place (Osaka Furitsu Taiikukan (Y))

1st floor, The Main Building

Scientific exhibit and exhibit of medical facilities, cameras, precision instruments, nutritive eatables and business instruments.

2nd floor

Scientific exhibit and exhibit of medical drugs and a part of medical facilities.

Middle garden

Medical Automobile and Automobile.

Annex

Color television and scientific exhibit, exhibit of medical facilities attached to operation room.

The second place (Kokusai Mihon-ichi Kaikan (In the International Hotel, Osaka (T)))

1st floor

Exhibit of photographic materials.

2nd floor

Exhibit of large-sized medical electronic facilities.

3rd floor

Scientific exhibit (performance of medical electronic facilities) and exhibit of medical electronic facilities.

Color Television

Place: Annex of Furitsu Taiikukaikan (Bekkan)

The television is put on a large screen and operation is as a rule explained by an expositor and interlocutor. Direct questions and answers are exchanged between operator and interlocutor, and audience.

Operation Room: The Operation Room, Osaka University

Talking Room: Room for Professors Emeritus, Osaka University

Program is expected to cover various fields of operation.

April 1st, p.m. 13:30 - 14:30, 15:00 - 16:00

2nd - 4th, a.m. 9:30 - 10:30, 11:00 - 12:00

place date		Mitsukoshi Theater	International Salon (Kokusai Salon)	Festival Hall	Mainichi Hall	Sankei Hall
Apr. 1	daytime	ground (Japanese and American)	surgical (Japanese and American)			
	night			surgical (Japanese and American)	ground (Japanese and American)	internal (Japanese and American)
Apr. 2	daytime	ground (Japanese and American)	surgical (Japanese and American)			
	night			surgical (Japanese and American)	ground (Japanese and American)	internal (Japanese and American)
Apr. 3	daytime	ground (Japanese and American)	surgical (Japanese and American)			
	night					
Apr. 4	daytime	ground (Japanese and American)	surgical (Japanese and American)			
	night				surgical (Japanese and American)	internal (Japanese and American)
Apr. 5	daytime		showing superior films repeatedly (Japanese and American)			

Contents

GROUND :

Problem of Cancer Cells
Pathology of Inflammation
Oliguria
Collateral Blood Flow due to Obliteration of the Coronary arteries
Problem of Allergy
Structure of Human Erythrocytes
Protein and Amino Acid
The World of Pulse

CLINICAL :

Internal : Problem of Tuberculosis
Elements for Neurological Diagnosis
Diagnosis of Viral Meningitis
Diseases of Cerebral Blood-vessels
Pathology and Treatment of vomiting
Cough

Surgical : Functional Anatomy of the Hand
Head Injuries
Refrigeration Treatment of stomach Ulcer
Problem of Burns
Clinical Anatomy of Hilus of the Lung
Tumor of the Ureter
A Collection of Rare Diseases (a film awarded American Surgical Association Prize)

Operative : A New Field of Blood Transfusion
Anesthesia of Old Person
Heart Massage
Hypothermy for the Cardiac Operation
Exogenous Circulation
Operation of Cancer of the Pancreas
Transplantation of the Kidney
Operation of Hypertensive Cerebral Hemorrhage
Adhesive Operation of Amputated Limbs
Operation of Patent Interventricular Septum
Urological Operation

Evening Forum

Clinical Experiences

Date: 7.00 p.m. April 3.

Place: Festival Hall (F),
Mainichi Hall (J), Sankei Hall (K).

Festival Hall

Some problems Concerning to Mastopathy

Yasumasa AOYAGI (Prof. Emeritus of Kyoto University)

Recollection in 45 years as a surgeon

Hito-o IWANAGA (Prof. Emeritus of Osaka University)

An adage from Abdominal Surgery

Masao MUTÔ (president, Fukushima Medical College)

Operation of Pseudopelade

Tomosuke MAEDA (Director of Maeda Surgical Hospital)

Mainichi Hall

Men put on years with blood vessel

Yoshio KATÔ (Prof. Emeritus of the Tokyo Jikei-kai University)

From my experience

Seizo KATSUNUMA (Prof. Emeritus of Nagoya University)

Actual state of Chemotherapy

Kanshi SASSA (director of Kanto Teishin Hospital)

Instruction from "The Periodic Examination"

Tandô MISAO (Prof. Emeritus of Kyûshû University)

Sankei Hall

Botulinus toxication in Japan—Epidemy, Clinic and Serotherapy—

Yutaka NAKAMURA (Prof. Emeritus of Hokkaido University)

Experience from Expert's Opinion

Yûshi UCHIMURA (Prof. Emeritus of University of Tokyo)

My Experience in 40 years with Infectious disease

Kenzaburo KUMAGAYA (Honorary director of Momoyama Hospital)

Panel Discussion

Date: 7.00 p.m. April 4

Places: Festival Hall (F)

Subject

On the Development of Medical Care in Japan

Chairman

Tarô TAKEMI (President of Japan Medical Association)

Speakers

Haruo KATSUNUMA (Prof. of Public Health, University of Tokyo)

Katsumi TAKASHIMA (Chairman of Special Committee of Survey and
Investigation, Japan Medical Association)

Teishiro SEKI (Prof. of Public Health, Osaka University)

Chôtârô TAKAHASHI (Prof. of Economics, Hitotsubashi University)

Ichiro NAKAYAMA (Prof. Emeritus of Hitotsubashi University,
Economics)

Honorary Officer

1. Honorary presidents

TARÔ TAKEMI	President of Japan Medical Association
TAKEO TAMIYA	President of the Japanese Association of Medical Sciences
YŪSHI UCHIMURA	President of the 15th General Assembly of the Japan Medical Congress
SHIN-ICHI MATSUMOTO	President of the 14th General Assembly of the Japan Medical Congress
YASHIRÔ KOTAKE	Former-president of Wakayama Medical College

2. Honorary members

Hamao Umezawa	Prof. Institute of Applied Microbiology, University of Tokyo, discoverer of KANAMYCIN
Munenori Enjōji	President of Kyūshū University
Tomio Ogata	Vice-president of the 15th General Assembly of the Japan Medical Congress
Yoshito Kobayashi	Vice-president of the 15th General Assembly of the Japan Medical Congress
Tomosaburo Ogata	Emeritus-prof. University of Tokyo
Naosuke Onodera	Emeritus-prof. Kyūshū University
Seizō Katsunuma	Former-president of Nagoya University
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Hiroshige Shioda	Emeritus-prof. University of Tokyo
Akira Takahashi	Emeritus-prof. University of Tokyo

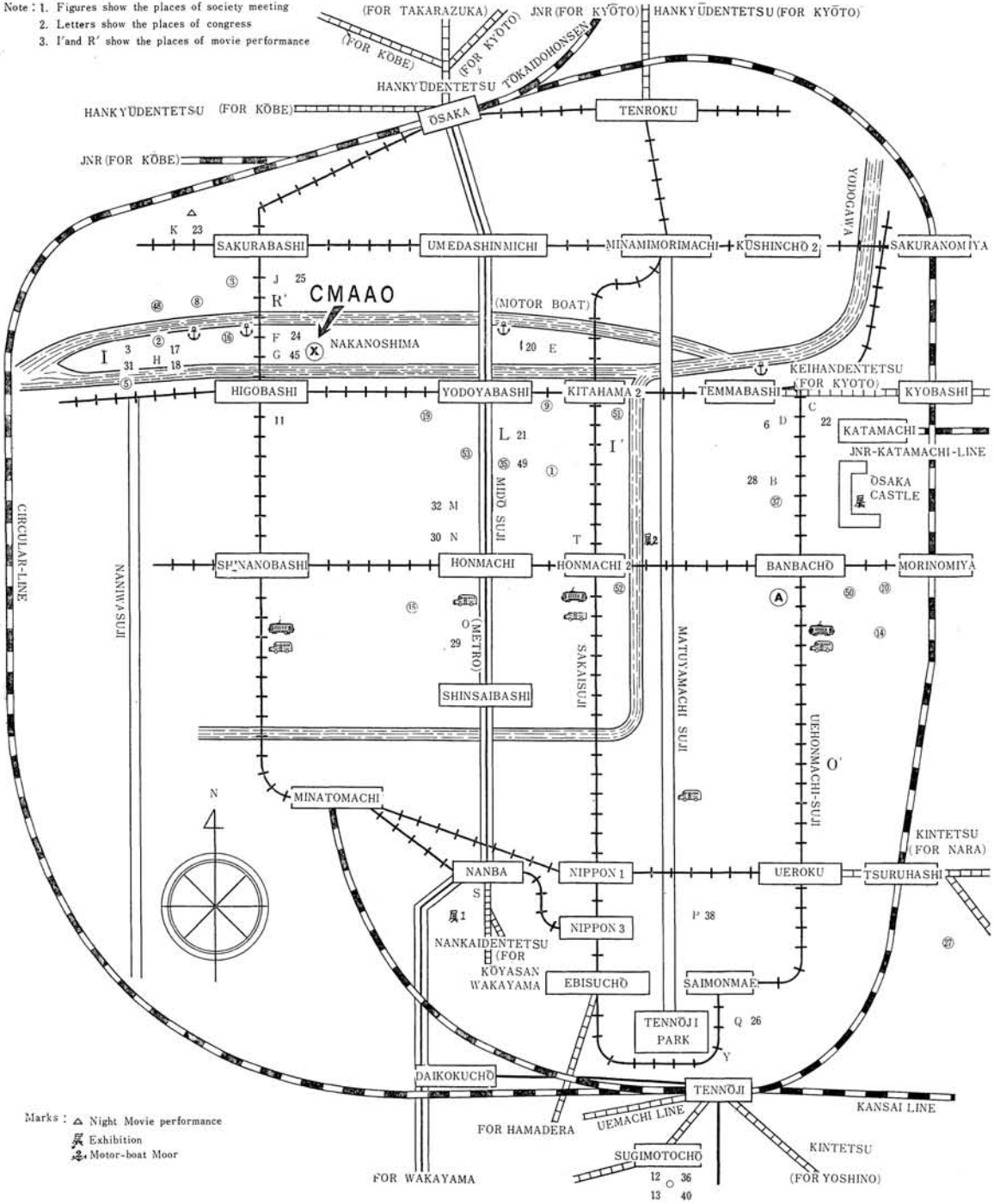
Yasaburô Taniguchi	Former-president of Japan Medical Association
Masatsugu Tomita	Member of Japan Academy
Kô Hirasawa	President of Kyoto University
Kenzô Futaki	Emeritus-prof. University of Tokyo
Tanemoto Furuhata	Emeritus-prof. University of Tokyo
Iwao Matsuo	Emeritus-prof. Kyoto University
Kensuke Mitsuda	Former-president of Nagashima-Aiseien (Leprosarium)
Tomizô Yoshida	Prof. University of Tokyo
Nobutaka Yoshimatsu	Former-president of Nara Medical College
Toyotane Wada	Emeritus-prof. Osaka University

3. Consultant

Shirô Akabori	President of Osaka University
Shirô Ohtagaki	President of Kansai Denryoku Co, Ltd.

Map of Osaka

- Note: 1. Figures show the places of society meeting
 2. Letters show the places of congress
 3. 'I' and 'R' show the places of movie performance



Places

Place of Opening Ceremony

A Osaka-shi Chuo Taiikukan

Place of General Assembly

B Kokumin Kaikan

C Ôtemae Kaikan

D Osaka Dental College Hall

E Chuo Kokaido

F Festival Hall

G A B C Hall

H Osaka University Hall

I Nissei Kenshujo

J Mainichi Hall

K Sankei Hall

L Nihon Seimei Hall

M Gas Building Hall

N Soai Gakuen Hall

O Mido Kaikan

P Furitsu Shakaijigyo Tandai Hall

Q Shitennoji Kaikan

Place of Closing Ceremony

F Festival Hall

Place for Movie Performance

I' Mitsukoshi Gekijo

R' Mainichi Kokusai Salon

F Festival Hall

J Mainichi Hall

K Sankei Hall

Place of Exhibition

S Osaka Furitsu Taiikukan

T Kokusai Mithon-ichi Kyokai

Y Osaka Municipal Museum

Place of the 100th death anniversary of Kôan Ogata

J Mainichi Hall

CMAAO

X Dentsu Building

Osaka Medical Center

O' Osaka Medical Association

SCIENTIFIC MEETINGS

APRIL 1 (Mon.)

Note: * Letters show the place

Morning

The Opening Ceremony

* A, Apr. 1, 9.00 a.m.-11.00 a.m.

On the Formation of Foreprotein

* A, Apr. 1, 11.00 a.m.-12.30 p.m.

(Memorial lecture at the opening ceremony)

Shiro AKABORI

(President of Osaka University)

Neuere Ergebnisse und Probleme der Biochemie

* A, Apr. 1, 11.00 a.m.-12.30 p.m.

(Memorial lecture at the opening ceremony)

Adolf Butenandt

(Direktor, Max-Planck-Institute für Biochemie, Deutschland)

Afternoon

Stomach Cancer among Japanese

* F, Apr. 1, 1.30 p.m.-2.30 p.m.

Toshio KUROKAWA

(President of Tohoku University)

Clinical and Pathological Studies on the Ovarian Solid Tumors

* E, Apr. 1, 1.30 p.m.-2.30 p.m.

Kazushige HIGUCHI

(Prof. of Jikei University)

Surgical Treatment of Frontal Lobe Epilepsy

* K, Apr. 1, 1.30 p.m.-2.30 p.m.

Theodore Rasmussen

(Prof. of McGill Univ., Canada)

Artificial Internal Organs

* J, Apr. 1, 1.30 p.m.-2.30 p.m.

Seiji KIMOTO

(Prof. of Tokyo University)

Muscle Relaxants and the Nature of the Neuromuscular Junction

* G, Apr. 1, 1.30 p.m.-3.30 p.m.

Yutaka ONCHI

(Prof. of Nara Medical College)

The Role of the Pituitary Gland on Metabolism

* B, Apr. 1, 1.30 p.m.-2.30 p.m.

Bernardo A. Houssay

(Director, Instituto de Biología y Medicina Experimental, Argentina)

Modern Trends in the Treatment of Congestive Heart Failure

* C, Apr. 1, 1.30 p.m.-2.30 p.m.

Magojiro MAEKAWA

(Prof. of Kyoto University)

- On the Formation of Cancer
* H, Apr. 1, 1.30 p.m.-2.30 p.m. Ryojun KINOSITA
(City of Hope Medical Center)
(Prof. Emeritus of Osaka University)
- Studies of the End-apparatus of Vegetative Nerve
* D, Apr. 1, 1.30 p.m.-2.30 p.m. Kiyoshi SUZUKI
(Prof. of Osaka City University)
- Sarcoidosis
* O, Apr. 1, 1.30 p.m.-3.30 p.m. Kanehiko KITAMURA
(Prof. of Tokyo Medical College)
- Allergy; Its Concept and Development
* N, Apr. 1, 1.00 p.m.-2.30 p.m. Tomio OGATA
(Prof. Emeritus of Tokyo University)
- Relation of Structure to Function of the Transfer RNA Molecule
* P, Apr. 1, 1.30 p.m.-2.30 p.m. Paul C. Zamecnik
(Prof. of Harvard Univ., U.S.A.)
- Etude thermodynamique de l'isohemagglutinine humaine
* L, Apr. 1, 1.30 p.m.-2.30 p.m. René Würmser
(Prof., Univ. de Paris, France)
- A Role of Japanese Mosquitoes in the Transmission of Filariasis
* I, Apr. 1, 1.30 p.m.-2.30 p.m. Nanzaburo OMORI
(Prof. of Nagasaki University)
- An Examination of the Traditional Concept of Medical Care
* Q, Apr. 1, 1.30 p.m.-2.30 p.m. Thomas McKeown
(Prof., Medical School of Birmingham, England)
- Cancer of the Stomach
* F, Apr. 1, 2.30 p.m.-5.00 p.m. Toichiro SAWADA
(Prof. Emeritus of Kyushu University)
- Chronic Renal and Vascular Changes following Apparent Toxemia of Pregnancy
* E, Apr. 1, 2.30 p.m.-5.00 p.m. Katsuji KUSHIMA
(Prof. of Tohoku University)
- Brain Tumors
* K, Apr. 1, 2.30 p.m.-5.00 p.m. Mizuho NAKATA
(Prof. Emeritus of Niigata University)
- Artificial Internal Organs
* J, Apr. 1, 2.30 p.m.-5.00 p.m. Jiro MIKAMI
(Prof. of Hokkaido University)

- The Modern Institution and its Equipment in the Operating Room, based on Central Supply System
 * G, Apr. 1, 3.30 p.m.-5.00 p.m. Tadanobu TAKAGI
 (Assistant Director of Surgical Center, Tokyo University)
- Central Control of Pituitary Hormones
 * B, Apr. 1, 2.30 p.m.-5.00 p.m. Shibanosuke KATSUKI
 (Prof. of Kyushu University)
- Coronary Circulation and Myocardial Metabolism
 * C, Apr. 1, 2.30 p.m.-5.00 p.m. Tôru HARA
 (Prof. of Osaka Medical College)
- Insecticides
 * M, Apr. 1, 2.30 p.m.-5.00 p.m. Hideo TANAKA
 (Prof. of Osaka City University)
- Medical Application of Tissue Culture
 * H, Apr. 1, 2.30 p.m.-5.00 p.m. Juntaro KAMAHORA
 (Prof. of Osaka University)
- Central Nervous Control of Micturition
 * D, Apr. 1, 2.30 p.m.-3.30 p.m. Masaru KURU
 (Prof. Emeritus of Osaka University)
 (Director of the Hospital, National Cancer Center)
- Experimental Studies on the Central Autonomic Nervous System
 * D, Apr. 1, 3.30 p.m.-5.00 p.m. Toshiyuki KUROTSU
 (Prof. Emeritus of Osaka University)
- Drug Resistant Staphylococcal Infections, with special emphasis in the Field of Surgery
 * O, Apr. 1, 3.30 p.m.-5.00 p.m. Yaemon SHIRAHA
 (Prof. of Osaka City University)
- Properties of Antibodies in Allergy
 * N, Apr. 1, 2.30 p.m.-5.00 p.m. Keizo NAKAMURA
 (Director of National Institute of Health)
- Biosynthesis of Proteins
 * P, Apr. 1, 2.30 p.m.-5.00 p.m. Shiro AKABORI
 (President of Osaka University)
- Recent Development and Medical Application of physico-chemical Biology
 * L, Apr. 1, 2.30 p.m.-5.00 p.m. Hideo KUBO
 (Prof. of Osaka University)
- Filariasis
 * I, Apr. 1, 2.30 p.m.-5.00 p.m. Hachiro SATO
 (Prof. of Kagoshima University)

- Proposals for the Medical Planning
 * Q, Apr. 1, 2.30 p.m.-5.00 p.m.
- Teishiro SEKI
 (Prof. of Osaka University)

APRIL 2 (Tue.)

Morning

- Progress in the Radiological
 Diagnosis of Malignant Tumors
 * F, Apr. 2, 9.00 a.m.-10.00 a.m.
- Kazuyuki NARABAYASHI
 (Prof. of Kobe Medical College)
- Liver Cancer among Japanese
 * E, Apr. 2, 9.00 a.m.-10.00 a.m.
- Toru MIYAJI
 (Prof. of Osaka University)
- Head Injury
 * K, Apr. 2, 9.00 a.m.-10.00 a.m.
- Kentaro SHIMIZU
 (Prof. of Tokyo University)
- Artificial Respiration
 * J, Apr. 2, 9.00 a.m.-10.00 a.m.
- Hideo YAMAMURA
 (Prof. of Tokyo University)
- Plastic Operation for Cleft Palate
 * G, Apr. 2, 9.00 a.m.-10.00 a.m.
- Iwao NAGAI
 (Prof. of Osaka University)
- Steroids and Electrolyte Metabolism
 * B, Apr. 2, 9.00 a.m.-10.00 a.m.
- Takeshi NAKAO
 (Prof. of Jikei University)
- Hemodynamics & Myocardial
 Metabolism in Congestive
 Heart Failure
 * C, Apr. 2, 9.00 a.m.-10.00 a.m.
- Ernst Wollheim
 (Prof., Univ., Würzburg, Deutschland)
- Acceleration Phenomenon of
 Development of Children in
 Japan
 * M, Apr. 2, 9.00 a.m.-10.00 a.m.
- Toshio TAKAI
 (Prof. of Osaka City University)
- Viral Encephalitis in Japan
 * H, Apr. 2, 9.00 a.m.-11.00 a.m.
- Keigo UCHIYAMA
 (Prof. of Nippon Medical College)
- Facilitation and Inhibition in the
 Nervous System
 * D, Apr. 2, 9.00 a.m.-11.00 a.m.
- Naoki TOIDA
 (Prof. of Kyushu University)
- Side Phenomenon of
 Chemotherapy
 * O, Apr. 2, 9.00 a.m.-11.00 a.m.
- Yuzo KAWAMORI
 (Prof. of Kumamoto University)

- Host Factors contributing to Protection against Infection
* N, Apr. 2, 9.00 a.m.-10.00 a.m. Tsunehisa AMANO
(Prof. of Osaka University)
- Immunochemistry of Biologically Active Proteins
* N, Apr. 2, 10.00 a.m.-12.30 p.m. Tsunehisa AMANO
(Prof. of Osaka University)
- Physiological Aspects of Protein and Amino Acid Nutrition
* P, Apr. 2, 9.00 a.m.-10.00 a.m. Masami SUDA
(Prof. of Osaka University)
- Mechanism of Active Transport through Biological Membranes
* P, Apr. 2, 10.00 a.m.-12.30 p.m. Hisato YOSHIMURA
(Prof. of Kyoto Prefectural University of Medicine)
- The Relationship between Metabolism and Action of Drug
* L, Apr. 2, 9.00 a.m.-11.00 a.m. Reiji IMAIZUMI
(Prof. of Osaka University)
- Diagnosis and Treatment of Paragonimiasis
* I, Apr. 2, 9.00 a.m.-10.00 a.m. Muneo YOKOGAWA
(Prof. of Chiba University)
- Toxioplasmosis, its Infection and Development
* I, Apr. 2, 10.00 a.m.-12.30 p.m. Hisakichi MATSUBAYASHI
(Prof. of Keio University)
- The Effect of Hypoxia during Exertion on Heart Function and Blood Elements
* Q, Apr. 2, 9.00 a.m.-10.00 a.m. Toshio ODA
(Prof. Emeritus of Osaka City University)
- Sports and Physical Control
* Q, Apr. 2, 10.00 a.m.-12.30 p.m. Toshiro AZUMA
(Prof. of Juntendo University)
- Radiation Therapy of Malignant Tumors
* F, Apr. 2, 10.00 a.m.-12.30 p.m. Hiroshi TACHIIRI
(Prof. of Osaka University)
- Girrhosis and Carcinoma of the Liver
* E, Apr. 2, 10.00 a.m.-12.30 p.m. Tadao TAKAHASHI
(Prof. of Jikei University)
- Traffic Accidents; Surgical, Psychiatric and Forensic-Medical Aspects
* K, Apr. 2, 10.00 a.m.-12.30 a.m. Syotaro MIZUNO
(Prof. of Osaka University)

- Hypothermia in the Field of Surgery
* J, Apr. 2, 10.00 a.m.-12.30 p.m. Shigetsugu KATSURA
(Prof. of Tohoku University)
- Cleft Palate Surgery for the Improvement of Speech
* G, Apr. 2, 10.00 a.m.-12.30 p.m. Tadashi UENO
(Prof. of Tokyo Medical and Dental University)
- Clinical Aspects of Steroid Hormones
* B, Apr. 2, 10.00 a.m.-12.30 p.m. Tadashi MIYAKE
(Prof. of Kyoto University)
- Essential Hypertension
* C, Apr. 2, 10.00 a.m.-12.30 p.m. Fusakichi NAKAZAWA
(Prof. Emeritus of Tohoku University)
- Some Features, Scientific and Administrative, of Hygiene in Japan
* M, Apr. 2, 10.00 a.m.-11.00 a.m. Saburo KAJIWARA
(Director of Osaka Prefectural Institute of Public Health)
- Vicissitude of Hygiene of the Mother and Child in Japan observed from the Maternity Hospital
* N, Apr. 2, 11.00 a.m.-12.00 a.m. Naotaro KUJI
(President of Tokyo Women's Medical College)
- Prophylaxis of Influenza, especially on Live Vaccine
* H, Apr. 2, 11.00 a.m.-12.30 p.m. Yoshiomi OKUNO
(Prof. of Osaka University)
- Neural Plasticity and the Memory Process
* D, Apr. 2, 11.00 a.m.-12.30 p.m. Horace W. Magoun
(Prof., Univ. of California, U.S.A.)
- Some Problems in the Chemotherapy of Infectious Diseases
* O, Apr. 2, 11.00 a.m.-12.30 p.m. Harry F. Dowling
(Prof., Univ. of Illinois, U.S.A.)

Afternoon

- Chemotherapy of Malignant Tumor
* F, Apr. 2, 1.30 p.m.-4.00 p.m. Tomizo YOSHIDA
(Prof. of Tokyo University)
- Development and Suppression of the Cancer
* E, Apr. 2, 2.30 p.m.-5.00 p.m. Shigeyasu AMANO
(Prof. of Kyoto University)
- Cerebral Apoplexia
* K, Apr. 2, 1.30 p.m.-2.30 p.m. Shigeo OKINAKA
(Prof. of Tokyo University)

- Cerebrovascular Disorders
* K, Apr. 2, 2.30 p.m.-5.00 p.m. Shigeo OKINAKA
(Prof. of Tokyo University)
- Problems in the Surgical Treatment of Aortic & Mitral Valves Insufficiency
* J, Apr. 2, 1.30 p.m.-2.30 p.m. Viking O. Björk
(Prof., Univ. of Uppsala, Sweden)
- Vascular Surgery
* J, Apr. 2, 2.30 p.m.-5.00 p.m. Seiji KIMOTO
(Prof. of Tokyo University)
- Plastic Surgery in Urology
* G, Apr. 2, 1.30 p.m.-2.30 p.m. Kyoichiro OCHIAI
(Prof. of Tokyo Medical and Dental University)
- La Nostra Esperienza in Chirurgia Cardiaca
* G, Apr. 2, 2.30 p.m.-3.30 p.m. A. Mario Dogliotti
(Direttore Clinica Chirurgica Generale, Italy)
- Physiological defect of Urogenital system
* G, Apr. 2, 3.30 p.m.-5.00 p.m. O. Swenson
(Children's Memorial Hospital, U.S.A.)
- Critical Analysis of Theories of Insulin Action
* B, Apr. 2, 1.30 p.m.-2.30 p.m. Rachmiel Levine
(Prof. of New York Medical Coll., U.S.A.)
- Obesity-A Disorder of Metabolism
* B, Apr. 2, 2.30 p.m.-3.30 p.m. Edwin B. Astwood
(New England Center Hospital, U.S.A.)
- Bone Neoplasm
* F, Apr. 2, 4.00 p.m.-5.00 p.m. Isaharu MIKI
(Prof. of Tokyo University)
- Regeneration, Hyperplasia, und Kanzerisierung
* E, Apr. 2, 1.30 p.m.-2.30 p.m. Franz Büchner
(Prof., Univ. Freiburg, Deutschland)
- Hypertensive Fundus Changes
* C, Apr. 2, 1.30 p.m.-3.30 p.m. Takashi MIZUKAWA
(Prof. of Osaka University)
- Metabolism of the Retina
* C, Apr. 2, 3.30 p.m.-5.00 p.m. Yoshi KURACHI
(Prof. of Kanazawa University)
- The Present Status of Balneotherapy in Japan
* M, Apr. 2, 1.30 p.m.-2.30 p.m. Takayoshi MISAWA
(Prof. Emeritus of Tokyo University)

- History of Dissection and Autopsy in Japan
* M, Apr., 2, 2.30 p.m.-4.00 p.m.
- Teizo OGAWA
(Prof. Emeritus of Tokyo University)
- Common Cold Viruses
* H, Apr. 2, 1.30 p.m.-2.30 p.m.
- Sir C. H. Andrews
(Director, Common Cold Research Unit, England)
- Respiratory Viral Diseases
* H, Apr. 2, 2.30 p.m.-5.00 p.m.
- Osamu KITAMOTO
(Prof. of Tokyo University)
- Biochemical Research in Mental Illness
* D, Apr. 2, 1.30 p.m.-2.30 p.m.
- Derek Richter
(Director of Medical Research Council, England)
- Pharmacotherapy of Mental Disorders
* D, Apr. 2, 2.30 p.m.-5.00 p.m.
- Ziro KANEKO
(Prof. of Osaka University)
- So called "Atypical" Acid Fast Bacilli
* O, Apr. 2, 1.30 p.m.-2.30 p.m.
- Tadao TODA
(Prof. of Kyushu University)
- Lung Tuberculosis caused by Drug-fast Tubercle Bacilli in Japan. Present Status and Countermeasures
* O, Apr. 2, 2.30 p.m.-5.00 p.m.
- Mitsuo HORI
(Prof. of Osaka University)
- The Genetics of Microorganisms
* N, Apr. 2, 1.30 p.m.-3.30 p.m.
- Tomoichiro AKIBA
(Prof. of Tokyo University)
- Inoculation of Mycobacterium Leprae into Experimental Animals, and Identification of Increased Bacilli in Infected Locus
* N, Apr. 2, 3.30 p.m.-5.00 p.m.
- Shinji NISHIMURA
(Prof. of Osaka University)
- Secretion and Absorption: from Morphological Aspect of Cytology
* P, Apr. 2, 1.30 p.m.-3.30 p.m.
- Toshio ITO
(Prof. of Gunma University)
- Metabolism of Tryptophan and Experimental Diabetes Mellitus
* P, Apr. 2, 3.30 p.m.-5.00 p.m.
- Yahito KOTAKE
(Prof. of Nagoya University)
- Action of Drugs and Cell Membrane
* L, Apr. 2, 1.30 p.m.-3.30 p.m.
- Tsuneyoshi TANABE
(Prof. of Hokkaido University)

- Concept of Body Fluid Circulation
* L, Apr. 2, 3.30 p.m.-5.00 p.m.
- Yasuyoshi NISIMARU
(Prof. of Hiroshima Jogakuin Women's University)
(Atomic Bomb Casualty Commission)
- Mycoses in Japan
* I, Apr. 2, 1.30 p.m.-2.30 p.m.
- Yoshisada TAKAHASHI
(Prof. of Tohoku University)
- Pulmonary Aspergillosis
* I, Apr. 2, 2.30 p.m.-5.00 p.m.
- Yoshio MIKAMO
(Prof. Emeritus of Tokyo University)
- Social Aspects of Disease
* Q, Apr. 2, 1.30 p.m.-2.30 p.m.
- A. Leslie Banks
(Prof., Univ. of Cambridge, England)
- Influence of Aging on Incidence and Malignancy
* Q, Apr. 2, 2.30 p.m.-3.30 p.m.
- E. V. Cowdry
(Research Prof., Washington Univ., U.S.A.)
- Studies of Industrial Toxicology (especially on Occupational Diseases due to Heavy Metals and Organic Solvents)
* Q, Apr. 2, 3.30 p.m.-5.00 p.m.
- Susumu HARASHIMA
(Prof. of Keio University)
- The Impact Continuing Education on Teaching, Research, and the Practice of Medicine
* B, Apr. 2, 3.30 p.m.-4.30 p.m.
- Seymour M. Farber
(Assistant Dean, University of California)

APRIL 5 (Fri.)

Morning

- Present Status of Lung Cancer in Japan.
* F, Apr. 5, 9.00 a.m.-10.00 a.m.
- Naoji KAWAI
(Prof. Emeritus of Chiba University)
- Cancer of the Lung
* F, Apr. 5, 10.00 a.m.-12.30 p.m.
- Naoji KAWAI
(Prof. Emeritus of Chiba University)
- Hypertrophy and Carcinoma of the Prostate
* E, Apr. 5, 9.00 a.m.-11.00 a.m.
- Tokuji ICHIKAWA
(Prof. of Tokyo University)
- Treatment of Epilepsy and Involuntary Movements (from the Standpoint of Stereotaxic Destroying Operation)
* K, Apr. 5, 9.00 a.m.-11.00 a.m.
- Chisato ARAKI
(Prof. of Kyoto University)

- Present Status of Research in Epilepsy Haruo AKIMOTO
(Prof. of Tokyo University)
* K, Apr. 5, 11.00 a.m.-12.30 p.m.
- Present Status of Open Heart Surgery Shigeru SAKAKIBARA
(Prof. of Tokyo Women's Medical College)
* J, Apr. 5, 9.00 a.m.-10.00 a.m.
- Rheumatic Arthritis Genichiro SHIMIZU
(Director of Osaka Welfare Pension Hospital)
* J, Apr. 5, 10.00 a.m.-12.30 p.m.
- Motion Sickness Takatoshi HASEGAWA
(Prof. of Osaka University)
* G, Apr. 5, 9.00 a.m.-10.00 a.m.
- Operation for Improving Hearing Shuji GOTO
(Prof. of Nagoya University)
* G, Apr. 5, 10.00 a.m.-12.30 p.m.
- Pancreatitis Shingo AOYAMA
(Prof. of Nagoya University)
* B, Apr. 5, 9.00 a.m.-11.00 a.m.
- Problems in Detection Drives for Diabetes Mellitus Yoshito KOBAYASHI
(Prof. Emeritus of Tokyo University)
* B, Apr. 5, 11.00 a.m.-12.30 p.m.
- Arteriosclerosis Hideo UEDA
(Prof. of Tokyo University)
* C, Apr. 5, 10.00 a.m.-12.30 p.m.
- Production and Destruction of Blood Cells Gyoichi WAKISAKA
(Prof. of Kyoto University)
* M, Apr. 5, 9.00 a.m.-11.00 a.m.
- Blood Transfusion and Circulating Antibodies Tadashi MATSUHASHI
(Assistant Prof. of Tokyo University)
* M, Apr. 5, 11.00 a.m.-12.30 p.m.
- Studies on Anterior Poliomyelitis Yoshito NISHIZAWA
(Prof. of Osaka University)
* H, Apr. 5, 9.00 a.m.-10.00 a.m.
- Enterovirus and its Clinical Picture Minoru TATSUMI
(Prof. of Osaka Medical College)
* H, Apr. 5, 10.00 a.m.-12.30 p.m.
- Development of Correctional Medicine and its Prospect in Japan Masao OTSU
(Research and Training Institute, Ministry of Justice)
* D, Apr. 5, 9.00 a.m.-10.00 a.m.

- Radiation Injuries
* N, Apr. 5, 9.00 a.m.-11.00 a.m. Moriji FUJINO
(Prof. of Osaka City University)
- Several Problems of Cardio-pul- Takashi NAKAMURA
monary Function Prof. of Tohoku University
* O, Apr. 5, 9.00 a.m.-11.00 a.m.
- Fine Structure of Microorganisms Tsunesaburo FUJINO
* D, Apr. 5, 10.00 a.m.-12.30 p.m. (Prof. of Osaka University)
- Vegetation of Poxvirus Juntaro KAMAHORA
* N, Apr. 5, 11.00 a.m.-12.30 p.m. (Prof. of Osaka University)
- Current Aspects of Lipid Nutri- Morio YASUDA
tion (Prof. of Hokkaido University)
* P, Apr. 5, 9.00 a.m.-11.00 a.m.
- Biological Significance of Oxy- Osamu HAYAISHI
genases (Prof. of Kyoto University)
* P, Apr. 5, 11.00 a.m.-12.30 p.m.
- Microelectrode Study of the Tsuneo TOMITA
Retina (Prof. of Keio University)
* L, Apr. 5, 9.00 a.m.-10.00 a.m.
- Neural Mechanism of Hearing Yasuji KATSUKI
* L, Apr. 5, 10.00 a.m.-11.00 a.m. (Prof. of Tokyo Medical and Dental
University)
- Studies in the Biochemical Shohei ISEKI
Genetics of Blood Group Sub- (Prof. of Gunma University)
stances
* L, Apr. 5, 10.00 a.m.-11.00 a.m.
- Hemoglobinopathy in Japan Susumu SHIBATA
* L, Apr. 5, 11.00 a.m.-12.30 p.m. (Prof. of Yamaguchi Prefectural
Medical School)
- Extermination and Prophylaxis Rikio YANAGISAWA
of Parasites in Japan, especial- (Prof. of Chiba University)
ly Ancylostoma
* I, Apr. 5, 9.00 a.m.-10.00 a.m.
- Pathology of Melanin and Taro KAWAMURA
Melanocyte (Prof. of Tokyo University)
* I, Apr. 5, 10.00 a.m.-11.00 a.m.

- The Actual Condition and Countermeasures to Acoustic Trauma in Traffic Work
 * Q, Apr. 5, 9.00 a.m.-10.00 a.m. Masao KUBO
 (Osaka Railways Hospital)
- Fatigue Problem under Automation
 * Q, Apr. 5, 10.00 a.m.-12.30 p.m. Shigemi KIRIHARA
 (Institute for Science of Labour)
- Angina Pectoris and Myocardial Anoxia
 * C, Apr. 5, 9.00 a.m.-10.00 a.m. Richard J. Bing
 (Chairman, Wayne State University)

Afternoon

- Cancer of the Esophagus
 * F, Apr. 5, 1.30 p.m.-4.00 p.m. Kômei NAKAYAMA
 (Prof. of Chiba University)
- Leukemia
 * E, Apr. 5, 1.30 p.m.-4.00 p.m. Susumu HIBINO
 (Prof. of Nagoya University)
- Formation and Treatment of Gallstones
 * K, Apr. 5, 1.30 p.m.-4.30 p.m. Hiroshi MIYAKE
 (Prof. of Kyushu University)
- Surgery of Congenital Malformations of the Digestive Tract
 * K, Apr. 5, 2.30 p.m.-4.00 p.m. Yoshiaki TAKEDA
 (Prof. of Osaka University)
- Rheumatic Fever
 * J, Apr. 5, 1.30 p.m.-4.00 p.m. Hideo NAGAI
 (Prof. of Kyoto University)
- Plastic Surgery; with special reference to Acquired Deformities
 * G, Apr. 5, 1.30 p.m.-4.00 p.m. Seiichi OHMORI
 (Lecturer of Tokyo University)
- Vascular Complications in Diabetes Mellitus
 * B, Apr. 5, 1.30 p.m.-4.00 p.m. Tsuneo YOSHIDA
 (Prof. of Osaka University)
- Physiology, Pathophysiology and Morphology of the Kidney
 * C, Apr. 5, 1.30 p.m.-4.00 p.m. Kenzo OSHIMA
 (Prof. of Nihon University)
- Blood Transfusion
 * M, Apr. 5, 1.30 p.m.-4.00 p.m. Noboru FUJITA
 (Prof. of Kobe Medical College)

- X-ray TV and X-ray Movies
 * D, Apr. 5, 1.30 p.m.-4.00 p.m.
 Toyoji MATSUKURA
 (Prof. of Osaka University)
- Yoshihiko KOGA
 (Prof. of Tohoku University)
 Acute Cardiac Death
 * O, Apr. 5, 1.30 p.m.-4.00 p.m.
- Myopathies and Other Medical, Neurological Disorders, Clinical and Epidemiological Aspects
 * H, Apr. 5, 1.30 p.m.-2.30 p.m.
- Shibanosuke KATSUKI
 (Prof. of Kyushu University)
- Mechanism of Virus Multiplication
 * N, Apr. 5, 1.30 p.m.-4.00 p.m.
- Konosuke FUKAI
 (Prof. of Osaka University)
- Clinical Application of Enzymology
 * P, Apr. 5, 1.30 p.m.-4.00 p.m.
- Haruhisa YOSHIKAWA
 (Prof. of Tokyo University)
- Hereditary Metabolic Abnormality
 * L, Apr. 5, 1.30 p.m.-4.00 p.m.
- Katashi ICHIHARA
 (President of Wakayama Medical College)
- Malformations
 * I, Apr. 5, 1.30 p.m.-4.00 p.m.
- Tameyoshi BABA
 (Prof. of Osaka City University)
- Population Problems in Asia
 * Q, Apr. 5, 1.30 p.m.-4.00 p.m.
- Takemune SODA
 (Institute of Public Health)
- Problems of Renal Tumor
 * E, Apr. 5, 11.20 a.m.-12.30 p.m.
- Einar Ljunggren
 (Prof., Göteborgs Universitet)
- The Closing Ceremony
 * F, Apr. 5, 4.00 p.m.-5.00 p.m.

Foreign Speakers in Symposia

(Title of Symposia)	(Foreign Speakers)
1. Blood Transfusion	Cater M. Ballinger (Assistant Prof., Univ. of Utah, U.S.A.)
2. Cirrhosis and Carcinoma of the Liver	Natth Bhamarapravati (Assistant Prof., Siriraj Hospital Medical Coll., Thailand)
3. Hypothermia in the Field of Surgery	Antonio Boba (Associate Prof., Albany Medical Coll., U.S.A.)
4. Rheumatoid Arthritis	F. Coste (Medicin des Hôpitaux de Paris, France)
5. Muscle Relaxants and the Nature of the Neuromuscular Junction Facilitation and Inhibition in the Nervous System	Sir John Eccles (Prof., Australian National Univ., Australia)
6. Acute Cardiac Death	Richard Ford (Prof., Harvard Univ., U.S.A.)
7. Clinical Aspects of Steroid Hormones	Peter H. Forsham (Prof., Univ. of California, U.S.A.)
8. Coronary Circulation and Myocardial Metabolism	W. H. Hauss (Prof. des Univ. Münster, Deutschland)
9. Secretion and Absorption: from the Morphological Aspect of Cytology	G. C. Hirsch (Prof., Univ. of Göttingen, Deutschland)
10. Pharmacotherapy of Mental Disorders	P. Kielholz (Prof., Univ. of Basel, Switzerland)
11. Several Problems of Cardio-pulmonary Function	R. Oberholzer (Prof., Univ. of Zürich, Switzerland)
12. The Relationship between Metabolism and Action of Drug	J. H. Quastel (Prof. & Director, McGill Univ., Canada)
13. Action of Drugs and Cell Membrane	M. H. SeEVERS (Prof., Univ. of Michigan, U.S.A.)
14. Sports and Physical Control	Aurthur H. Steinhaus (Prof., George Williams Coll., U.S.A.)
15. Hypothermia in the Field of Surgery	Robert W. Virtue (Prof., Univ. of Colorado, U.S.A.)
16. Radiation Therapy of Malignant Tumors	F. Wachsmann (Prof., Univ. of Erlangen, Deutschland)

17. Action of Drugs and Cell Membrane
W. Wilbrandt
(Prof. Univ. of Bern, Switzerland)
18. Recent Development and Medical Application Physico-chemical Biology
S. Filliti-Wurmser
(Univ. de Paris, France)
19. Muscle Relaxants and the Nature of the Neuromuscular Junction
Eleanor Zaimis
(Prof., Royal Free Hospital, England)
20. Biosynthesis of Proteins
Paul C. Zamecnik
(Prof. of Harvard Univ., U.S.A.)
21. Immunochemistry of Biologically Active proteins
Elvin A Kabat
(Prof., Columbia Univ., U.S.A.)
22. Several Problems of Cardio-pulmonary Function
James K. Alexander
(Assistant Prof. of Baylor Univ., U.S.A.)

Abstracts

Special Lectures

by guest speakers

Symposia

Special Lectures by Guest Speakers



Sir C. H. Andrewes

Common Cold Research Unit, Salisbury

Common Cold Viruses

In this lecture I shall first describe the work leading up to the successful cultivation of Common Cold or Rhinoviruses; second, I shall say something about the properties of these and some other viruses associated with colds: finally I shall discuss the effect of cold and of season on the incidence of colds and shall consider the prospects for prevention of colds.

I. Work at the Common Cold Research Unit at Salisbury.

In 1946 a unit for studying the common cold was set up. Its primary object was to discover a laboratory method for identifying and studying viruses causing common colds. To attain this end, it was necessary to use the only technique available for detecting the virus: dropping material up the noses of volunteers and noting whether they developed colds. The work has involved tests on more than 7,000 volunteers. We met with no success in attempting to grow the virus in eggs nor in infecting experimental animals. Some facts, however, came to light about the properties of the active agent and methods of transmission.

In 1953 we cultivated an agent (DC strain) in cultures of human embryonic lung; colds were produced with material up to the 10th serial culture. Then we failed to confirm our own results and spent several years vainly trying to do so.

In 1959 Tyrrell and his colleagues at the unit discovered that viruses could be grown from infective cold secretions in human embryonic kidney cultures, kept rotating in a roller drum at 33°C, at a rather acid pH. A few strains could be cultivated also in monkey kidney cultures (M strains), while most (H strains) could not. Now it has been found that many of these H strains will grow in diploid lines of cells derived from human lung; so fresh supplies of human embryonic tissue are not necessary. Further, the 1953 DC strain of cold viruses has now been grown in these diploid cells.

II. Properties of Rhinoviruses and Others.

We have called these agents Rhinoviruses or Nose-viruses. They are small, ether resistant viruses, having the same basic properties as Enteroviruses (Polio-myelitis, Coxsackie and Echo). They differ from these in their special requirements as regards temperature and pH in culture, in their greater liability towards acid, in their habitat and clinical manifestations. There are, however, viruses with intermediate properties-the ECHO 28 and Coxsackie A 21 (or Coe) viruses. The name Picornavirus has been proposed to cover the whole group of very small ether-resistant RNA viruses; this includes Enteroviruses, Rhinoviruses and many viruses pathogenic for animals such as that of Foot-and-Mouth disease. Pico is a prefix meaning very small and RNA indicates the type of nucleic acid composition. Other viruses-(para-influenza, respiratory syncytial etc.) causing some colds will be discussed.

III. Chilling and Season in Relation to Colds.

Many people believe that chilling will induce colds; we have been unable to confirm this experimentally. Undoubtedly colds are particularly frequent in winter. It does not seem that this is a direct effect of temperature, humidity nor of greater overcrowding in winter. Possibly sudden drops in temperature upset the nasal mucosa, thus making it more vulnerable.

Prospects for preventing colds with vaccines would be quite good were it not that there seem to be very many serological types of Rhinoviruses. It may be more profitable to study the natural history of colds, to discover the cause of their seasonal incidence and thus learn how to lessen the trouble caused not only by Rhinoviruses but by other respiratory viruses also.



E.B. Astwood

*Pratt Clinic, New England Center Hospital
and the Department of Medicine. Tufts
University School of Medicine, Boston,
Massachusetts*

Obesity as A Disorder of Metabolism

Currently, widespread opinion holds that obesity is merely a manifestation of gluttony and that the overeating is consequent upon an emotional disturbance. That dietary therapy is so difficult suggests a deeper cause, and the findings that psychotherapy is of little use and psychoanalysis ineffectual indicate a non-psychological etiology.

Experimentally, obesity has been induced in a variety of ways. Mechanically induced hypothalamic lesions have led to obesity in rats, mice, dogs, monkeys and man, and similar lesions induced by gold thioglucose cause obesity in mice and hamsters. Enforced inactivity has led to overweight in rats, cattle and geese and forced feeding has induced a similar effect. Endocrine manipulation, castration, thyroidectomy, corticoids and insulin have induced excessive weight gain but probably the most striking obesity is that determined by genetic causes.

Artificial selection led to the obesity of the domestic pig and certain breeds of dog. Mutation has led to the development of three types of obesity in the genetic cause in man as well.

Recent knowledge of the metabolism of fat suggests that any one of a number of possible enzymatic or hormonal defects could lead to obesity by impairment of the use of fat for energy. Fat tissue is highly active; calories eaten are quickly converted to fat and deposited. The fat stores constantly release free fatty acids which are carried to the muscles and organs to provide energy. Any excess of free fatty acid in the circulation is converted to triglyceride in the liver and transported back to the depots in that form. Various hormones strongly influence these events.

As free fatty acid is the main source of energy, any defect in its ready access to tissue would lead to hunger, and consequently, obesity.

Release of free fatty acid from depot fat, a crucial step, is under hormonal and neural control. It is stimulated by catecholamines in all species studied both

in vivo and in vitro. Corticotropin has a similar effect, while in certain species such as the rabbit both α - and β -melanophore hormones are active. Recently we have isolated two peptides from the pituitary of pig and of man which are potent fat-mobilizing agents which differ from known pituitary hormones. Unlike other factors stimulating mobilization these peptides lead to lipemia when given in large doses.

Mobilization of fat is strongly inhibited by glucose and insulin and doubtless insulin plays a central role in regulation of the use of fat. The adrenal corticoids are essential in a permissive way for some of these processes and growth hormone seems to exert an indirect stimulus to mobilization.

Some patients with extreme obesity have shown evidence of reduced capacity to release free fatty acid. Definition of the defect in these and other cases of obesity might lead to the development of effective therapy.



A. Leslie Banks

*Department of Human Ecology,
University of Cambridge*

Summary of Lecture on the Social Aspects of Disease

In ancient times the pattern of disease throughout the world was uncontrolled by man. The mortality in infancy and childhood was high, the average expectation of life was short, and war, pestilence, and famine, combined to make the growth of populations slow and sometimes to remain stationery or decline.

Today, in certain countries, the average expectation of life from birth is seventy years or more, the principal causes of death are the cardiovascular disasters and the cancers, and the increasing numbers of old people are causing concern. In other countries the control of the great killing diseases is resulting in rapid expansion of populations with resulting "land hunger" and exacerbation of the diseases associated with malnutrition. With the speed and complexity of life in highly industrialised communities the problems of death and disability due to accidents are serious and mental illness is beginning to receive as much, or more, consideration as physical disease.

How have these changes come about? It was not until some 400 years ago that the possibility of controlling disease and thus lengthening the span of life began to receive serious consideration, and it is only within the past 150 years that this has proved to be practicable.

For the purpose of discussion it is convenient to divide man's environment into three parts, physical, biological and social. The first active steps taken were

directed towards the improvement of the physical environment, by the purification of water sources, the better disposal of excreta, the improvement of dwelling houses and the control of overcrowding, by the supervision of the quality and quantity of food, and by the enforcement of proper standards for working conditions. This stage is sometimes referred to as "the era of sanitation".

Meanwhile the discoveries in pathology and bacteriology, in which Japanese scientists have played a most important part, made it practicable to control the biological environment by means of a direct attack on the great killing infectious diseases. That process, which began in the second half of the last century, is still continuing, with the result that it is now possible to provide protection against nearly all the epidemic diseases.

During the present century it has become apparent that the third component of the environment, that is, the social environment, is of the greatest significance. Every country has come to recognize that the community must accept responsibility for the health and welfare of its members and thus we may see the increasing development of medical and health services sponsored by local and national governments and designed to prevent disease, to cure the sick, and to restore the disabled speedily to their former place in society. The most recent extension of this process has been the establishment of the great international agencies, such as the World Health Organization, charged with improving the health and well-being of all men regardless of race, creed, or colour.

In general it would be true to say that members of the medical profession have been so preoccupied with the diagnosis and treatment of the sick individual that the social developments of the present century have taken them by surprise and they now find themselves increasingly involved with the machinery of government which they do not fully understand.

The purpose of this lecture is to examine these various developments in detail, with particular reference to the administration of health and welfare services, the growth of ancillary and paramedical workers, and the part to be played by the medical profession in the years to come.



Richard J. Bing

Wayne State University, Michigan

Angina Pectoris and Myocardial Anoxia

In this lecture the artificial production of myocardial infarction will be discussed. The release of enzymes by the heart muscle will be mentioned with particular reference to phosphocreatine kinase. The measurement of coronary reserve will then be discussed. Particular emphasis will be placed on the myocardial clearance of Rubidium-84, a positron emitting isotope. A coincidence counting system is being used. The method will be demonstrated and the effect of various factors on coronary blood flow as measured with the coincidence counting system will be discussed. Special emphasis will be placed on the effect of nitroglycerin, Persontin and emotion. The role which anoxia plays in the production of angina pectoris will then be discussed with a discussion of the calculation of the oxidation-reduction potential of heart muscle as determined by the difference in redox potential between coronary vein and arterial blood.



Viking Olov Björk

University Hospital Uppsala, Sweden

Problems in the Surgical Treatment of Aortic and Mitral Valves Insufficiency

1. Treatment of aortic insufficiency without prosthetic material.

A. Pure aortic insufficiency can be treated by internal bicuspidalization only if one cusp is normal.

B. In aortic insufficiency with combined stenosis. Commissurotomy with or without removal of calcium will only increase an already present incompetence in the aortic orifice.

2. Treatment of aortic insufficiency with teflon cusp prosthesis.

Individual teflon cusp prosthesis according to Bahnson and the total prosthesis of teflon as designed by Muller was used in 13 cases.

Although the ability to obtain completely competent valves is most encouraging the thickening with decreased mobility as well as the rupture of one prosthetic cusp after one year demonstrates that better material must be searched for aortic valve replacement.

3. Treatment of aortic insufficiency with a ball valve prosthesis (Starr)

In a series of 20 patients a ball valve prosthesis was used. Eight cases are surviving showing such improvement and reduction in heart size that the method has encouraged to continued use after improvement of the ball valve prosthesis. All patients are kept on anticoagulant treatment.

The surgical technique for aortic valve replacement.

The patient is operated in the supine position with a midline sternum split incision. The left main coronary artery is dissected and a tape is placed around it in the same manner as around both cava. The heart-lung-machine with the spinning disc oxygenator is started with a very small flow, usually one litre per minute for the first minute until a clamp has been placed over the ascending aorta. Once the aorta is occluded the perfusion rate is increased to a calculated value of 2.2 litres per sqm body surface area. From a sidebranch in the arterial line blood is pumped through a catheter and a special cannula, which is introduced in the left coronary orifice and left there during the whole of the procedure, kept in place by the tape which is tightened. The average perfusion time for aortic valve replacement has been 100 minutes. Primary tracheostomy with postoperative respirator treatment is used in all cases for the first week.

Franz Büchner

Universität Freiburg, Freiburg i. Br.

Regeneration, Hyperplasia, and Cancerization

One of the central subjects of modern pathology is the comparative study of metabolism and structure. For a longer period of time already the pathology has connected morphology and biochemistry by studying the deposition of certain substances in organic structures. In this manner the attempt has been made to determine the *amount* of substances in the cytoplasm of parenchymal and mesenchymal cells or in the mesenchymal ground substance. To-day such depositions of substances can be measured in quantity, at least partially, by cytophotometrical methods.

Further, the morphological pathology is since recently particularly interested in the turnover of substances in the cells as well as in the interstitial space. This means, the pathologists are nowadays interested in the question of the intensity of the turnover of certain substances—organic substances in particular—in a certain tissue and its cells. By such investigations the morphological pathology tries to get specific statements on the *dynamics of metabolism*.

The method used for this purpose is mainly the radioautography. Radioactively labeled precursors of organic substances are injected intravenously or intraperitoneally and are in a short period of time equally distributed over the whole organism. They become incorporated into cells or intercellular structures at sites where during the disposal time a synthesis is in action which can utilize the labeled substance. Then microscopic sections of organs are made and stained slightly with the usual staining methods. The mounted sections are covered by a photographic emulsion. The disintegration of the radioactive isotopes, which are incorporated into the tissue, causes in the overlying emulsion black silver granules.

The picture is especially well marked if the soft radiation of tritium is used. By these methods we have studied the turnover of DNA, RNA, and protein in the physiological regeneration, the pathological regeneration, and during the cancerization.

In the *physiological regeneration*, the granular blackening following injection of Tritium-Thymidine—a labeled precursor of DNA—can be traced in the nuclei of the indifference zone of the epidermis, the squamous epithelium of mucous membranes and the mucous membranes of stomach and bowel.

These nuclei belong to those cells which alone are able to mitotic division because of their relatively undifferentiated structure. One of the daughter cells always develops endoplasmatic reticulum, that is specific structure proteins. Consequently one of the two daughter cells becomes inactive for mitosis. This means, the formation of structure proteins brings the reduplication of DNA, acting as a feed back system, to a stop. If the animals are sacrificed not before 24, 48, or 72 hours, one notices that one of the labeled daughter cells travels after the division to the surface and is there pushed off.

In the normal parenchyma of the liver DNA reduplication very rarely occurs. In the *pathological regeneration* after resection of a part of the liver, a reduplication of DNA is to be seen in the peripheral epithelium cells of the liver lobules after 24 hours. After 32 hours this reduplication of DNA is also to be found in the intermediate zone of the liver lobule and after 36 hours in the whole lobule. 48 hours after resection, that is after restitution of the normal number of cells, the DNA reduplication ceases again. Similar DNA reduplications can be observed in the epithelium of the tubules of a damaged kidney after necrosis caused by sublimate, and in the epithelium cells of the liver parenchyma after necrosis caused by Thioacetamide.

During the *cancerization* of the epidermis in mice by Methylcholanthren the reduplication of DNA increases in the indifference zones, that is in the basal layer of the epithelium, already after the first painting. As is well known, during further painting a growing hyperplasia of the epidermis is to be observed. In this hyperplastic epidermis the DNA reduplication is also markedly increased, but exclusively in the basal indifference zone. This can even be observed when further painting is discontinued. If with further painting a pillomas develop, then these too have a marked increase in DNA reduplications but also only in the basal indifference zone.

On the other hand, the DNA reduplication is to be observed in the upper layers of the epidermis too, shortly before the carcinoma develops. This occurs in those groups of cells which can be spotted by the ordinary light microscope as reticulation zones. Moreover, there is a still greater increase of DNA reduplications in the invasively growing cancer except for the horn pearls after the beginning of cornification of the squamous cell carcinoma.

A similar increase of DNA reduplications is to be met with in the parenchyma of the rat liver after Diethylnitrosmine. This happens during the advanced stages particularly in the centre of the lobules, where after 130 days the

microcarcinomas are to be seen.

During the cancerization of the epidermis and the liver parenchyma as well, there also occurs a marked raise of the RNA and protein synthesis. During regeneration after partial resection of the liver as well as after Thioacetamide, but most distinctly during the cancerization, there is a particularly pronounced hydrops of part of the parenchymal cells of the liver to be observed. These epithelial cells, which show a marked alteration of their cytoplasm, are at the same time the site of a raised RNA synthesis in the nucleus, whereas the RNA in the cytoplasm diminishes and the DNA reduplication increases.

This alteration corresponds electronic-microscopically to an increasing destruction of structured ergastoplasma and the adjacent ribosomes as well as to the formation of a vesicular not structured ergastoplasma, whereas the mitochondria stay unaltered. This fact corresponds well with the statement of biochemists that carcinogenic substances are to be found in the microsome fraction, that is in the fragments of the structured ergastoplasma. On the other hand the structured ergastoplasma resembles the specific structure protein with the adjacent ribosomes. After its destruction and the destruction of the RNA the feed back system between cytoplasm and nucleus is disturbed, reversible in the Pathological regeneration, irreversible in the cancerization. This feed back system causes the discontinuation of DNA reduplication, if the content of structure protein and RNA in the cytoplasm is a physiological one. When the structure proteins and the RNA are destroyed, this substance is abolished and the DNA will be reduplicated. If, during the cancerization, in the cytoplasm a normal organ-specific structure protein and RNA no longer are being formed, then the DNA reduplication will go on, whilst the intermitotic time is markedly shortened. There occurs a pathological increase of the mitoses and henceforth the invasive growth of the malign anti-tumor.



E.V. Cowdry

Washington University, St. Louis

Influence of Aging on Incidence and Malignancy

Though age measured, the incidence of cancer may depend on carcinogenic exposure more than upon age, while the operation of promoting and inhibiting factors alter the situation. Thus, the fetus *in utero* is not exposed to many carcinogens which are effective in later life, but is exposed to a few carcinogens of maternal origin *in utero* which are not effective in later life. Excessive sunlight is an example. There remain, however, numerous instances of the age related incidence of cancer which are not simply explainable as due to differences in carcinogenic exposure. In some cases one and the same kind of cancer regularly exhibits two widely separated peaks in age of maximum incidence. Incidence is partly a factor of susceptibility which is not readily explained as simply a factor of age. Susceptibility is not conditioned solely by the percentage of cells in mitosis. Aging itself is more rapid in some tissues than in others. Latent periods generally increase in length as a function of length of life span, but there are exceptions. The incidence of precancerous lesions seems also to be related to the life span.

To detect the age factor almost exclusively, *per se*, it is necessary to equalize exposure of individuals of various ages. This is feasible only to a limited extent as body chemistry can be divergent. Such human experimentation is unjustified. Our experiments with New Buffalo mice have shown that more cancers develop more quickly in young than in old mice equally exposed to methylcholanthrene. In CBA mice some hereditary factor must suppress this youthful of this strain respond in the same way to this carcinogen. Action of the A.Y. gene in promoting lung tumors in mice can be suppressed by dietary restriction

(Heston and Blahkis, 1961). This suggests that hereditary susceptibility can possibly also be checked in human beings. Probably genes also exist that decrease the incidence of some cancers.

An age factor exists in malignancy. With increase in age breast tumors and some others exhibit a decrease in malignancy measured by an increase in percentage of 5 year survivals, by decrease in metastases and aggressive behavior. Many carcinoma-looking lesions in the prostates of very old men exhibit little if any aggressive activities. Contrarywise, some melanomas in children are benign compared with those in adults. How much such differences in malignancy depend on increases or decreases in promoting and or inhibiting factors is not known. Qualitative or quantitative changes in latent cancer viruses are also wholly unknown but may be influential.

In assessing the influence of aging on cancer influence and malignancy it should be realized that age factors operate on at least two major levels; on susceptible cells themselves and on the larger level of tissue protection against them. Each level poses different complications and qualifications in reaching conclusions. Moreover, there are stages in carcinogenesis peculiar to the several tissues and in phases of malignant behavior some of which only may be influenced by age factors.



Harry F. Dowling

University of Illinois

Some Problems in the Chemotherapy of Infectious diseases

The wide use of chemotherapeutic agents in the treatment of infectious disease and the increase in the number of such agents available have increased the problems of chemotherapy for the practicing physician. Selection of the proper drug for staphylococcal infections requires knowledge of the newer penicillins: methicillin, oxacillin, and ampicillin. The two former drugs are fulfilling their promise as antistaphylococcal agents, and so far no proven case of increasing resistance of the *Staphylococcus* under therapy is on record. Ampicillin is an addition to the group of drugs effective against the gram-negative rods. It is effective in a fair percentage of cases of urinary tract infections; its role in *Salmonella* infections is yet to be clarified.

Fewer mixtures of antibiotics are being promoted than were advocated several years ago. The only one for which there is any rationale is the combination of a tetracycline with nystatin or amphotericin. Combined therapy with two or more antibiotics is indicated in endocarditis caused by streptococci, in tuberculosis, and in occasional cases of serious infection caused by staphylococci and gram-negative rods.

Little progress has been made in diminishing the number of analogues of antibiotics that have the same pharmacologic effect as the original drug in the group. Thus, there is little difference among the analogues of tetracycline; new sulfonamides continue to appear; analogues of neomycin and of erythromycin make the practicing physician's task difficult, and a host of new penicillins are apparently being developed with actions similar to those of methicillin.

There is increasing evidence that chemotherapeutic agents are of some value in viral disease of animals; it is possible that effective agents for use in man may be developed in the near future.

Another continuing problem is the prevalence of untoward reactions to antibiotics. Deaths are most frequent as a result of anaphylactic reactions to penicillin and blood dyscrasias produced by chloramphenicol. Elimination of these reactions depends upon education of the profession. Reactions continue to be discovered that result from therapy with drugs that have been used for some time, such as the hepatitis that follows the use of erythromycin estolate and oleandomycin.

With respect to the emergence of antibiotic-resistant microorganisms, interest is shifting from the staphylococci (probably because they are so well controlled by the newer penicillins) to the gram-negative rods. The latter have been shown to spread in the hospital in the same way that staphylococci do, and again as in the case of staphylococci, certain strains of *Escherichia coli* seem to predominate in hospital patients. The placement of synthetic substances within the heart and blood vessels has increased the frequency of infections caused by microorganisms that in the past have been considered to be non-pathogenic.



Seymour M. Farber

University of California

The Impact of Continuing Education on Teaching, Research and the Practice of Medicine

The advance of both technical aspects and concepts of medicine has enormously increased in tempo since 1930. The need for understanding the body of new information is such today that no physician can remain out of contact with advanced medical thought for more than a year or two and still be able to give of his best to his patients. This acceleration implies that the need for post-graduate education is greater now than it was 10 years ago; 10 years from now the satisfaction of this requirement for best medical practice may well become critical.

There are many direct and indirect approaches to continuing education in medicine and the health sciences. The specialty societies meet regularly, but there is too much to discuss in too little time; inevitably presentations must lack the ordered sequence and complete discussion of ideas necessary for full comprehension of information. The voluntary health agencies such as the tuberculosis or heart associations in many cases have been interested in education of the physician. A voluntary agency is naturally concerned with the disease entities for which it takes a community responsibility and therefore cannot easily present the whole implications of new material. Professional societies endeavor in a sincere fashion to maintain the highest educational standards at scientific meetings; the very multiplicity of needs of their membership make this task most difficult.

That body not only most detached from pressures, but most intimately involved in the advance of medicine, is the Medical School and Medical Center. Through Departments of Continuing Education acting independently and in

collaboration with the groups previously mentioned, it is possible to arrange basic educational experiences which cannot but have the greatest impact on the practice of medicine. The broad vision within a medical faculty can act as a major factor in the best practice of medicine in the community at large. Moreover, the frequent contact of a medical school faculty with men practicing medicine daily in a community increases the breadth of understanding of the problems of medicine within the medical school itself.

Continuing Education in Medicine and the Health Sciences at the University of California Medical Center in San Francisco has had a dynamic evolution over the last eight years. An attendance of 500 for 10 general courses, has developed into an enthusiastic group of 12,000 doctors, dentists, nurses, ancillary health workers and lay public attending nearly 90 programs last year. These now range from specific demand programs in general fields, through specialized seminars into the broadest possible interdisciplinary program on important aspects of human well-being. The most recent development has been the exploration of the relationship of the humanities to medicine, an expanding field of interest.

From systematic evaluation of the reactions of registrants to our program, it is clear that an increasing acceptance and interest exists which is considered by those who attend to have an important influence on those engaged both in teaching and research, and in the daily practice of medicine.



Bernardo A. Houssay

*Institute of Biology and Experimental
Medicine, Buenos Aires*

Role of the Pituitary Gland on Metabolism

The hypophysis is one of the most important factors in the endocrine balance which regulates metabolism. Some of the pituitary hormones act directly on the peripheral tissues (somatotropin, prolactin); others act indirectly (adrenocorticotropin, thyrotropin, gonadotropin) through the effects they have on the internal secretions of the adrenals, the thyroid or the gonads.

Basal metabolism:

Thyrotropin exerts its effect by maintaining thyroid endocrine secretion. Hypophysectomy generally causes a decrease in thyroid output and in basal metabolism. Subsequent total thyroidectomy is followed by a further decrease in basal metabolism.

Carbohydrate metabolism:

The pituitary has a continuous effect on carbohydrate metabolism in normal and in diabetic conditions. In hypophysectomized animals intestinal absorption is retarded and the hyperglycemic curve after oral administration of glucose is lower than in normal animals.

In pituitary insufficiency carbohydrate levels are not maintained during fasting. Glycemia and glycogen content are normal after meals, but both diminish rapidly during fasting and hypoglycemic convulsions and death may occur. These accidents can be prevented by feeding carbohydrate or protein, but not by giving fats alone. They are corrected by the early administration of glucose and other sugars, or of adrenalin.

Hypophysectomized animals and men are highly sensitive to the hypoglycemic and toxic effects of insulin and other agents which provoke hypoglycemia. They are more sensitive to insulin than adrenalectomized animals.

Hyperglycemia provoked by adrenalin and other agents is less marked than in normal animals.

Extirpation of the hypophysis or of its anterior lobe (*pars distalis*) is followed in animals and man by marked attenuation of pancreatic and phlorhigin diabetes. This decrease in the severity of pancreatic diabetes caused by hypophysectomy is shown by:

- a) a decrease in hyperglycemia and glycosuria during fasting, in some cases the blood sugar falls to hypoglycemic convulsive levels;
- b) Hypersensitiveness to insulin;
- c) There is no ketosis, nor fatty liver and kidneys;
- d) Protein catabolism diminishes;
- e) The loss of weight is less marked than in animals with an intact hypophysis, and survival is prolonged. Tissues taken from these animals have a better glucose consumption, deamination and ketogenesis than those of pancreatectomized animals with an intact hypophysis.

Fasting hypoglycemia and the attenuation of pancreatic diabetes can not be attributed to a predominance of insulin because they are seen in totally pancreatectomized animals. They are caused by:

- a) an increase in glucose consumption, and
- b) the fact that during hypoglycemia there is no increase in the inflow of glucose from the liver to the blood, as occurs in normal animals.

All these disturbances produced by pituitary insufficiency can be prevented or corrected by the administration of pituitary hormones: somatotropin, adrenocorticotropin, prolactin (which is not so active), and by glucocorticoids. Large doses of these hormones produce disturbances in the opposite direction:

- a) resistance to insulin;
 - b) an increase in hyperglycemia produced by adrenalin and other substances;
 - c) fasting does not deplete glycogen stores, specially those of muscle.
- The role of the hypophysis in diabetes was demonstrated by:

- a) a decrease in severity of pancreatic diabetes after removal of the hypophysis or of its *pars distalis*;
- b) an increase in the severity of diabetes following injection of the *pars distalis* or its hormones:

There is a balance between the hypophysis and the pancreas as is shown by the following facts: 1) reduction of the pancreatic mass increases the diabetogenic effect of the hypophysis, and 2): removal of the hypophysis increases sensitiveness to insulin.

The intensity of pancreatic diabetes is conditioned by two factors: first, lack of insulin, and, second, the presence of pituitary and adrenal hormones, which have a synergic action, usually of reinforcement.

The diabetogenic action of pituitary hormones consists in:

- a) first, an increase in insulin resistance;
- b) a decrease in glucose tolerance and consumption;
- c) Hyperglycemia and glycosuria;
- d) an increase in fat (ketosis) and protein metabolism.

In normal animals (dog, cat, man, but not in rats), it is possible to produce diabetes by the administration of pituitary extracts or hormones. Insulin resistance first appears, then a hypophyseal or idiohypophyseal diabetes develops; there is hyperglycemia, glycosuria, ketosis, lesions in the B cells (degranulation, vacuolization) and insulin secretion diminishes. If the treatment is discontinued, the diabetic symptoms disappear and after a few days the B cells recover their normal aspect and secrete normally. If the treatment is prolonged irreversible damage is caused in the B cells and the diabetic state becomes permanent (metahypophyseal diabetes).

In the initial stages and in certain conditions somatotropin provokes a short lasting hypoglycemia and increase in the uptake of glucose (insulinoid effect). Later resistance to insulin and diabetic symptoms appear.

Hypophyseal diabetes can not be produced in the absence of the liver. Hepatectomy causes a rapid fall or the hyperglycemia. Hypophyseal diabetes can be produced in the absence of the thyroid, the gonads and sympathetic nervous system, but it is difficult to provoke in the absence of the adrenals. The hormones of the pituitary and the adrenals have a concurrent diabetogenic action.

An excess of pituitary hormones specially of somatotropin is probably the cause of the high frequency (19 to 32%) of diabetes in acromegaly.

Protein metabolism:

In the absence of the pituitary growth is interrupted or markedly decreased; there is less fixation of nitrogen and less formation of protein. The presence of the pituitary is not necessary for growth during fetal life (rabbits, rats) or the first weeks after birth, but later hypophysectomy suppresses growth in most animals.

Hypophysectomized animals catabolize well proteins and amino acids given them, but endogenous protein (in fasting, pancreatic diabetes etc.) is mobilized and catabolized subnormally.

Somatotropin produces supernormal growth, or giantism in rats. Only human or monkey somatotropin provokes growth (or diabetes) in human subjects with pituitary insufficiency. Somatotropin of other animal species is inactive.

Injection of somatotropin produces an initial fall in non-protein nitrogen and amino acids in plasma. Later there is fixation of nitrogen by the tissues and formation of proteins.

Some hypophysectomized animals (rats, etc.) develop anorexia, but nitrogen equilibrium can be maintained by forced feeding. In this case the animals do not grow but become obese.

Nitrogen fixation, increase in protein formation, and growth are not produced by somatotropin in the absence of insulin.

Fat metabolism:

In pituitary insufficiency body fat increases easily if sufficient food is available. Somatotropin produces an increase in body protein and water, and a decrease in body protein and water, and a decrease in body fat. Hypothalamic lesions in rats can provoke hyperphagia and obesity, even in hypophysectomized animals.

Somatotropin and adrenocorticotropin increase lipemia and the fat content of the liver and other organs.

Hypophysectomy prevents the appearance of ketosis and fatty liver in pancreatectomized and phlorhizinized animals (dog, rat and baboon). Ketosis is specially suppressed.

Intense ketosis observed in pancreatic diabetes is due to the absence of insulin and the simultaneously presence of pituitary and adrenal hormones.

The hypophysis plays an important part in the storage and transport of fats. An excess of hypophyseal (and adrenal) hormones causes depletion of fat stores, and increases fat transport and metabolism. In insulin insufficiency these hormones produce a great increase in ketone bodies.



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Concerning the Mechanism of Action of Insulin

The historical review of the field demonstrates that by about 1947 it was concluded that the effects of insulin could best be explained by an action on glucose phosphorylation. Further work did not substantiate this hypothesis. In 1949-1952 Levine and co-workers postulated that glucose entry into cells is rate limiting and that insulin increases the rate of the glucose transport system in tissues like muscle. The work was confirmed and extended by Ross, Randle, Park etc. The present evidence shows that most cells possess a specific sugar transport system at their membranes. However some tissues (brain, kidney, gut, rbc) do *not* react to insulin, while muscle, heart, adipose cells, fibroblasts are activated by insulin. Our working hypothesis at present is that the insulin sensitive tissues possess a "cover" for the transport system with which insulin combines, thus temporarily removing it.

The questions which will be discussed are:

1. Does this action of insulin account for all the phenomena seen when the hormone is given?
2. What is the action of insulin on liver, which does not have a sugar transport system?
3. What are the possible modes of action of the hormone at the molecular level?
4. What is the relation of insulin to other substances which activate sugar transport?
5. How do the hypotheses of Fisher, Randle, Ball and Chain fit in with the concepts outlined here?



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Neural Plasticity and the Memory Process

As a contribution toward understanding neural plasticity, the considerable body of information and hypothesis which has accumulated concerning the memory process has been reviewed.

Both clinical and experimental evidence points to the exceedingly important role of the hippocampus and entorhinal cortex of the temporal lobe in processing novel information into storage, as well as in its early consolidation and recall. Well-established memories are obviously stored more widely in the brain, however, for they are preserved and can be recollected after temporal lobectomy.

The ubiquitous pattern of electrical activity called the theta rhythm is an invariable expression of excitation of the hippocampal part of the brain. Its large-amplitude slow waves, which display characteristic alterations in pattern, frequency and distribution during the formation and recall of memories, are proposed to serve as phase comparators or carrier waves in the induction of memory traces.

The long duration of each theta wave, as well as its relation to graded response and steady potential mechanisms, suggests a temporal factor in memory processing considerably more prolonged than the brief duration of the classical nerve impulse. Additionally, evidence is accumulating for the excitable properties of glial cells, the time course of which is very much longer, as well as indications that the neuron and its surrounding glia form a kind of biochemical and functional micromodule.

Current advances in genetic biochemistry, pointing to the capacities of DNA and RNA for information-coding, specification and replication, now seem directly applicable to neural function as well. The Nissl substance of nerve cells has been found to be composed of myriads of RNA granules, which increase markedly during neural activity. Hydén has recently proposed: that induction of the memory trace may depend upon the specification of neural RNA by neuralglial

impulse patterns transducing environmental changes; that memory storage may involve the preservation of this RNA specification by replication during turnover in metabolism; and that memories may be recalled by neural-glial impulse patterns which provoke protein dissociation, transmitter-substance production and sequential excitation of neurons, in a manner like that induced by the original experience.

If these recent possibilities, which relate memory function to the nucleic acid metabolism of nerve cells, develop along the lines proposed, the consequences for understanding and improving the normal function of the brain, as well as for providing effective therapy when it is impaired, are likely to be as significant as those which are currently anticipated from nucleic acid studies in the field of genetics itself.



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An Examination of the Traditional Concept of Medical Care

A review of the trend of mortality during the past century, and of the current demands for hospital care, suggests that the outstanding problems with which medicine will be confronted in future are: prenatally-determined mortality, malformation and disability; mental defect and mental illness; and the disease and disability associated with aging. This conclusion is significant, since it is in respect of two of these problems—the care of the mentally ill and of the aged sick—that the medical services have hitherto been least successful.

The reasons for this failure are to be found in the history of the public medical services. For while the problems confronting medicine and the methods available for their solution have both changed radically during the past hundred years, the pattern of care has tended to remain relatively constant. In essentials this pattern was determined in a period when the main challenge was from infectious disease, and when the concept of public responsibility for health services was much more limited than it is today.

There are three features of the medical tradition which are significant in the present context: the separation of mental, chronic and acute hospitals; the isolation of domiciliary from institutional care; and the long-standing divorce between preventive and curative medicine. It will be essential to eliminate these features, and to plan a medical service appropriate to contemporary and future problems.

Questions for discussion

- (1) What should be the size of a hospital centre?
- (2) To what extent would plans for hospital centres in large urban areas have to be modified in small urban or in rural areas?

- (3) What is meant by the suggestion that a common staff should serve the centre? Would this affect all doctors and nurses or only selected groups?
- (4) Are plans for hospital development based on a contemporary analysis of need likely to be affected seriously by changing demands for institutional care?
- (5) In what respects do the proposals for "a balanced hospital community" differ from the hospital centre concept developed in the thirties?
- (6) To what extent are the proposals applicable in urban areas in view of limitations of sites and the nature of the existing hospital services?
- (7) What bearing have the proposals on medical and nursing education? Are the proposals applicable to teaching centres?



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Surgical Therapy of Frontal Lobe Epilepsy

Frontal lobe epilepsy is, in our experience, the second largest group of the focal epilepsies and is about half as frequent as temporal lobe epilepsy. Two hundred and fifty patients with seizures arising in the frontal lobe and intractable to medical treatment have been operated upon at the Montreal Neurological Institute in the period from 1929 through 1960. In 67 patients the lesion was neoplastic in nature and 183 patients non-neoplastic lesions were disclosed. This report concerns the latter group. Cerebral scarring and atrophy secondary to head injury, gun shot wound or surgical removal of brain abscess, tumor or hematoma accounted for 56% of these non-neoplastic lesions. Birth trauma (14%) and encephalitis (7%) were the next most common.

Accurate follow-up data were available in 168 of the 183 patients in the non-neoplastic group. The duration of the follow-up ranged from a minimum of 1 to a maximum of 31 years. The median follow-up period was 8 years. Follow-up data were complete up to the date of this analysis or to the patients' subsequent death in 123 patients (73%).

Fifty-five patients (33%) have become seizure-free. Half of these have had no attacks since discharge from the hospital, the other half have had a few attacks during the first few postoperative month or years and then have had none for the remainder of the follow-up period. Fifty of these patients (30%) have had a marked but incomplete reduction in their seizure tendency. Sixty patients (36%) have had less marked reductions in seizure tendency and are classed as unsatisfactory results.

Long-range follow-up studies in our patients operated on for relief of focal cerebral seizures indicate that as a rule the epileptogenic lesion consists of a sizable area with varying levels of epileptogenicity within it, rather than

being a single discrete discharging lesion. Removal of most of all of the epileptogenic area is necessary to abolish the seizure tendency. The fringe of a focal epileptogenic lesion may be so weakly epileptogenic, however, that once it is deprived of the activating effect of a neighbouring more intensely epileptogenic area, its epileptogenicity runs down over a period of a few months of years or is successfully suppressed progressively by the normal inhibitory functions of the rest of the brain.

The failures in this series seemed clearly to be due to inadequate reduction of the original seizure tendency and in no instance was there evidence of the surgical removal resulting in the later development of a new epileptogenic lesion. In this connection it is emphasized that the follow-up period was over 10 years in 67 patients, 40% of the series.

With the steady increase in automobile accidents and the high incidence of head injuries, and particularly frontal fractures which occur in such accidents, it seems highly probable that patients with focal epileptic lesions of the frontal lobes will constitute an increasing proportion of the epileptic population of the future. Until such time as completely effective anticonvulsant medical agents are available, surgical therapy can provide important benefits for a significant proportion of those patients whose seizure tendency cannot be adequately controlled medically.



Derek Richter

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Biochemical Research in Mental Illness

Some forms of mental disorder are associated with physical damage to the brain; but in many cases no structural damage can be found. It is known that certain chemical substances, such as ethanol and the hallucinogenic drugs can cause mental symptoms. This suggests the possibility that the mental symptoms found in some kinds of mental illness might be due to a metabolic disturbance or a 'biochemical lesion' in the brain. Certain links can be found between the behavioural abnormalities seen in mental illness and metabolic factors that can be recognized and defined. Mental symptoms occur, for example, in myxoedema and in infective conditions in which a bacterium or virus produces a toxic substance which acts on the brain.

Mental patients are characterized by behavioural symptoms which may result from a number of different causes. Some investigators have studied mainly the 'organic' physical and chemical causes, while others have been impressed by factors which operate at a psychological or social level. Others again have emphasized the part played by inborn hereditary disposition. These three types of causal factors are not mutually exclusive; there can be no case in which all three do not play a part in contributing to the mental disorder. The problem is to assess their relative importance in any particular condition.

The biochemical investigation of patients suffering from depression has shown that many have an abnormal retention of sodium, which is corrected when they recover. Should this be considered as a cause or a result of their mental state? Biochemical abnormalities have also been described in schizophrenics, and this is now an active field of investigation. Some biochemical characteristics appear to depend on factors such as diet and exercise rather than on the mental condition.

In trying to understand the operation of pathological factors, it is necessary to know more about the normal biochemical mechanisms of the brain. It used

to be believed that the brain is metabolically inert, but recent work has shown that it is metabolically active and in many respects unique. Of particular interests is the very active amino acid and protein metabolism of the brain. The conversion of glucose carbone into amino acids is many times more active in the brain than in other organs examined: this is due to the characteristic properties of enzymes of the Krebs cycle in the brain. The evidence suggests that the brain, which has little reserve of glycogen in relation to its metabolic activity, is specially adapted to utilize amino acids and proteins to provide the energy required for functional activity. The involvement of amino acids in the energy metabolism of the brain offers an explanation for the relatively high content of utilizable amino acids and the high rate of turnover of proteins in the brain.



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Physiologic Defects in the Genitourinary System

Anomalies of the urinary system below the kidney can be divided into two groups: first, those with mechanical obstruction, and second, those with defects in physiological function. Our established techniques of investigation are designed primarily to detect mechanical obstruction such as ureteropelvic block produced by a stricture or an aberrant renal vessel. The detection of defects in function is a much more difficult problem. Although beginnings have been made in the effort to study these patients, much remains to be accomplished.

The child with defects in physiologic function of some part of the urinary system usually presents with infection, while in small infants the presenting complaint may be failure to gain weight normally or failure to gain at all. When such patients are seen in our clinic, a more extensive urological investigation is performed than has been the custom in the past.

Our procedure in studying these patients is as follows: we catheterize the child for a residual urine, and it has been found that if this is more than about 15 cc. it is cause for some concern. Leaving the catheter in place, the child is taken then to the X-ray Department and contrast material is instilled slowly through the catheter into the bladder under a low head of pressure (18 cm. of water). Often the filling of the bladder is observed during this period, using cinefluorographic equipment. This enables us to make movies if reflux should be detected during the filling cycle. When the bladder is distended to a normal capacity for the patient's age, the catheter is removed. Infants usually will void simultaneously, permitting a movie to be made of the bladder outlet and urethra. The following day an excretory urogram is made to determine the status of the upper urinary tract. We also secure a clean voided specimen for culture and colony count. We have found this to be a great advantage, because

patients do not have to be catheterized in order to secure reliable cultures. This particular technique has proved to be extremely valuable in following patients with chronic urinary tract infections; because it obviates the necessity of repeated catheterization, it is a much safer procedure. In those patients with enlargement of the bladder, providing they are over 2½ years of age, a cysto-metrogram tracing is made to determine the status of the detrusor function of the urinary bladder. In patients with evidence of cord bladder, these tracings are extremely important. A cystoscopy is performed, and during this examination not only is the usual careful visual examination of the posterior urethra, bladder neck, and bladder done, but in addition a special catheter is inserted into each ureter and ureteral peristaltic tracings are made. A large number of normal children have been traced, so that the type of tracing is now quite standard. In certain situations, we find grave defects in ureteral peristalsis which are related to their disease. Also, during cystoscopy we insert a needle through the abdominal wall into the bladder and measure the resistance to voiding. In older patients, a small polyethylene tube is threaded into the bladder in order to secure tracings of bladder pressure during conscious voiding. The common finding in a large group of patients one studies is that they do not have mechanical lesions or definite defects in physiologic function.

The treatment of the various conditions detected by these methods will be discussed.



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Herz-und Gefässinsuffizienz- Hämodynamik und Herzstoffwechsel

Zwei grundsätzlich verschiedene hämodynamische Situationen können zu einer ungenügenden Blutversorgung der peripheren Organe führen, die Herzinsuffizienz und die Gefässinsuffizienz. Das Herzminutenvolumen ist bei der Herzinsuffizienz meist gegenüber der Norm verkleinert. Es kann aber auch innerhalb der normalen Grenzen oder sogar vergrössert sein (high output failure). Stets unterscheidet sich aber das insuffiziente vom suffizienten Herzen dadurch, dass der in der Norm mit 0.8 bis 0.85 gefundene Quotient aktive Blutmenge zu Herzminutenvolumen über 1.0, oft sogar bis 2.0 ansteigt. Für die Entstehung der Herzinsuffizienz können in 90% der Fälle bestimmte auslösende Ursachen nachgewiesen werden (Flimmerarrhythmie, körperliche oder psychische Belastungen, klimatische Einflüsse, Gravidität, Thyreotoxikose, hohes Fieber). Durch diese Ursachen der Herzinsuffizienz wird primär die aktive Blutmenge vergrössert. Zur Herzinsuffizienz kommt es, wenn das Herzminutenvolumen nicht entsprechend steigerungsfähig ist. Der Quotient aktive Blutmenge zu Herzminutenvolumen steigt über 1.0. Hierdurch wird ein circulus vitiosus ausgelöst. Die ungenügende Sauerstoffversorgung der Peripherie steigert das Missverhältnis zwischen Blutmenge und Herzminutenvolumen weiter hin. Die in der Kreislaupерipherie sich entwickelnden Folgen der Herzinsuffizienz werden als cardio-vaskuläre Dekompensation, und in diesem Fall wegen der zentralen Stellung des vergrösserten Blutvolumens als Plasdekompensation, bezeichnet. Die klinischen Erscheinungen dieses Dekompensationstypus werden besprochen.

Für die Entwicklung der bei jeder Plasdekompensation initial bestehenden Herzinsuffizienz lassen sich nach Untersuchungen meiner Mitarbeiter Hochrein und Nagano Stoffwechselveränderungen im Myokard nachweisen, die zur Kontraktionsinsuffizienz führen. Am Herzlugenpräparat des Meerschweinchens

wurde bei verschiedenen Arten experimentell ausgelöster Insuffizienz eine Verminderung des Gehaltes an ATP und Kreatinphosphat bei fast unverändertem ADP und vermehrtem Gehalt an anorganischem Phosphor und Milchsäure nachgewiesen. Gleichzeitig nimmt der intrazelluläre Kaliumgehalt des Herzmuskels ab, sein Natrium- und Wassergehalt nimmt zu. Hochrein und Nagono konnten ferner charakteristische Veränderungen der Fermente des Kohlenhydratstoffwechsels im insuffizienten Herzen nachweisen. Für den Übergang aus der Suffizienz in die Insuffizienz könnte gezeigt werden, dass die Abnahme des Kaliumgehaltes und die Zunahme des Natrium- und Wassergehaltes sich bei zunehmender Belastung am Herzlungenpräparat bereits während der suffizienten Phase entwickelt. Zwischen suffizienten und insuffizienten Herzmuskel besteht hier nur ein quantitativer Unterschied.

Unter Digitalisierung ist beim Gesunden und Kompensierten nur eine Abnahme der aktiven Blutmenge nachweisbar. Das Herzminutenvolumen kann gleichzeitig kleiner werden, oder es bleibt unverändert. Die Herzleistung kann beim Suffizienten oder Normalen unter Digitalis nicht verbessert werden. Beim insuffizienten Herzen dagegen nimmt bei Abnahme des aktiven Blutvolumens das Herzminutenvolumen zu. Derin der Insuffizienz erhöhte Quotient aktive Blutmenge zu Herzminutenvolumen wird dadurch normalisiert. Am Herzlungenpräparat lässt sich gleichzeitig zeigen, dass der in der Insuffizienz herabgesetzte Kaliumgehalt ansteigt und der abnorm hohe Natrium- und Wassergehalt verkleinert wird. Diese Stoffwechseleränderungen treten ebenso wie die positiv inotrope Wirkung nur am insuffizienten Herzen auf.

Während die absolute oder relative Verkleinerung des Herzminutenvolumens (im Verhältnis zur Blutmenge) bei der Herzinsuffizienz auf die Veränderungen des Herzstoffwechsels zurückzuführen sind, wird bei der Gefässinsuffizienz das Herzminutenvolumen kleiner, weil mit verringerter Blutmenge das diastolische Angebot an das Herz abnimmt.

Herzen von im hämorrhagischen Schock getöteten Tieren zeigen in den untersuchten Stoffwechselgrößen entsprechend keine Veränderungen gegenüber der Norm.

Gefässinsuffizienzen sind hämodynamisch entweder als einfache Hypovolämien oder als Schocksyndrome mit Hämokonzentration zu differenzieren. Als Kollaps werden diejenigen Zustände bezeichnet, bei denen die tiefe arterielle Drucksenkung und gleichzeitig die arterielle Regulation gestört sind. Bei manchen Gefässinsuffizienzen kann zusätzlich, wie z.B. bei 12% der Myokardinfarkte, eine Herzinsuffizienz beobachtet werden. Hier kann ein selbst dem verkleinerten Blutvolumen entsprechendes Herzminutenvolumen nicht gefördert werden. Die Gefässinsuffizienzen können sich bei Kreislaufgesunden postinfektiös, postoperativ, posttraumatisch, nach Blutverlusten oder nach Vergiftungen entwickeln. Tritt unter diesen Bedingungen eine Gefässinsuffizienz beim vorher kompensierten Herzkranken auf, so wird sie als Minusdekompensation, unter Berücksichtigung der zentralen Bedeutung der Abnahme des Blutvolumens, bezeichnet. Die klinische Symptomatik der Minusdekompensation wird der Plusdekompensation gegenübergestellt.

Für die Behandlung der Gefässinsuffizienzen im allgemeinen und der Minusdekompensation im besonderen wird die Anwendung sympathikomimetischer Stoffe, von Volumenexpandern und von anderen pressorischen Substanzen erörtert.

Die Gegenüberstellung von Herzinsuffizienz und Gefässinsuffizienz, von Plusdekompensation und Minusdekompensation entspricht den therapeutischen Erfordernissen, wie an Beispielen belegt wird.



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Etude Thermodynamique de l' Isohémagglutinine Humaine

L'étude thermodynamique des réactions entre antigènes et anticorps est susceptible de fournir des données quantitatives caractéristiques de la structure de ces substances, mais elle est généralement rendue difficile par le polyfonctionnalité de l'antigène de l'anticorps et l'hétérogénéité de ce dernier.

Les travaux effectués dans notre laboratoire par un groupe de chercheurs, S. Filitti-Wurmser, Y. Jacquot-Armand, G. Aubel-Sadron, M. Mavrides et moi-même, ont montré que l'isohémagglutination humaine du système ABO présente un cas particulièrement favorable d'application de la méthode thermodynamique. Dans les processus d'agglutination on peut s'attendre que même un anticorps bifonctionnel réagira au point de vue de la loi d'action de masse comme si ses molécules ne possédaient chacune qu'un groupe actif, le plus grand nombre d'entre elles n'étant, pour des raisons géométriques, fixées que par un seul de ces groupes. En outre les isoagglutinines du système ABO présentes dans les sérums humains normaux se comportent comme des système homogènes en ce qui concerne leur affinité pour les agglutinogènes correspondants. C'est pourquoi il a été possible de déterminer dans ce cas de façon précise les chaleurs de réaction et, avec une certaine approximation, les variations d'entropie.

La réversibilité de l'agglutination ayant été établie, la méthode employée consiste à mélanger des quantités connues de sérum et d'hématies et à doser l'agglutinine restée libre une fois que l'équilibre a été atteint. On détermine ainsi une constante d'équilibre intrinsèque. On déduit la chaleur de réaction ΔH de la variation de cette constante avec la température. La concentration de l'agglutinine dans le sérum avant le mélange est obtenue en valeur relative par la mesure du nombre maximum N_1 d'hématies qui peuvent être agglutinées à 4°C dans 1 mm³, dans des conditions standards. La concentration de l'agglutinine restée libre est représentée par le nombre maximum N_2 d'hématies qui

peuvent être agglutinées, dans les mêmes conditions, par le surnageant provenant de la centrifugation du mélange après que l'équilibre a été atteint. L'agglutinine fixée N_f est égale à $N_t - N'_t$.

On doit avoir d'après la loi d'action de masse

$$\frac{N_t}{N_f} = \frac{6 \times 10^{17}}{m\varphi} + \frac{6 \times 10^{17}}{mKN'^4}$$

où N_t est le nombre d'hématies présentes dans 1 mm³, m le nombre de groupes agglutinogènes accessibles sur une hématie, φ le rapport de N_t à la molarité de l'agglutinine. Si l'on porte N_t/N_f en fonction de $1/N'_t$ on doit obtenir une droite dont la pente P est inversement proportionnelle à mK .

Il a été trouvé en étudiant les sérums de 83 donneurs: 1° que pour un sérum normal (ou pour l'agglutinine extraite de ce sérum par absorption puis élution) on obtient effectivement une droite. 2° que la pente de cette droite est la même pour tous les sérums provenant d'individus de même génotype. 3° que la pente varie avec la température d'une manière différente suivant le génotype.

Le tableau indique les valeurs de la chaleur de réaction, en K cal/mole trouvées dans notre laboratoire pour les principales agglutinines α et β . Le terme entre parenthèses indique le génotype du formateur. On y a ajouté la valeur obtenue par Salmon pour le phénotype très rare Ax.

β (A ₂ O)	+ hématies B	16
β (A ₁ A ₁)	+ „	6.5
β (A ₂ O)	+ „	9
β (Ax)	+ „	14
β (OO)	+ „	1.7
α (BO)	+ hématies A ₁	19
α (B)	+ hématies A ₂	20
α (BO)	+ hématies A ₁	33
α_1 (O)	+ „	8.7
α (O)	+ „	5.3

Ces résultats montrent que l'isohém agglutinine normale possède une double spécificité, l'une vis-à-vis de l'antigène auquel elle peut se combiner, l'autre qui est la marque d'origine de l'anticorps. Un cas particulièrement remarquable est celui de l'agglutinine hybride β (A₁D).

Il a été possible pour quelques agglutinines de mesurer par ultracentrifugation en cellule cloisonnée les coefficients de sédimentation et d'évaluer les poids moléculaires. On a d'autre part au moyen de microdosages d'azote déterminé les valeurs du coefficient φ . On a ainsi obtenu une estimation du nombre m des groupes agglutinogènes accessibles sur une hématie et finalement de la variation d'entropie correspondant à la fixation de ces agglutinines. Il est apparu que les différences d'affinité trouvées entre agglutinines se combinant à un même agglutinogène sont dues à des différences non pas dans le groupe spécifique mais dans l'entourage de ce groupe ou même dans l'ensemble de la protéine.

La même méthode a fourni des renseignements sur les différences structurales existant entre des agglutinogènes tels que A₁ et A₂.



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Relation of Structure to Function of The Transfer Ran Molecule

The transfer RNA molecule occupies a central position in the translation of the language of the nucleic acid to that of the protein. On the one hand, a particular transfer RNA molecule recognizes its own activating enzyme, from which it then accepts an activated amino acid, in an ester bonding. The aminoacyl charged transfer RNA molecules then seek out by base pairing their appropriate sites on the template RNA molecule of the ribosome, and amino acids are added in the correct sequence to a growing peptide chain.

Whether these two separate recognition functions are performed by the same base constellation or by two separate ones remains to be determined. The detailed nature of the coding mechanisms is also obscure.

We are at present pursuing this general problem from two approaches. We are engaged in efforts to prepare sufficient amounts of a single purified species of transfer RNA (i.e. valyl-RNA) to permit sequential degradation studies to be initiated. We are also endeavoring to label the purified valyl-RNA with C^{14} and H^3 in order to facilitate this approach.

We have also under way endeavors to modify the structure of S-RNA molecules by chemical means, and to determine the effect on their biological specificities.

Symposia



F. Coste

Université de Paris

Situation Nosologique de la Polyarthrite Rhumatoïde (P. R.)

L'individualité nosologique de la P.R. ressort de nombreux caractères cliniques, de données histopathologiques et d'anomalies biologiques qui l'accompagnent: parmi ces dernières, l'existence du *système rhumatoïde* imprime à la maladie du même nom le cachet d'une spécificité qui, jusqu'alors, n'était que pressentie. La valeur pratique des tests qui détectent le F.R. est discutée et les critères actuels du diagnostic passés en revue: ils aboutissent à un démembrement de l'ancien cadre morbide que la terminologie française désignant sous le nom de polyarthrite chronique évolutive (P.C.E.). Ce démembrement se poursuit actuellement, menant à circonscrire avec plus de précision que naguère le domaine de la P.R. Il existe un groupe nombreux et divers de *polyarthrites atypiques*, sans doute destinées à s'exclure de la P.R. et que, à la lumière des critères de l'A.R.A. modifiée, il importe de différencier de celle-ci.

Tels sont: le *rhumatisme psoriasique*, la *pseudo polyarthrite des ceintures*, le *rhumatisme palindromique*, l'*hydathrose intermittente*, les *gonites et coxites primitives isolées* (bilatérales ou non), certaines *polyarthrites chroniques à réaction de W.R. obstinément négative*. D'autre part la situation nosologique des *polyarthrites chroniques juvéniles* soulève certaines difficultés d'interprétation. Enfin le problème des relations nosologiques entre la P.R. et les *Connectivites* (lupes érythémateux systémique, périartérite noueuse, polymyosites, sclérodermie, etc.) continue d'être débattu.

Si l'autonomie de ces diverses maladies vis-à-vis de la P.R.—bien que probable—demeure parfois discutée, il n'y a plus actuellement aucune hésitation en ce qui concerne la spondylarthrite ankylosante très généralement distraite aujourd'hui du cadre de la P.R.

Si l'analyse nosologique en cours permet aujourd'hui de circonscrire avec plus de précision que naguère le domaine de la P.R., nos connaissances sur son *étiologie* n'ont malheureusement pas progressé au même rythme.

Si les explications que voient dans la P.R. une "maladie de l'adaptation" ou le résultat d'un déséquilibre hormonal ne retiennent aujourd'hui plus guère l'attention, l'étude du système rhumatoïde a canalisé les hypothèses vers une conception immuno-pathologique de la maladie.

Une analyse méthodique des caractères du F.R., du "réactant" et du rôle qu'on pouvait être tenté de leur attribuer dans l'étiologie de la maladie a mené à des résultats plutôt décevants. La recherche des manifestations d'hypersensibilités dans la P.R. et de la part attribuable respectivement aux hypersensibilités sérique et cellulaire font actuellement l'objet de très nombreuses recherches. L'hypothèse d'un désordre immunologique d'origine héréditaire retient surtout l'attention: déduite de nombreuses constatations cliniques, biologiques et génétiques, elle demeure cependant entachée d'incertitudes. Mais les explications non immunologiques de la P.R. semblent à l'heure actuelle plus fragiles encore.



John C. Eccles

Australian National University

Physiological investigations on neuro-muscular transmission

There is a remarkable similarity in the fine structure of the vertebrate neuromuscular junctions so far examined with the electron microscope. All have a 200 Å cleft separating muscle and nerve membranes, with numerous mitochondria and many 300-500 Å vesicles in the nervous element. Matching this anatomical similarity is a physiological similarity. In the 1930s Dale and his co-workers performed the fundamental experiments that led to the postulate that neuromuscular transmission was effected by a chemical transmitter, acetylcholine. At the end of this decade Schafer and his colleagues discovered that on nerve stimulation a local potential (depolarization) was set up in the muscle membrane—the end-plate potential (EPP); and this work was confirmed and developed by Feng in China and by a research group at the Kanematsu Institute in Sydney. The general picture had then developed that the acetylcholine liberated by nerve impulses depolarised the end-plate membrane of the muscle fibre and so evoked muscle impulses, which in turn induced muscle contraction. With the advent of intracellular recording a new phase of investigation began. In particular the work of Katz and his school established chemical transmission by acetylcholine beyond doubt, and showed that the acetylcholine was always released in small packets or quanta. The random emission of single quanta produces miniature end-plate potentials. A nerve impulse causes momentarily an enormous increase in the rate of release, so setting up EEPs.

More recently, investigations have been concentrated upon the properties of motor nerve terminals, particularly the changes in the terminals which initiate and follow transmitter release. Techniques have been discovered for recording and stimulating the terminals and also for polarizing them with applied currents. Some of the results of these investigations will be discussed.

This recent work relates particularly to the problems of the mobilization and replenishment of transmitter that must occur during the normal activity of the neuromuscular junction.

Presynaptic and Postsynaptic Inhibition in the Central Nervous System

In 1957 Frank and Fourtes first clearly showed that there were two distinct types of inhibition. Subsequent investigation has revealed that in the spinal cord a large proportion of the inhibitions is effected by the presynaptic type of inhibition, in which a special type of chemically transmitting synapse depolarizes excitatory presynaptic fibres and so depresses their synaptic action. Virtually all medullated primary afferent fibres in the spinal cord are in this way depolarized by conditioning volleys with a consequent depression of their synaptic efficacy. A comprehensive investigation of the presynaptic inhibitory action on a wide variety of afferent fibres of limb nerves has shown that three major types can be distinguished in relation to the identity of the recipient afferent fibres, but no topographical pattern has been discerned.

In contrast to presynaptic inhibition, postsynaptic inhibition action is effected by a special type of chemically transmitting synapse that depresses excitatory synaptic transmission by exerting an antagonistic action on the postsynaptic membrane. The depolarizing action of excitatory synapses is thus directly counteracted by postsynaptic inhibitory synapses which, when activated, cause ionic fluxes across the subsynaptic membrane that tend to hyperpolarize the postsynaptic membrane. Many, but not all, types of postsynaptic inhibition in the spinal cord display a topographic pattern of action.

The presynaptic and postsynaptic inhibitory actions on the pathways ascending the spinal cord will be described. An account will also be given of the relative roles of presynaptic and postsynaptic inhibition at the synaptic relays of the dorsal column tracts in the cuneate nucleus and at the synaptic relays of the dorsal column tracts in the cuneate nucleus and at the synaptic relays of the medial lemniscus in the thalamus. It will be shown that in the cuneate nucleus inhibition is predominantly of the presynaptic type, whereas in the thalamus it is predominantly postsynaptic.



Peter H. Forsham

University of California, San Francisco

Pituitary-Adrenocortical Relationships

Less than one international unit (IU) of ACTH is released daily from the pituitary in a diurnal cycle. Secretion is increased either by stress, through corticotropin releasing factors in the midbrain, or by a fall in circulating cortisol. These lead to a moderate rise in the level of ACTH in the blood, which is normally less than 2 microunits per ml. Certain chromophobe pituitary tumors producing bilateral adrenocortical hyperplasia have very high ACTH levels. The usual diurnal variation of adrenal secretion may be absent in Cushing's syndrome and in central nervous system diseases. ACTH secretion is regulated by a sensitive feedback mechanism of the free, active form of cortisol, dissociated from transcortin and albumin. The greatest value of the test of pituitary ACTH reserve by mepyrapone is in the diagnosis of ACTH inhibition by adrenal cortisol secreting tumors. As a prognostic test before surgical operation it is of limited value. The depressing effect of psychotic states and various psychotropic drugs on ACTH discharge has been evaluated with mepyrapone and results suggest that response to stress may be critically impaired. Alpha corticotropin (Li), a pure porcine ACTH polypeptide consisting of 39 amino acids (150 IU per mg), activates the secretion of all the important adrenal hormones. With continued administration of ACTH, increasing quantities up to a certain maximum are secreted, except for aldosterone, which decreases. Human growth hormone is not a direct aldosterone Stimulant, whereas angiotensin is. Human ACTH (39 amino acids) behaves the same in man as alpha corticotropin but its potency is less. The absence of antigenicity and anaphylaxis of human ACTH is shared by synthetic products prepared by Li. The 17 amino acid chain (3 IU per mg) and the 19 amino acid derivative (30 IU per mg) are fully active. The clinical use of nonallergic synthetic ACTH will soon be a reality, some 20 years after its original isolation.



W. H. Hauss

*Medizinische Klinik u. Poliklinik der
Universität Münster*

Bedeutung der Acceleration des Mesenchymstoffwechsels in der Pathogenese der Coronarsklerose und des Herzinfarktes

Im letzten Jahrzehnt wurde durch Forschungen, an denen Kliniken und Laboratorien fast in aller Welt teilgenommen haben, versucht, die Rolle zu klären, die *Fettstoffwechselstörungen* in der Pathogenese der Arteriosklerose, insbesondere ihrer praktisch wichtigsten Form, der Coronarsklerose, spielen. Viele Autoren erblicken in Anomalien des Fettstoffwechsels einen wichtigen ätiologischen Faktor. An der Medizinischen Universitätsklinik in Münster haben wir unser Augenmerk in der Arterioskleroseforschung auf den *Mesenchymstoffwechsel* gerichtet.

In der Grundsubstanz des Mesenchyms finden bekanntlich ständig Sulfatierungen statt. Der Schwefeleinbau in das Chondroitinschwefelsäuresulfat der Grundsubstanz des Mesenchyms kann durch Verwendung von radioaktivem Schwefel Rontrolliert werden. Bei Untersuchungen menschlicher Leichenaorten fanden wir, dass der Schwefeleinbau im Alter langsamer vor sich geht. Demgegenüber ist er in arteriosklerotischen Gefäßen beschleunigt. Diese Acceleration des Mesenchymstoffwechsels haben wir auch bei der tierexperimentellen Arteriosklerose aufgezeigt.

Der beschleunigte Schwefeleinbau ist bereits zu einem Zeitpunkt nachweisbar, in dem morphologische Veränderungen der Gefäßwand noch nicht eingetreten sind. Der accelerierte Mesenchymstoffwechsel stellt also den ersten Schritt dar, der zur Arteriosklerose führt.

Bemerkenswerterweise ist die Acceleration des Mesenchymstoffwechsels in unseren Tierversuchen nicht nur im Bindegewebe der Gefäße sondern im Mesenchym aller Organe nachweisbar. Dieser Befund zeigt, dass die Arteriosklerose

eine generalisierte Erkrankung, eine Erkrankung des gesamten Mesenchym-systems darstellt.

Durch Probeexcisionen aus der Haut konnte die Mesenchymstoffwechselacceleration auch an Arteriosklerosepatienten, insbesondere bei Herzinfarktpatienten in der Klinik aufgezeigt werden. Kontrollen der Glucosaminausscheidung im Harn (sogenannte AS TRUP-Fraktion) und des Mucoproteinspiegels im Plasma zeigten ebenfalls den pathologisch erhöhten Mesenchymstoffwechsel an.

Bezeichnenderweise reagiert der Mesenchymstoffwechsel auf Einwirkungen, denen man nach klinischem Urteil einen Einfluss auf Entstehung und Progression der Arteriosklerose zuzuerkennen geneigt ist. In zahlreichen Versuchsserien haben wir den S^{35} -Einbau gemessen. So kann zum Beispiel durch Infektionen, Toxin-Injectionen, Injektion von Fettsäuren, Hypoxämie, mechanische Einwirkungen, Verabreichung von Hormonen, diätetische Umstellung, Wetterumschlag und excessive Muskelarbeit eine Beschleunigung des Mesenchymstoffwechsels bewirkt werden.

Unsere Befunde begründen folgende Vorstellungen über die Pathogenese der Arteriosklerose:

1) Die Arteriosklerose ist eine Erkrankung, die mit einer Acceleration des Mesenchymstoffwechsels beginnt.

2) Der Grundsubstanzstoffwechsel ist bei generalisierter Arteriosklerose nicht nur im Mesenchym der Gefäßwand sondern in allen Organen beschleunigt.

3) Die Acceleration des Mesenchymstoffwechsels wird reaktiv bewirkt durch eine grosse Anzahl von Reizen (lokale oder universelle unspezifische Mesenchymreaktion).



G.C. Hirsch

Zoologisches Institut d. Univ. Göttingen

**Dynamics of Cell Secretion, Integration
of morphological and biochemical
on the pancreas**

Ingestion of raw material, passive and active transport. No-transport of macromolecules.

Synthesis of secretions: Distribution of amino acids. The ribosome-ER-unit. Non-portable proteins (Ph. Siekevitz). The metabolic pathway. Correlation between ribosomes and ER. Condensation and movement of proteins in the ER. The lamellar-vacuolarfield or "Golgi Complex": The pattern, the two functions, the biochemistry of this field.

Extrusion of secretions: Hiatus in the integration. Look-out to further experiments.



R. J. H. Oberholzer

Zürich and Basle, Switzerland

**Afferent fibers from the Cardio-pulmonary
region and their connection to the
respiration centers in the medulla
oblongata**

Technical progress generally opens new fields of investigation to doctors and research workers. Thus the development of cardiac catheterization, of rapidly reacting and continuously recording gas-analyzers, spectro-photometry and the radio-isotope technique led to an extensive experimental and clinical investigation of cardio-pulmonary function. Haemodynamics and respiratory gas-exchange mechanisms in healthy and diseased persons are much better understood than 20 years ago. Other fields however have received less attention, especially as there were no methods at hand which could be used in clinical research. We think of the less frequently discussed subject of the title.

Following the interest of our teacher the Nobel prize winner *W. R. Hess*, Professor Wyss and his group first investigated the reflex regulation of respiration through the afferent vagal nerves. This led to a better understanding of the so-called Hering-Breer mechanisms. Within this group the speaker and collaborators then investigated the central connection of afferent nerves of cardiopulmonary origin in the medulla oblongata. This was followed by an attempt to localize in the bulbar region the responsible area for the inspiratory drive by *Vassella*. Finally *Jasargil* made a functional analysis of afferent and efferent fibers in the phrenic nerve, thus closing the loop of our investigational program.

Chemo-, presso- and volume receptors from the heart, the lungs and the pulmonary vascular bed and the part they play in respiratory and cardio-vascular

regulation will be discussed. The bulbar relay stations of the pulmonary vagal and the aortic and carotid baropressor fibers will be demonstrated. Reference will be made to anatomical and electrophysiological investigations of other groups and evidence of a functional organization of the respiratory and vasomotor centers in the medullary region will be presented. Finally the problem of the distribution of efferent impulses to the diaphragm, i.e. the tonic and periodic innervation of this respiratory muscle will be discussed if time permits.



Arthur H. Steinhaus

George Williams College, Chicago

Mysterious Backaches

For many backaches there are easily definable causes and in many cases satisfactory remedies. There are, however, some backaches for which there appears to be no demonstrable cause. Frequently, these are considered to be psychosomatic and sometimes malingering is suspected. When people who suffer such aches say that almost anything that ails them, be it a cold, ordinary fatigue, or an other physical or mental stress, precipitates an attack that always "settles" in the same region, the suspicion of a psychic cause increase.

Observations reported by Buchthal and Clemmensen of Copenhagen ("On the Differentiation of Palpable Muscle Affections by Electromyography", *Acta. Med. Scandinav.* 105, 48-65, 1940) and by Denslow and Krem ("Quantitative Studies of Chronic Facilitation in Human Motoneuron Pools," *Amer. Jour. Physiol.* 105, 29-38, 1947), in the opinion of the present author, provide a neurologic explanation for at least some of these conditions, as follows:

Faulty vertebral alignment and postural defects may evoke persistent exaggerated stretch reflexes to the point where these create a hyperirritable cord segment. All reflex paths as well as ascending sensory tracts that synapse in such a segment would share in this raise "central excitatory state" (Sherrington). In consequence otherwise unnoticed stimulation as from light skin touch, a cold, or even psychic stress may be sufficient to excite the already partly activated neurone pools to the "firing" threshold thus giving rise to pain and perhaps other conditions.

By this explanation the psychic factor becomes the "last straw" that breaks the camels back and not the real cause.



F. Wachsmann

*Institut für Strahlenkunde der Universität
Erlangen, Deutschland*

Die Entwicklung der Therapie mit ultraharten Strahlungen

In der frühzeit der Strahlentherapie dachte man nur daran, eine ausreichende Dosis an den Herd zu bringen. Erst als die Rolle des ungeschädigten Tumoruntergrundes für die Abheilung erkannt worden war, wurde die Forderung nach "Konzentration der Dosis auf den Herd und Schonung der gesunden Umgebung" erhoben.

In der Hauttherapie und der Therapie oberflächlich gelegener Tumoren lässt sich die Strahlenwirkung leicht durch wahl kleiner Fokusabstände oder besser weicher Strahlungen auf die Oberfläche beschränken. Schwieriger liegen dagegen die Verhältnisse bei tiefliegenden Herden. Hier wurde die klassische Kreuzfeuertherapie durch die Bewegungsbestrahlung ersetzt. Diese gestattet durch lückenlose Ausnützung der als Strahleneintrittspforte zur Verfügung stehenden Oberfläche bei kleinen Herddurchmessern ausreichende Tiefendosen zu erzielen. Um auch grössere Tumoren mit genügend grossen Dosen bestrahlen zu können, müssen jedoch neben der Bewegungsbestrahlung sogenannte ultraharte Strahlungen grösserer Durchdringungsfähigkeit benützt werden.

Derartige Strahlungen, deren Erzeugung früher unmöglich war, stehen uns seit etwa einem Jahrzehnt von verschiedenen Strahlenquellen geliefert zur Verfügung.

Die harten Gammastrahlen künstlich radioaktiver Stoffe—von allem des Caesium-137 und noch besser des Kobalt-60 ermöglichen es, bei gleicher Oberflächendosis bereits etwa zweifach höhere Tiefendosen zu erreichen.

Ähnlich gute relative Tiefendosen ergeben auch die künstlich mit Hilfe von Linearbeschleunigern von meist 4 bis 8 MeV erzeugten ultraharten Röntgenstrahlen. Nochmals um den Raktor 2 bessere Verhältnisse, d.h. 4 mal höhere

Tiefendosen lassen sich mit Hilfe des Betatron erzielen, dessen Strahlenenergie bei tragbarem technischen Aufwand bis 20 oder 30 MeV oder noch höher geht. Besonders günstige Dosisverteilungen ergeben sich dabei durch eine Kombination der ultraharten Strahlungen mit der Bewegungsbestrahlung während die schnellen Elektronen vor allem zur Behandlung oberflächlicher und oberflächennahe gelegener Prozesse verteilhaft sind.

Einige Beispiele zeigen, wie die "relative Herdraumdosis" als Maß für die Konzentration der Dosis auf den Herd durch die Einführung der ultraharten Strahlungen in die Strahlentherapie verbessert werden konnte.



W. Wilbrandt

*Department of Pharmacology, University
of Bern, Switzerland*

The Concept of Carrier Transport

The carrier concept, introduced 30 years ago by Osterhout, has in recent years been increasingly used for the interpretation of biological transport. This is mainly due to two reasons. First it offers possibilities to explain some features frequently observed and not fitting the classical concepts of porosity and lipid solubility including high structural specificity, particularly stereospecificity, and limited capacity leading to saturation kinetics and competition. Second it is likely to be the basic mechanism of many transports occurring against gradients of chemical (or electrochemical) activity ("uphill transport", most commonly termed "active transport"). This is concluded from the fact that a number of characteristic features derived from the basic carrier assumption have been observed in uphill transport systems.

Since this mechanism as such only leads to equilibration additional features must be assumed to enable the system to transport uphill. One of the possibilities suggested by previous authors (Solomon, Shaw) is a chemical reaction on one side of the membrane between the carrier molecule and metabolites transforming the carrier from a form Z with low substrate affinity to a form C with high affinity. If this change is reversed on the other side of the membrane (either by a spontaneous reaction or by a second metabolic reaction) an uphill transport of the substrate ensues. The energy for this transport, then, is furnished by the metabolic reaction (or reactions).

The kinetics of such a system have been worked out. It offers explanations for a number of observations. They include the finding that dinitrophenol inhibits some uphill transports into cells, not (as predicted on the basis of the commonly assumed "leak and pump" scheme) by decreasing influx but by increasing efflux (observations of Horecker et al. on bacterial permeases and of Weatherall on heart muscle. The capacity of some systems to transport different substrates uphill in opposite directions (observations of Lassen in the red cell) can also be accounted for by the system.



Sabine Filitti-Wurmser

*Centre National de la Recherche
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Relations entre les gènes ABO et les Propriétés des isohémagglutinines humaines

L'étude thermodynamique des isohémagglutinines du système ABO présentés dans le sérum des individus normaux a établi que la structure de ces anticorps "naturels" dépend du génotype de l'individu qui a synthétisé l'anticorps (Filitti-Wurmser, Y. Jacquot-Armand, G. Aubel-Lesure, R. Wurmser).

Cette dépendance apparaît de manière toute nouvelle dans les travaux récents de Salmon et al. Ces auteurs ont trouvé que dans les leucémies aiguës tout se passe comme s'il se produit une mutation somatique du gène de groupe sanguin A et que la modification porte à la fois sur l'antigène et l'anticorps.

La relation entre la structure de l'anticorps et le génotype n'a jusqu'à présent été observée que pour les isohémagglutinines normales. Elle n'a pu être recherchée par la même méthode pour les isohémagglutinines immunes en raison de leur hétérogénéité. Néanmoins il est probable que la relation entre les gènes ABO et les propriétés thermodynamiques des isohémagglutinines normales est spéciale à ces dernières, et résulte des conditions de la synthèse protéique dans les cellules où elles se forment. Ce seraient les différences dans les lieux de formation des agglutinines normales et immunes qui seraient responsables des caractères distinctifs, dont certains sont employés pour déceler les cas graves d'immunisation foetomaternelle.

Le mécanisme du contrôle génique constitue un problème non encore résolu. Il est particulièrement difficile d'expliquer la formation de la molécule hybride anti-B découverte dans les sérums A₁O. On n'a pas affaire dans ces sérums à un mélange β (A₁A₁) et β (OO) mais à une substance β (A₁O) qualitativement différente de ces deux agglutinines. Il en est de même dans le cas de l'isohémagglutinine naturelle de la chimère découverte par Dunsford et al. Nous avons trouvé en effet avec Y. Jacquot-Armand qu'à la précision de notre technique, le sérum de cette chimère ne contient comme isoagglutinine anti-B que la substance β (A₁O).



Eleanor Zaimis

*Department of Pharmacology, Royal Free
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Evaluation of Neuromuscular Blocking Drugs in Animals and Man

For the successful evaluation of neuromuscular blocking drugs there are several points which have to be considered. It is important to know first of all that the measurement represents an interruption of neuromuscular transmission. Secondly, one must keep in mind the complicated situation which arises because of differences which exist not only between the muscles of different species but even between various muscles of the same species. Furthermore, that in comparisons between drugs, their modes of action are of primary importance, and finally, that heterogeneity of experimental techniques and the use of isolated muscle preparations are likely to explain part of the discrepancies found during the study of neuro-muscular blocking drugs.

Entertainments Schedule of the Participating Members from Abroad of The 16th General Assembly of the Japan Medical Congress

Congresses				Entertainments					
Date	Time	Events	Places	Time	Events	Given by	Fees	Places	Participants
Mar. 31 (Sun.)				5.30 p.m.	Dinner	Dr. Imamura President, JMC	not required	Hotel New Osaka	limited members
Apr. 1 (Mon.)	9.00 a.m.	Opening ceremony of the 16th General Assembly of JMC	Osaka Municipal Central Gym	5.30 p.m.	Reception	Governor & Mayor of Osaka	"	Talkoem	All members
Apr. 2 (Tues.)	All day	Scientific meetings of JMC	Osaka	9.15 a.m. 7.23 p.m.	*Excursion tour	J. M. C.	\$ 10 Per capita	Toha & Ise (Mikimoto Pearls)	Volunteers & their families
Apr. 3 (Wed.)	7.00 p.m.	Evening Forum (Clinical Experi- ences)	Festival Hall	9.00 a.m. 4.00 p.m.	**Girls' revue	J. M. C.	admission free	Takarazuka Theatre	Ladies program Husbands also invited
				5.00 p.m.	Men meeting	J. M. C.	"	Osaka Noh Music Hall	Volunteer & their families
Apr. 4 (Thur.)	7.00 p.m.	Panel discussion (On the Development of Medical Care in Japan)	Festival Hall	4.30 p.m. 6.00 p.m.	Tea party	Mrs. Imamura Chairman Ladies comm.	not required	Alaska	Ladies only
				9.00 a.m. 8.00 p.m.	**Factory visiting Tea ceremony	J. M. C.	"	Matsushita Electric Industrial Co. Ltd. Shugakuin detached Palace (Kyoto)	Volunteers & their families
Apr. 5 (Fri.)	4.00 p.m.	Scientific meetings	Osaka	10.00 a.m. 3.00 p.m.	**Kimono show	J. M. C.	admission free	Daimaru Department Store, Osaka	Ladies' program Husbands also invited
				5.30 p.m.	Cocktail party	Dr. Takeuti President, JMA	not required	Hotel Osaka Grand	All members
Apr. 6 (Sat.)		Closing ceremony	Festival Hall	9.00 a.m. 5.00 p.m.	***Excursion tour	J. M. C.	not required	Nara & Horyuji Temple	Volunteers & their families

Notice

* Those who wish to go to this excursion tours may start from Ueroku station, at 9.15 a.m.

** A bus will take you from your hotel.

*** A bus will leave your hotel at 9.00 a.m.

Place for General Meeting of Each Member Society

* refer to the map on page 31.

* meetings of societies (12, 13 36 and 40) are to be held jointly.

Location No.	Name of each member society	Place of meeting
1	Japanese Society of Medical History	Takeda Chemical Industries, Ltd. Hall
2	Japanese Association of Anatomists	Osaka Univ. School of Medicine, Great Hall
3	Physiological Society of Japan	Nissei Kenshujo (2)
5	Japanese Pharmacological Society	Y M C A Hall
6	Japanese Pathological Society	Osaka Dental College Hall
8	Japan Haematological Society	Osaka Chamber of Commerce & Industry
8'		Chuo Denki Club
9	Japan Bacteriological Society	Ohyi Bulding Hall
10	Japanese Society of Parasitology	Osaka-fu Kosei Kaikan
11	Medico-Legal Society of Japan	Daido Seimei Hall
12	The Japanese Society for Hygiene	Osaka City University
13	Japanese Society of Race Hygiene	"
14	Japanese Society of Food and Nutrition	Osaka Jogakuin
15	Japanese Association of Physical Medicine Balneology and Climatology	Osaka-fu Shoko Kaikan
16	Japan Endocrinological Society	Kanden Hall
17	The Japanese Society of Internal Medicine	Osaka University Hall
18	Japanese Paediatric Society	"
19	The Japanese Association for Infectious Diseases	Aijitsu Primary School Hall
20	The Japanese Society for Tuberculosis	Chuo Kokaido
21	Gastro-enterological Society of Japan	Nihon Seimei Hall
22	Japanese Circulation Society	Otemae Kaikan
23	The Japanese Society of Psychiatry & Neurology	Sankei Hall

24	Japanese Surgical Society	Festival Hall
25	Japanese Orthopaedic Association	Mainichi Hall
26	Japanese Obstetrical and Gynecological Society	Shitennoji Kaikan
27	Japanese Ophthalmological Society	Rhoto Seiyaku Hall
28	The Oto-Rhino-Laryngological Society of Japan	Kokumin Kaikan
29	The Japanese Dermatological Association	Mido Kaikan
30	Japanese Urological Association	Soai Gakuen Hall
31	The Japanese Stomatological Society	Nissei Kenshujo (1)
32	Japan Radiological Society	Gas Building Hall
35	Japanese Leprosy Association	Fujisawa Pharmaceutical Co., Ltd. Hall
36	Japanese Society of Public Health	Osaka City Univ., Main Bldg.
37	The Japan Society of Sanitary Zoology	Osaka-fu Shokuin Kaikan
38	The Medical Association of Trans- portation Hygiene and Casualties of Japan	Furitsu Shakaijigyo Tandai Hall
40	Japan Society of Industrial Medicine	Osaka City Univ., Main Bldg.
45	Japan Society of Anesthesiology	A B C Hall
48	Japan Society of Blood Transfusion	Osaka Univ. Hospital Hall
49	The Japanese Society for Medical Mycology	Fujisawa Pharmaceutical Co., Ltd. Hall
50	The Japanese Association of Rural Medicine	Osaka-fu Norin Kaikan
51	Japan Diabetic Society	Imabashi Club
52	The Japanese Association of Correctional Medicine	Fukusuke Tabi Hall
53	Japanese Society of Neurology	Asahi Seimei Hall



*Place of Closing Ceremony
(Festival Hall)*



*Place of Exhibition
(Osaka Furitsu Taiikukan)*



*Place of General Assembly
(Nakanoshima Kokaidō)*



*Place for Movie Performance
(Sankei Hall)*



*Place of The 100th Death Anniversary
of Kōan Ogata
(Mainichi Hall)*

List of International Medical Meetings

to be held in Japan

Compiled by

Japan Medical Association

Date	Name	Place	Participant from abroad	Organization concerned
1963				
Mar. 27 - 30	International Symposium on Cellular Chemistry	Ohtsu, elsewhere	30	Headquarters of Japanese Society on Cellular Chemistry: Dep. Pathology, Medical School, Okayama University, Okayama
Mar. 30	3rd Congress of CMAAO (Business Session)	Tokyo, Japan Medical Association House	100	Japan Medical Association: 5, 2-chome Kanda-surugadai, Chiyoda-ku, Tokyo
Apr. 1	3rd Congress of CMAAO (Scientific Session)	Osaka, Dentsu Building	100	"
Apr. 1	The 1st Asian & Oceanic Regional Congress of Constitutional & Diathetic Medicine	Osaka	10	The Japanese Association of Constitutional Medicine: c/o Dep. Pathology, Faculty of Medicine, Kyoto University, Sakyo-ku, Kyoto
Apr. 1 - 5	16th General Assembly of Japan Medical Congress	Osaka, 17 places	300	Headquarters of the 16th General Assembly of Japan Medical Congress: c/o The Memorial Hall of Osaka University Medical School, 33 Jyoan-cho, Kita-ku, Osaka
May 13 - 17	The Joint Meeting of the Japanese Society of Psychiatry & Neurology and the American Psychiatry Association	Tokyo, Hotel Ohkura	300	The Japanese Society of Psychiatry & Neurology: Dep. Psychiatry, University of Tokyo, 1, Motofuji-cho, Bunkyo-ku, Tokyo
Aug. 26 - 28	Asian Congress of Blood Transfusion	Hakone		Dep. Surgery, School of Medicine, Keio University: 35, Shinano-machi, Shinjyuku-ku, Tokyo

1964

May 10 - 14
The 3rd Congress of the
Asian Pacific Society of
Cardiology

Kyoto, Kyotokaikan
Kyoto City Hall

200

Japanese Circulation Society: c/o 3rd Medical Clinic of
Kyoto University, Yoshida-cho, Sakyo-ku, Kyoto

October
16th Congress of International
Medico-Athletic Federation

Tokyo

Japanese Physical Fitness Society: c/o Natori Laboratory,
Dep. Physiology, Jikeikai Medical School, Shiba Atago-
cho, Minato-ku, Tokyo

1965

July 5 - 10
11th International Congress
of Paediatrics

Tokyo, Hotel Ohkura

Headquarters of the 11th International Congress of
Paediatrics: Dep. Paediatrics, University of Tokyo

Sept. 3 - 10
The XXIII International Con-
gress of Physiological Sci-
ences

Tokyo

c/o Investigation Section, Science Council of Japan: Ueno
Park, Taito-ku, Tokyo

Oct. 24 - 30
VIII International Congress
of Oto-Rhino-Laryngology

Tokyo, Tokyo-
Bunka-Kaikan

Oto-Rhino-Laryngological Society of Japan: c/o Kojimachi-
Mansion 3, 5-chome Kojimachi, Chiyoda-ku, Tokyo

1966

March
International Congress of the
Gastroenterological Endos-
copy

Tokyo

Japan Gastroenterological Endoscopic Society: c/o 1st
Dep. Internal Medicine, Faculty of Medicine, University of
Tokyo

Oct. 23 - 29
The 9th International Con-
gress of Cancer

Tokyo

Cancer Institute: 2-2615, Nishisugamo, Toshima-ku,
Tokyo

Undecided
3rd World Congress of
Gastroenterology

Tokyo

Gastroenterological Society of Japan: c/o Ichio Hospital,
2-3, Uchisaiwai-cho, Chiyoda-ku, Tokyo

The 3rd Congress of the Confederation
of
Medical Associations in Asia and Oceania



The Organizing Committee for the 3rd Congress of the CMAAO
c/o The Japan Medical Association
5, 2-chome Kanda-surugadai
Chiyoda-ku
Tokyo, Japan



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Immediate-past President



Dr. Heraldo del Castillo
President



Dr. Hideo Yagi
President-Elect



Dr. Kôhei Toyokawa
*Secretary
Organizing Committee*



Dr. Victorino de Dios
Secretary-Treasurer



Dr. Takeo Tamiya
*Chairman
Organizing Committee*

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President-Elect : HIDEO YAGI, M.D.
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Immediate Past President : TARO TAKEMI, M.D.

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Vice-Chairman : HIDEO YAGI, M.D.
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CHINA MEDICAL ASSOCIATION
INDONESIA MEDICAL ASSOCIATION
IRAN MEDICAL ASSOCIATION
JAPAN MEDICAL ASSOCIATION
PAKISTAN MEDICAL ASSOCIATION
PHILIPPINE MEDICAL ASSOCIATION
SOUTH KOREA MEDICAL ASSOCIATION
THAILAND MEDICAL ASSOCIATION

Office Address (Secretariat)
2114 JUAN LUNA, MANILA, PHILIPPINES

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AUSTRALIA

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Theodore Morley	Member of Aust. M. A.
W. H. Hill	Member of Aust. M. A.
H. E. W. Lyons	Member of Aust. M. A.

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Myint Han	Joint Secretary-General, Burma Medical Association No. 249, Theinbyu Road, Rangoon
Min Sein	Council Member, B. M. A.
Ohn Maung	Honorary Treasurer, B. M. A. (Moulmain Branch)
Saw Ba Heng	Member, B. M. A.

CHINA

Chi-Fu, Wu	169 Chong-cheng 4 Road Kaohsiung-city Taiwan
Jau-Chin, Lii	Member, China Medical Association
Tung-Shiang, Pen	Member, C. M. A.
Yin-Ho, Su	Member, C. M. A.

INDONESIA

IRAN

Saeed Hekmat	Ministry of Health Teheran Medical Association, Teheran
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JAPAN

Hideo Yagi	President-Elect of the 3rd CMAAO
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Takeo Tamiya	Chairman of the Organizing Committee for the 3rd Congress of the CMAAO c/o Japan Medical Association 5-2 chome Kanda-surugadai, Chiyoda-ku, Tokyo
Haruo Katsunuma	Standing Director of Japan Medical Association Prof. University of Tokyo
Kôhei Toyokawa	Secretary of the Organizing Committee for the 3rd Congress of the CMAAO Prof. University of Tokyo

PAKISTAN

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Ho Sup. Shim	Honorary President K. M. A.

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The Executive Committee for the 3rd Congress of the CMAAO

Heraldo del Castillo	Philippines
Rodolfo Gonzalez	”
Victorino de Dios	”
Refael Enrile	”
Antonio Cañiza	”
Hideo Yagi	Japan
Takeo Tamiya	”
Haruo Katsunuma	”
Kôhei Toyokawa	”
Yoshiaki Miura	”
Chi-Fu, Wu	China
M. Rafiquiddin	Pakistan
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Office Address

The Organizing Committee for the 3rd Congress of the CMAAO
c/o The Japan Medical Association
5, 2-chome Kanda-surugadai
Chiyoda-ku
Tokyo, Japan



*Japan Medical Association
House*

Registration

Date:

March 29, 30, 1963

Time:

March 29 2.00 p.m.-5.00 p.m.

March 30 9.30 a.m.-5.00 p.m.

Place:

Second floor of the Nihon Ishikaikan, Room No. 2
(Japan Medical Association House)
5, 2-chome Kanda-surugadai, Chiyoda-ku, Tokyo

Registration Fees:

Free

Important:

Please register early and secure two badges and program.

Business Session

Date :

March 30, 1963
9:30 – 12:00 p.m.
2:00 – 5:00 p.m.

Place :

The Dining Hall of the Nihon Ishikaikan (4th floor)
(Japan Medical Association House)
5, 2-chome, Kanda-surugadai, Chiyoda-ku, Tokyo

Agenda

PART I

1. Call to order by the chairman.
2. Opening remarks by the presiding officer.
3. Presentation of gavel and banner of the Philippine Medical Association.
4. Short talk to welcome the delegates—Dr. Taro Takemi.
5. Introduction of the delegates by the secretary.
6. Approval of the proceedings of the last congress held in Quezon City, Philippines, April, 1961.
7. Report of the President, Dr. Heraldo del Castillo.
8. Report of the Secretary-Treasurer, Dr. Victorino de Dios.
9. Approval of the financial statement as submitted by the Executive Committee.
10. Approval of the budget—1963-1965.
11. Report of the committee on membership and public relations—Dr. Rodolfo Gonzalez
12. Short address by the chairman of the different delegates on the affairs of their respective association.
13. Fix annual dues.
14. Creation of different reference committees.
15. Election of President-elect and two councilors.
16. Induction to office of the newly elected officers.

17. Introduction of the incoming President, Dr. Hideo Yagi.
18. Inaugural address of the incoming President, Dr. Hideo Yagi.

PART II

Turn over of the Presidency to Dr. Hideo Yagi
Presiding officer—Dr. Hideo Yagi

1. Determination of the place of the 4th Congress.
2. Invitation of the Australian Medical Association to the Australian Medical Congress.
3. Planning of the program of activities for the next congress.
4. Appointment of other officers of the CMAAO if necessary.
5. Invitation of the West Pacific Regional Office of the WHO to a conference of Deans of Medical schools to be held in Manila, Philippines, on November 7 to November 8, 1963.
6. Proposal that the CMAAO will adopt his own banner.
7. Other matters

Scientific Session

Date :

April 1, 1963
2.00 – 5.00 p.m.

Place :

Dentsû Building, Nakanoshima, Osaka

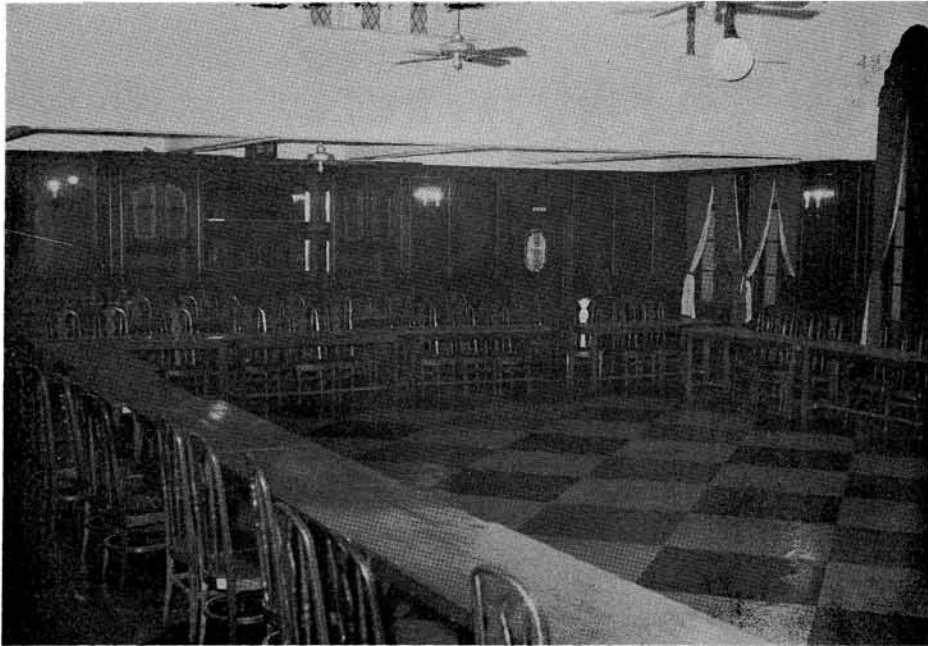
Papers

1. Report on Study of Recent Medical Professional Liability in Taiwan.
Dr. Chi-Fu, Wu (China)
2. Responsabilite Medicale
Dr. Saeed Hekmat (Iran)
3. Deliveries after Cesarean Section.
Dr. Julita R. Jalbuena (Philippines)
4. A report on the 1962 Cholera (EL TOR) Outbreak in Taiwan.
Dr. Yin-Ho, Su (China)
5. EL TOR Type Vibrio in the Recent Outbreak of Cholera.
Dr. P. R. Aragon (Philippines)
6. Japanese Contributions to the knowledge of Cholera due to EL TOR Vibrios.
Dr. Keizô Nobechi (Japan)
7. Form of the National Health Service in Australia.
Dr. F. R. Fay (Australia)

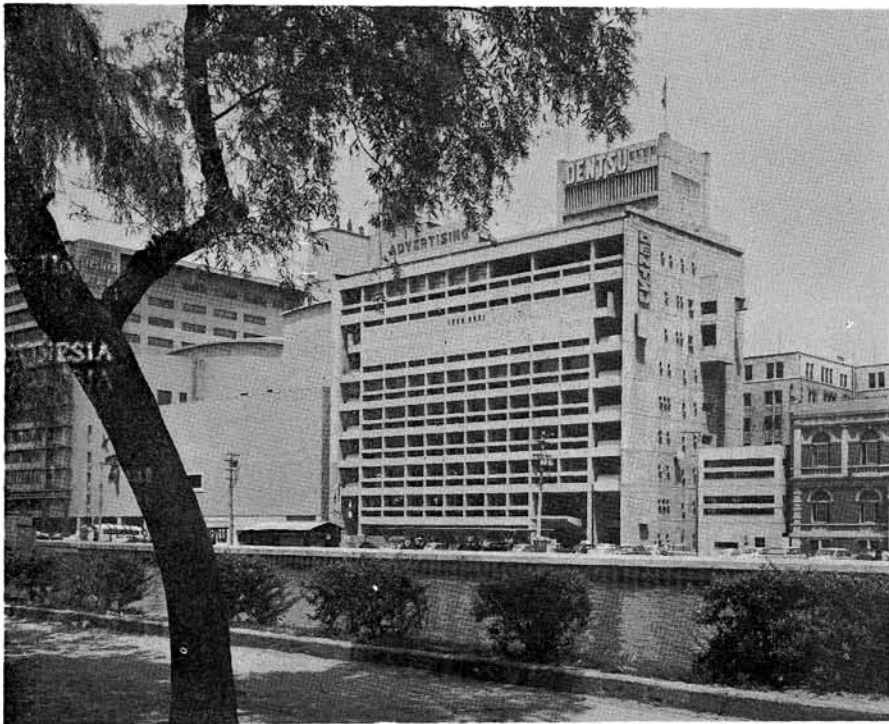
Rules of Procedure Governing the Scientific Session

1. The Chairman is responsible for the conduct of the meeting.
2. There will be strict adherence to the time limit (30 minutes) allowed each speaker and no deviation will be permitted.

3. Speaker must be in the room at the time announced for their papers. If they are not present, an alternate paper will be substituted.
4. Each person discussing a paper must give to the secretary of the session a written copy of his comments on the special form provided by the secretary.
5. Those speakers who are to use films, slides etc., to illustrate their papers are asked to give this material, plainly marked, to the projectionist at the beginning of the session. The size of slides must be 36mm×24mm (mounting 50.8mm×50.8mm).
6. The Congress will not publish complete papers. Authors are permitted to submit their manuscripts for publication in any journal they wish.
7. Papers should be presented in English.



*Place for Business Session
(The Dining Hall of the Nihon Ishikaikan)*



*Place of Scientific Session
(Dentsû Building, Osaka)*

Abstracts

**Report on Study of Recent Medical
Professional Liability in Taiwan.**

Dr. Chi-Fu, Wu

Deliveries after Cesarean Section.

Dr. Julita R. Jalbuena

**A Report on the 1962 Cholera (EL TOR)
Outbreak in Taiwan.**

Dr. Yin-Ho, Su

**EL TOR Type Vibrio in the Recent Out-
break of Cholera.**

Dr. P. R. Aragon

**Japanese Contributions to the Knowledge
of Cholera due to EL TOR Vibrios**

Dr. Keizo Nobechi

Report on Study of Recent Medical Professional Liability in Taiwan

Dr. Chi-Fu, Wu

Throughout the United States, England and all over the world medical malpractice are alarmingly on the increase year by year. Taiwan is also not the exception. Since 1948, after the World War the medical malpractice in Taiwan has become the important social problem and has caused the crisis of the medical profession.

The members of the Taiwan Medical Association, almost all, have been experienced to quarrel with patient about medical treatment, one half of them payed money for liability claims, 1/6 of them were used for malpractice.

Under such conditions, the physicians in Taiwan are now afraid of doing dangerous operations, are afraid of using penicilline, avoid to see the difficult cases. In view of this medical situation, the Taiwan Medical Association decided to take the action to prevent the medical malpractice and has been engaged in following major activities in this field during the past two years.

I. To investigate the malpractice cases. These informations were obtained from answers of 807 physicians, concerning:

- (a) doctor
- (b) patient and his family
- (c) type of incident
- (d) analysis of appeal cases
- (e) economic impact of liability claims.

The cases acquired from 1948 through March 1961 are only 261 cases out of 4101 physicians, but it should be borne in mind that the cases represent only a small fraction of the actual number of cases, because the physicians do not like to tell their cases to the Medical Association except the cases have already been reported on the newspaper. According to the exact investigations of the Kaohsiung Medical Association in 1960, 135 physicians out of 270 of their members have been involved in the medical malpractice.

II. With the conclusions of the above mentioned investigations the Taiwan Medical Association has organized the Security Committee on Medical Professional Liability and the Testimony Committee on Medical Science in October 1962.

The Security Committee on Medical Professional Liability serves on the following objects:

1. To protect, support and safeguard the professional character and interests of physicians.
2. To give advice and assistance to physicians sued with regard to any question or problem of law, and to represent physicians sued to resolve the liability claims with patient in court.
3. To manage the special insurance, our type of professional liability insurance coverage available to all members of the Medical Association deals only with the administration expenses and attorney fees, except the indemnity for malpractice claims.

The Testimony Committee on Medical Science serves on the following objects:

1. To testify on the medical science in the medical malpractice cases for the judgment of jury.
2. To rule or codify the limit on the medical malpractice by medical expert.
3. To exclude the law of the medical malpractice from the criminal law, and to codify it in the civil law.

Deliveries after Cesarean Section

Julita Ramoso Jalbuena, M.D., F.P.C.S., E.P.O.G.S.

Jose Villanueva, M.D., F.P.C.S., F.P.O.G.S.

and Hilario Ramos, M.D.

This study involved 525 patients with previous Cesarean section admitted in the Charity Obstetrical Service, Philippine General Hospital, covering a ten year period from July 1, 1948 to June 30, 1958. Each patient was evaluated carefully with vaginal and abdominal operative set up available. Two hundred sixty five (50.5 per cent) had a repeat Cesarean section and 260 (49.5 per cent) had a vaginal delivery.

Of the 260 patients permitted to delivery vaginally, 97.7 per cent delivered successfully with minimal complications and 2.3 per cent or six of the patients incurred symptomatic rupture of the uterus (5 complete 1 incomplete). It is true that vaginal delivery following Cesarean section is always fraught with danger of rupture of the uterus. However, it is worth while to note that there was no maternal death among these patients. There was no report of dehiscence in any of these patients. There were two stillbirths and one neonatal death. These three perinatal mortalities were from patients with previous classical scars. The uncorrected perinatal mortality in patients with vaginal delivery was 4.5 per cent.

Elective Cesarean section was the operation of choice in the presence of two or more Cesarean scars. More than half of the cases of dehiscence were observed in this condition. The percentage of uterine defect therefore seemed to increase with increasing number of sections. Fortunately, one-half of these defects were minor and incomplete and therefore were only repaired.

In those instances in which elective Cesarean section was indicated every effort was made to eliminate prematurity. The date selected for the performance of an elective repeat section was determined after careful evaluation of all factors such as age gestation, condition of the cervix, and fetal size. Elective section was not performed if the fetus was estimated to be less than 3,000 grams in weight. It was safer to wait for the onset of spontaneous labor in cases of doubt. This was the best way to avoid fetal loss due to prematurity.

In performing a repeat section it was advisable to use the same type of uterine incision previously used for this avoided the creation of another scar on the uterine wall. This also enabled the operator to excise any possible uterine defect which had been incurred in the previous operation and therefore assured a better repair; besides it was observed that disruption occurred more frequently when there was more than one type of scar (transverse and vertical) in the uterus.

The maternal mortality in repeat Cesarean section was 0.8 per cent and uncorrected perinatal mortality of 3.7 per cent.

There were seven dramatic ruptures and 14 uterine defects (dehiscence) encountered incidentally in the 265 patients who underwent a repeat section. 85.7 per cent of these patients admitted were already in labor on admission. A total of 13 ruptures (2.4 per cent) were therefore encountered, six following vaginal delivery and seven discovered on repeat section. In six of these patients, rupture occurred after one or two successful vaginal deliveries, indicating that success of vaginal deliveries following a section is no guarantee that rupture will not take place in subsequent pregnancies.

There were seven classical scars that ruptured in the 84 patients with history of classical section, an incidence of 8.33 per cent; and rupture of six low segment scars, an incidence of 1.5 per cent, in 441 patients with previous low sections.

During pregnancy, dramatic or symptomatic rupture of a previous scar almost invariably happened in the classical incision with high incidence of maternal and infant mortalities. Limitations of the classical incision to the minimum greatly reduced the incidence of symptomatic rupture prior to the contemplated date of repeat section. Low segment scars rarely produced symptomatic rupture prior to the onset of labor (observed in only one of our cases). Dewhurst and Pedowitz and Schwartz had similar observations.

Maternal mortality in rupture of the uterus was 7.6 per cent and perinatal mortality, 28.5 per cent. These figures showed a marked decrease from those given in a previous report from the Philippine General Hospital. This decrease was due to the following factors—(a) improvements in surgical technique and anesthesia; the frequent use of the low Cesarean section and the limitation of classical incisions, (b) early diagnosis and immediate surgery, (c) availability and liberal use of blood and antibiotics, (d) careful control of fluid and electrolyte balance, (e) better nutrition and health of patients and (f) better prophylaxis and treatment of postoperative complications.

Up to the present, there is no means of predicting accurately the behaviour of a uterine scar during the course of pregnancy and labor. This is still being studied further with the hope of discovering a reliable predictor in the near future.

RECOMMENDATIONS

The following recommendations are based on the observations and knowledge gathered in this study. There is need for a more liberal employment of repeat Cesarean section and allowing vaginal delivery in situations which are extraordinarily favorable for such

delivery. However, it is also recommended that the following criteria be considered by obstetricians who would want to hold on to the practice of allowing majority of patients to deliver vaginally after a Cesarean section.

(a) There should be no indications for repeat section in the current pregnancy and labor.

(b) There should be a previous lower segment section and knowledge of good surgical technique by the previous obstetrician. One should favor repeat section if the previous section was classical or unknown. This is based on the findings that rupture of a classical Cesarean scar is more frequent and disastrous to both the mother and baby during pregnancy and labor than lower segment scar rupture. This was also the observation of Dewhurst.

(c) There should be an uncomplicated postoperative course following the previous section, that is, no postoperative fever, wound infection, or dehiscence. It should be taken into consideration however, that an afebrile postoperative course does not guarantee a good scar.

(d) Placentography near term is of value in order to determine placental locations because of the possibility that implantation beneath the scar area is more prone to rupture, particularly of the classical scar. This was observed in three of the patients. Furthermore, more bleeding and unusually high fetal loss was manifested among those patients whose placenta was located beneath a uterine scar that ruptured.

(e) There should be a normal occiput presentation and average sized baby. One should perform a repeat Cesarean section in the presence of abnormal presentations. If the approximate weight of the baby is 3,500 gms. or more, and in the presence of any condition giving rise to overdistention of the uterus like multiple pregnancy and polyhydramnios one should favor repeat section, for excessive stretching may unduly weaken the uterine scar.

(f) The patient should be cooperative and have an intelligent understanding of the situation and should live within a reasonable distance from the hospital.

(g) There should be constant observation and care by a competent obstetrician.

(h) At least 500 cc. of blood, if not more, should be available.

(i) The operating room and staff should always be in constant readiness. There were 2 cases in these series where rupture occurred while waiting for the availability of the operating room due to other emergencies.

(j) There should be astute observation on the character of uterine contractions, and its effect on cervical effacement and dilatation, presence of abdominal tenderness, changes of the fetal heart tone, pulse rate and blood pressure.

(k) The second stage should be shortened by the use of prophylactic forceps.

(l) The practice of routine systematic manual exploration of the uterus immediately after vaginal delivery should be performed by a competent obstetrician. This gives valuable information regarding the presence of uterine defects, especially rupture of the uterus, before the patient develops shock. This also permits reliable assessment of the competency

of scar for future pregnancy and vaginal delivery. Hysterography performed three months after the original Cesarean section may be of value as a guide to the mode of delivery in subsequent pregnancy.

(m) A complete and accurate record of the patient's previous section especially surgical technique (including location of extra hemostatic sutures, hematomas, etc.) amount of blood loss and replacement, nutritional and health status of the patient should be readily available.

An obstetrician should, however, be very perceptive and flexible enough to terminate labor by performing an immediate Cesarean section whenever the following abnormalities are manifested by the patient: 1. Uterine dysfunction, 2. lack of full dilatation of cervix and descent of the head within 8 to 10 hours, 3. signs of fetal distress in utero, 4. abnormal bleeding, 5. abdominal tenderness, 6. bloody urine and 7. any other abnormalities of labor.

Whether to consider dehiscence as a type of rupture of the uterus or just a uterine defect is not clearly understood. Majority of these are minor wound deficiencies and some patients who have them are capable of a "safe" vaginal delivery. It is therefore recommended that a reclassification of the various types of uterine defects is necessary so that the description of them is clear to all.

This report is really a ticklish affair, in the sense that there are avid followers of both extremes as far as the management of a previously cesareanized patient is concerned. Almost all of them are backed up by their own experiences and observations. However, this study has shown that there is need for a more liberal employment of repeat Cesarean section and permitting vaginal delivery in situations which are extraordinary favorable for a safe vaginal delivery.

REFERENCES

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2. Kalrieder, F. & Krone, W.F.: Delivery Following Cesarean Section. *Clin. Obst. Gynec.* 2:1029-1042, 1959
3. Pedowitz P. & Schwartz, R.M.: True Incidence of Silent Rupture of Cesarean Section Scars. *Amer. J. Obst. Gynec.* 74:1071-1081, 1957
4. Poidevin, L.O.S. & Backner, V.Y.: Hystero-graphic Study of Uteri After Cesarean Section. *J. Obst. Gynec. Brit. Emp.* 65:278-283, 1958
5. Poidevin, L.O.S.: The Value of Hysterography in the Prediction of Cesarean Section Wound Defects. *Amer. J. Obst. Gynec.* 81:67-71, 1956
6. Ramoso-Jalbuena, J.: Postcesarean Scar Rupture. *Acta med. Philipp.* 13:303-314, 1956-1957
7. Ramoso-Jalbuena, J., Villanueva, J. & Ramos, H.: Perinatal Mortality in Deliveries after Cesarean Section (to be published).

8. Villanueva, J. & Ramoso-Jalbuena, J.: Rupture of Uterus. *Acta med philipp.* 8:235-243, 1952
9. Vilarama, A. & Galang, J.: Cesarean Section—Review of Cases in the Free Obstetrical Service, P.G.H. from 1921 to September 1930 inclusive. *J. Philipp. med. Ass.* 13:7, 1939

A Report on The 1962 Cholera (El Tor) Out-break in Taiwan

Dr. YIN-HO, SU

The out-break of Cholera (El Tor) in TAIWAN occurred in the summer of 1962 (17/July—4/September); it was of 49 days duration, and was most prevalent on the south-west coast; the main out-break was seen in the TAINAN area (227 cases, 59.3%), and the KAOHSIUNG area (108 cases, 28.2%), while the TAIPEI and TAICHUNG areas had only 34 cases and 14 cases respectively.

In this epidemic, 383 cases with 24 deaths were reported. Among them, 185 cases or 48.3% were male and 198 cases or 51.7% were female. The incidence rate was 3 cases for 100,000 population, and the case fatality rate was 6.2%. The highest case fatality rate was seen among children and aged men and women. The majority of cases occurred in the second week and further in the fourth week (after heavy rain in KAOHSIUNG area), more among housewives and farmers, and the highest incidence of this disease was in the age-group of 5-9 and 55-59.

The symptoms and process of this disease were comparatively mild and *Vibrio comma* in the faeces of patients disappeared within about ten days.

As soon as the first cases were discovered, the government started effectively to control environmental sanitation and finished the vaccination with Cholera vaccine of about 10,593,254 people, involving almost 80% of the total population of TAIWAN, and very fortunately after 7 weeks duration the out-break ceased.

El Tor Type Vibrio in The Recent Outbreak of Cholera

P.R. Aragon, M. D.

E.G. Famatiga, B.S Hyg.

On September 22, 1961, there were brought to the Philippine General Hospital two adults whose symptoms were compatible with that of cholera. The cases came from Pandacan, district of Manila but as far as can be ascertained, they were not related to each others. Before the patients were transferred to the communicable disease pavillon of the San Lazaro Hospital, the stool of Case No. 750191 was referred to us for the isolation and identification of enteric pathogens. We were able to isolate a vibrio whose morphological and biochemical properties are comparable with a pathogenic vibrio; and even more important is the fact that the isolate agglutinated with anti-V. comma serum. The epidemic that followed enabled us to study bacteriologically and serologically additional cases that substantiated our initial findings. This study consists of three parts viz., isolation and identification of the organisms, serological reaction of the cases, and observation of the organism in food and water.

Part I. Isolation and Identification of the Organisms

In the isolation and identification, out of one hundred twelve specimens from cases and contacts examined, twenty-six were found positive for vibrio the morphological and the biochemical characteristics of which place them in Group I by Heiberg's scheme of classification of vibrios. The results of the isolation procedure under different circumstances are summarized in Table I.

Table I

Isolation of Agglutinable, Hemolytic Vibrios
From Various Sources

Sources	Nature of Sources	Number Examined	Number Positive
San Lazaro Hospital (Manila and Suburb)	cases	41	11

Samar—Leyte Prov.	cases	41	13
National Montal Hosp.	contacts*	30*	2
Total		112	26

* 79 examinations were done on 30 contacts.

Table II shows the comparative efficiencies of Dieudonne and gelatin agar isolation plates used during the isolation procedure. Comparing the two, we found the gelatin agar plate very satisfactory. The material of gelatin agar are readily available, it does not need blood, easier to prepare, it does not need storage before use, is less inhibitory, and the vibrio colonies can be more easily spotted conforming with the finding of Smith (2). It can be seen in Table II that the 7 positive specimens picked up by the Dieudonne plate were also positive on the gelatin agar plate but there were 3 positives in the latter that were negative on Dieudonne.

Table II
Comparative Efficiencies of Isolation Plates

Isolation Plate used	Total Specimen examined	Growth on Plates		Positive	
		None	Present	for El Tor vibrio	Positive to non-Agglutinable vibrio
Dieudonne	40	12	28	7	2
Gelatin agar	40	0	40	10	0
Alkaline agar**	1	—	1	1	0
Total	41			11*	2

** used on first specimen.

* the 7 positive in Dieudonne plates were also positive in the gelatin agar so that only 11 were positive out of 41 specimens examined.

Slide agglutination test was done on the isolates for confirmation by emulsifying a loopful of a 24-hour growth on an agar slant in normal saline solution and mixing with it a drop of 1:30 dilution of O Group I rabbit anti-cholera vibrio serum. The presence or absence of agglutination was noted within 45 seconds. The slide agglutination test was repeated on isolates that agglutinated O Group I serum using a monospecific anti-cholera vibrio agglutinating serum at a dilution of 1:40 of the Ogawa and Inaba types. The isolates were agglutinated by anti-V. comma serum, type "O", Group I of Gardner and Venkatraman (8), and by anti "Ogawa" monospecific serum placing them under subtype "Ogawa". The isolates were also homolysin producers as shown by the tube homolysin test, their most significant characteristic which is almost exclusively found in the El Tor type vibrio that frequently causes epidemic in Indonesia, particularly the island of Celebes. Other tests such as soda-serum agglutination, sublimate precipitation, and agglutination after heating at 56°C in specific sera were done according to the procedure described by Tamanal (6,7), also for the purpose of identifying because of the

conflicting results of various workers (9,10,11) in connection with the hemolytic test. The results of these differential tests are shown in Table IV.

Table III shows the different types of specimens submitted to us and the results of the examination. In the rectal swab specimen, the character of the stools were not determined but in instances where stools were submitted, rice-water stool yielded the most number of positives. On the other hand, brownish to greenish stool, though diarrheic did not yield any positive result.

Table III
Isolation From Different Types of Specimen

Type of Specimen	No. of Specimen	No. positive for El Tor type vibrio
Rectal swab	47	7
Rectal swab on nutrient agar pH 9.2	17	13
Watery stool-colored brownish to greenish	17	0
Rice-water stool	7	6
Total	88	26

Table IV
Results of Soda-Serum-Agglutination, Soda-Sublimate-Precipitation and Agglutination of Heated Antigen

Organisms	Titer of Agglutinins		Soda-sublimate-precipitation	Bacteria Heated at 56°C for 3 hrs. plus serum
	Soda-serum agglutination	Nss-serum agglutination (control)		
1. <u>V. comma</u>				
Ogawa	1:8	1:16	no flocc.	Agglutination
Inaba	1:8	1:32	” ”	”
2. <u>Isolate (P.I.)</u>				
Laurente	1:32	1:8	Flocculation	”
C—39	1:32	1:8	”	”
Crosco	1:16	1:4	”	”
C—87	1:64	1:16	”	”
3. <u>El Tor (Celebes)</u>				
Bandung	1:64	1:16	”	”
Semarang	1:64	1:16	”	”
Purwakata	1:64	1:16	”	”
Tijiandur	1:64	1:16	”	”

T A B L E V
Agglutinative Titers of Sera From Cases Against *C. vibrio* and
El Tor Type *Vibrio* From Philippines and Celebes

Case No.†	Stool examination	Immuni- zation Status**	Period from onset to bleeding	Serum agglutinative titers for live organisms against									
				Cholera vibrio		El Tor type vibrio (P.I.)		El Tor type vibrio (Celebes)		El Tor type vibrio (Celebes)		El Tor type vibrio (Celebes)	
				Inaba	Ogawa	C-15***	C-19	Lau- rente	Ban- dung	Sema- rang	Purwa- kata	Tijan- dur	
C-51	+	none	10 days	1:160	1:640	1:160	1:80	x	x	x	x	x	x
C-52	+	"	17 "	1:640	1:1280	1:320	1:320	x	x	x	x	x	x
C-13	—*	"	13 "	1:20	1:20	1:20	1:20	—1:40	—1:40	—1:40	—1:40	—1:40	—1:40
C-16A	+	"	11 "	1:160	1:640	1:160	1:320	1:80	1:160	1:160	1:160	1:320	1:160
C-16B	—	"	20 "	1:60	1:640	1:320	1:80	1:40	1:160	1:160	1:160	1:160	1:160
C-24	+	"	9 "	1:320	1:1280	1:640	1:640	1:160	1:640	1:640	1:640	1:640	1:640
C-10	+	"	12 "	1:20	1:160	1:40	1:80	1:80	1:80	1:80	1:80	1:80	1:80
C-15	+	"	11 "	1:320	1:160	640	1:320	1:160	1:160	1:640	1:640	1:640	1:640
C-25	+	"	18 "	1:160	1:1280	1:320	1:160	1:80	1:320	1:320	1:320	1:320	1:320
C-53	+	"	23 "	1:40	1:320	1:40	1:20	1:40	1:40	1:40	1:80	1:80	1:40
C-17	+	" &	23 "	1:40	1:640	1:80	1:40	1:40	1:40	1:160	1:160	1:160	1:160
C-37	+	none	9 "	1:160	1:1280	1:160	1:80	x	x	x	x	x	x
C-54	?	"	? "	—1:20	1:160	—1:20	—1:20	—1:40	—1:40	—1:40	—1:40	—1:40	—1:40
C-55	—	"	12 "	1:40	1:320	1:40	1:40	1:40	1:40	1:80	1:80	1:80	1:80
C-56	+	"	10 "	1:20	1:320	1:20	1:20	1:40	1:40	1:40	1:40	1:40	1:40
C-101	+	"	3 "	1:40	1:40	x	x	1:40	1:40	1:160	1:80	1:160	1:80
C-102	+	"	6 hrs.	1:40	1:40	1:40	1:40	1:40	1:40	1:40	1:40	1:40	1:40
C-103	+	"	2 days	"	"	"	"	"	"	"	"	"	"
C-104	—	"	2 "	"	"	"	"	"	"	"	"	"	"
C-105	+	"	3 hrs.	"	"	"	"	"	"	"	"	"	"
C-106	+	"	1 day	"	"	"	"	"	"	"	"	"	"
C-107	+	"	4 "	"	"	"	"	"	"	"	"	"	"
C-108	+	"	—1 "	"	"	"	"	"	"	"	"	"	"
C-109	+	"	6 "	1:160	1:160	x	x	1:160	1:160	1:160	1:160	1:160	1:160

x not done
* positive for non-agglutinable vibrio
& one dose of vaccine

** for the past 6 months
*** isolate from patient and sera of patient

† C-101 to C-56—cases from Manila
C-101 to C-109—cases from Leyte and Samar

T A B L E VI

Agglutinative Titers of Sera From Non-cholera Cases Against *C. vibrio* and El Tor Type Vibrios From Philippines and Celebes

Non-Cholera Case	Immunization Status & dose*	Serum agglutinative titers for live organisms against											
		Cholera vibrio		El Tor type vibrio (P.I.)		El Tor type vibrio (Celebes)		El Tor type vibrio (P.I.)		El Tor type vibrio (Celebes)			
		Inaba	Ogawa	C-19	C-15	Lau-rente	Ban-dung	Sema-rang	Purwa-kats	Tijan-dur	Tijan-dur		
NC-1	+	1:320	1:160	1:40	-1:40	-1:40	-1:40	-1:40	-1:40	-1:40	-1:40	-1:40	-1:40
NC-2	+	1:320	1:320	-1:40	"	"	"	"	"	"	"	"	"
NC-3	+	1:1280	1:160	1:160	1:80	"	"	"	"	"	"	"	"
NC-4	+	1:1280	1:160	-1:40	-1:40	"	"	"	"	"	"	"	"
NC-5	+	-1:40	-1:40	"	"	1:80	1:80	1:80	1:80	1:80	1:80	1:80	1:80
NC-6	-	1:640	1:40	"	"	"	"	"	"	"	"	"	"
NC-7	-	1:80	1:40	"	"	"	"	"	"	"	"	"	"
NC-8	-	-1:40	1:80	"	"	"	"	"	"	"	"	"	"
NC-9	-	1:80	-1:40	"	"	"	"	"	"	"	"	"	"
NC-10	-	-1:40	"	"	"	"	"	"	"	"	"	"	"
NC-11	?	1:320	1:160	"	"	"	"	"	"	"	"	"	"
NC-12	?	1:160	-1:40	"	"	"	"	"	"	"	"	"	"

* either cholera or cholera-typhoid-paratyphoid A

Part II. The Serological Reaction of the Cases

Preliminary epidemiological investigation showed that majority of the cases during the early part of the epidemic have had no cholera immunization within the past six months. The absence of immunization makes the sera of cases suitable for study of the serological reactions of the cases. To determine if specific antibodies are engendered against the El Tor type vibrio, the agglutinative titers of the sera were determined. This established the connection between the isolate and the disease condition and confirmed the identity of our isolate. The serologic test also confirms the clinical and the bacteriological diagnosis.

In this study, we determined the agglutinative titers of sera from cases and sera from non-cholera cases against *V. comma* subtypes Inaba and Ogawa; El Tor type vibrio Philippine isolates—C-15, C-19, and Laurente; and El Tor type vibrio from Celebes—Bandung, Semarang, Purwakarta and Tajiandur. Since we were not aware of the published normal titer for El Tor type vibrio, but because of the similarity of the antigenic make up of the El Tor and *V. comma* organisms, we interpreted our results using the agglutinative titer against *V. comma* of normal (cholera-free unvaccinated) individual which has been reported anywhere between 1:10 by Grieg to 1:80 by Krishnan and Dutta according to the review by Pollitzer (5). We also used the uninoculated titers as baseline. The significant increase in the titers against El Tor Celebes vibrio and our isolates as can be seen in Table V are good evidences that the cases are reacting to the isolated organisms, and that there is similarity between the organisms from the two places. The much higher titers against the "Ogawa" subtype of *V. comma* than the "Inaba" subtype confirms that our isolates belong to the subtype "Ogawa". Although there were increase in titers against Inaba, it only proved the existing antigenic overlapping between the two subtypes.

The titers of vaccinated and non-vaccinated non-cholera cases as presented in Table VI showed that the vaccine was able to increase the agglutinative titer against *V. comma* but there was practically no effect against the El Tor type vibrio. The observed higher titers against *C. vibrio* than the isolated El Tor vibrio among cases may be explained by: (1) the individuals have had cholera immunization sometime before (we only inquired about an immunization six months previously) and that the present infection had the effect of a booster dose, accepting that the agglutinin reaction in the serum to *C. vibrio* and El Tor vibrio are not necessarily similar in kind and degree; (2) the long standing question in the bacteriology of *V. comma*, viz. changes in agglutinability according to Harvey (21), who observed that "There is a greater tendency to find agglutinable vibrio or less agglutinable vibrio during convalescence than when symptoms are still at their height.

Part III. The El Tor Vibrio in Water and Food

The mode of infection in Asiatic cholera is through ingestion of contaminated food and water. In the Philippines, as elsewhere, drinking water, sea water, and sea food

have played an important role in many epidemics. In the present epidemic, the earliest cases came from Manila, a seaport, and towns bordering Manila Bay. In the majority of instances, the towns near the sea were first affected and had a higher attack rate. In the Manila outbreak, the early cases usually gave a history of eating raw, fine, soft-shelled shrimp locally called "alamang". Because of the above considerations, attempts were made to isolate the El Tor vibrio in water samples from various sources and from sea foods. In this connection, we did a survival study of the V. comma and of the El Tor vibrio in: raw, filtered (membrane filter HAWP), and sterilized (autoclaving) sea water.

In the outbreaks involving Manila and the nearby towns, we were not able to isolate the vibrio from sea food, sea water sewage. The short survival time might explain our negative findings. After considering the time relationship between the evolution of the epidemic and our examination of water we found that we did the latter quite late. In the Samar-Leyte outbreak, however, the sea water was examined early, probably the reason why we were able to isolate El Tor vibrio in two samples of water. The result of the examination of water and sea food from various epidemic areas is presented in Table VII.

Table VII
Result of the Examination of Water and Seafood
from Various Epidemic Areas

Source and Type of Sample	Number of Specimens examined	Number Positive	Remarks
A. Water			
1. Sea water	41	2	Positive water samples
2. Surface water	21	0	(1) Barrio Rawis, Laong, Samar, taken Nov. 29, 1961
3. Artesian well	10	0	
4. Sewage	12	0	(2) Sea water Catbalogan wharf, Dec. 16, 1961
5. Unknown	5	0	
B. Seafood (shrimps, sea weeds, oysters, snails, crabs, fish and fish washings)			
	38	0	
Total	127	2	

With regards to the survival study, under the condition of the test, the El Tor vibrio survived for a longer period than V. comma in all the types of sea water studied. Both organisms survived longest in autoclaved sea water and shortest in raw water. In filtered

sea water the El Tor vibrio survived for 18-21 days while V. comma could no longer be demonstrated after four days. The result of this experiment is shown in Table VIII.

Table VIII
Survival of C. vibrio and El Tor Type Vibrio in Raw,
Filtered and Autoclaved Sea Water

Type of Sea Water	Inoculum	Estimated organism/ml at start	Result of Experiment After (Days)								
			4	8	10	15	18	21	28	34	39
Raw	C. vibrio Ogawa	5900	-	-	-	-(F)	×	×	×		
Raw	El Tor vibrio	1800	+	-	-	-	-	-(F)	×		
Filtered	C. vibrio Ogawa	1300	-	-	-	-(F)	×	×	×		
Filtered	El Tor vibrio	3600	+	+	+	+	+	-	-	-(F)	×
Autoclaved	C. vibrio Ogawa	1100	+	-	+	+	-	-	-(F)	×	×
Autoclaved	El Tor vibrio	2100	+	+	+	+	+	+	+	+	+

+ Positive for inoculated organism

- Negative for inoculated organism

(F) Filtration through membrane filter of the rest of the water

× No more examination done after filtration of the whole remaining amount of water

In another experiment wherein the survival time of El Tor was determined in sea water and shallow well water, the result is in Table IX. The survival of El Tor vibrio in sea water and open shallow well water showed that the organism survives longer, 37 days, in well water than in sea water, 3-4 days only. Further studies in the survival of the El Tor vibrio in sea, well and river water together with an estimate of the bacterial density, pH and chloride content is shown in Table X.

Summary and Conclusion

1. The cholera epidemic in Manila which began during the last week of September 1961 is caused by El Tor type vibrio, subtype "Ogawa", by virtue of its morphological, physiological, and hemolytic properties, and agglutinability in O Group 1 anti-V. comma.
2. The increase of agglutinative titers of the sera from cases against the isolates and Indonesia isolates in almost the same titers showed that the organisms from the two places are identical.
3. The serological results confirmed the identity of the isolates as subtype "Ogawa".
4. The high serum titers among the El Tor cases against V. comma emphasize the antigenic similarities of V. comma and El Tor vibrio.
5. An incidental finding in this study is the absence of agglutinins against El Tor type vibrio but the presence of agglutinins against V. comma in persons vaccinated with cholera vibrio vaccine.
6. The isolation of El Tor type vibrio from two samples of sea water from towns in Samar might be the explanation for the coastal origin and spread of the infection.
7. By the condition of our experiment, the El Tor type vibrio seems to survive longer than V. comma in well water. Like the V. comma, El Tor vibrio survives for a shorter period in raw sea water than in filtered or autoclaved water.

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Japanese Contributions to the Knowledge of Cholera due to El Tor Vibrios

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The classical or the Egyptian or the Zam-Zam type El Tor vibrios have generally been known to be almost non-pathogenic. The Celebes type El Tor vibrio caused lethal infection, namely, so-called paracholera. But it used to prevail only in endemic form localized in Celebes Island, so that the Committee on International Quarantine of W H O resolved in 1958 to exclude paracholera from the term cholera defined as the quarantinable disease in the International Health Regulation (I H R). Under such circumstances, not much outcome of the studies on El Tor cholera or vibrios has been so far accomplished yet in Japan. However, I will try to review it for your reference today.

I. Bacteriological.

1) Immunological Types of El Tor vibrios.

a. Early work by Nobechi.

In my studies on cholera vibrios from 1921 through 1923, I included a strain of the classical El Tor vibrio, secured from Koch Institute, among the working strains for comparative study purpose. This strain revealed the same bacteriological characteristics with the authentic cholera vibrios, except producing a stronger hemolysin than the latter, and immunologically corresponded exactly to the Inaba type.

b. Finding as to 65 Epidemic Strains isolated in 1961.

We collected 65 strains of EL Tor vibrios isolated from 1961 epidemics in Celebes, Java, Sarawak, Hongkong and Philippines. The bacteriological examination carried out as to these strains by Hamano, Member of the newly established Research Committee on El Tor Cholera (RCETC) elucidated that all displayed characteristics compatible with vibrio el tor of the Ogawa type, except two strains which belonged to the Inaba type. We are also in possession of a strain of El Tor vibrio of the Hikojima type isolated in Djakarta in 1961, and forwarded by Dr. Felsenfeld on my request.

2) Differentiation of Classical Cholera and El Tor Vibrios.

a. Hemolysis Test.

Reporting their own observation of the strains of the authentic cholera vibrios possessing as strong hemolysin producing power as the vibrio el tor, Kabeshima (1913), Nobechi (1923), Chairman of RCETC, and Sato (1946), RCETC Member, discussed that this test could not be held as the absolute key to distinguish vibrio el tor from vibrio cholerae. Ushiba (1962), RCETC Member, demonstrated that El Tor strains displayed considerably wide variation in the intensities and the timely courses of their hemolysin production.

On the other hand, Hamano found that, following Greig's method, 65 El Tor strains as aforementioned and 40 more strains to be described later, unanimously displayed positive hemolysis test, while all of the strains of the classical cholera vibrio, taken as control, the negative. The hemolysis test may, thus, be regarded as one of the practical methods for differentiation of those two kinds of vibrios.

b. Bacteriophage sensitivity test.

Takeya (1962), RCETC Member, confirmed that the phage sensitivity test against Mukerjee's Type IV phage was one of the best methods to distinguish El Tor from the cholera vibrios, in spite of some exceptions contrary to Mukerjee's statement.

3) Differentiation of the strains of the Celebes Type and those of the others among El Tor Vibrios.

In execution of international quarantine work, methods to distinguish the Celebes type vibrio el tor strain from the less or non-pathogenic El Tor vibrio strains of the other types are demanded. Takeya (1962) found that all of the 67 El Tor strains of the Celebes type isolated from the present epidemics in SEA, produced without exception, a specific bacteriophage affecting one particular strain (218) of Inaba type vibrio cholerae, whereas none of the 16 strains of El Tor vibrio of the other types, including 3 Ubol type strains and 5 classical El Tor vibrios, did so. This lysogenesis phenomenon will meet the aforementioned need for differentiation of the types of El Tor vibrios.

4) Findings as to the Isolates from 40 Carriers detected at Japanese Quarantine Stations in 1962.

Hamano and his collaborators (1962) found that all of the Isolates from 40 Carriers discovered at Japanese Quarantine Stations in 1962, were identified with the El Tor vibrios of the Ogawa type.

II. Epidemiological.

1) Early Perception of the Pandemic Nature of El Tor Cholera in September, 1961.

September 25, 1961, while WHO weekly reports had been announcing that a dual epidemic of cholera was prevailing in SEA, namely, paracholera in Mid-

Java and classical cholera in Sarawak, Macao and Hongkong, I visited the Epidemiological Intelligence Station of WHO in Singapore and exposed to Dr. Yung, Director of EIS, my view that the prevailing epidemics of cholera in SEA must have been a unitary prevalence of El Tor cholera originating from Celebes. Requested by Dr. Yung, accordingly, I addressed him a memorandum recommending to carry out the hemolysis test for differentiation of the kinds of the prevailing epidemic strains, which had been neglected so far in the medical institutions concerned there. The results of the hemolysis test carried out by them accordingly, were reported to have turned out positive. So, it was proved that my supposition mentioned above had been warranted. Thus, I would be the first person to have perceived and to have caused to prove the pandemic nature of the Celebes type El Tor cholera. Because of the masking of this nature, the cholera el tor must have been taken in the past for an endemic disease localized in Macassar Peninsula of Celebes Island.

2) Rates of Carriers against Patients in El Tor Cholera Epidemics.

On the rates of carriers to patients of the classical cholera, Ishiwara and Noda reported coinciding figures 2:1 in Tokyo and in Osaka respectively. In contrast to this, Japanese Navy found that the corresponding rates of El Tor cholera at two places in Celebes in 1944 were 4:1 and 6:1. In the event of Mikage Maru, which brought 22 El Tor carriers to Port Moji in 1962, the rate of the healthy carriers to the convalescent ones was 19:3. Such greater incidence of carriers in relation to the patients in the case of the El Tor than in those of the classical Cholera, would be the cause of the greater tendency of the former to take an apparent sporadic form than the latter.

III. Preventional.

1) Proposal to include El Tor Cholera into the Term "Cholera" in IHR.

Although as stated above, I already succeeded to make the local institutions in the South, to prove the epidemics prevailing in Sarawak and Hongkong in 1961 as the unitary pandemic due to the El Tor vibrio of Ogawa type, identical with the causative organism of the Indonesian epidemics, I secured 18 epidemic strains from Java, Sarawak and Hongkong, and had them ascertained by NIH and IID to be El Tor strains of the Ogawa type without mistake. Hereupon, I advised the MHW to propose to WHO to cancel the aforementioned resolution made by CIQ in 1958 which excluded the so-called "Paracholera" from the term "Cholera" in IHR, and further to have the former reincluded into the latter, and still further to introduce a logical appellation for the cholera due to El Tor vibrio in stead of "paracholera". Due actions were taken accordingly, and our proposal was fully adopted on the 15th WHA.

2) Proposal to amend IHR so as to enable to cheque the importation of cholera carriers.

The MHW proposed to WHO last fall to amend the present IHR, which is based upon the principle to cheque the importation of the patients and not the carriers as far as cholera is concerned, into such a form that enables to cheque the carriers importation. This has not been approved yet, but drew the attention of various countries to this problem. And it would not be long before this proposal would also be adopted duly.

Constitution & By-Laws

(Amended 1961)

C M A A O

**CONFEDERATION OF MEDICAL ASSOCIATIONS IN ASIA AND
OCEANIA CONSTITUTION AND BY-LAWS (AMENDED 1961)**

I. NAME—The Association shall be known as the Confederation of Medical Associations in Asia and Oceania (CMAAO)

II. OBJECTIVES—

- (a) To promote closer ties among the national medical organizations and among physicians in countries of Asia in particular and of the world in general by personal contact and all other means available.
- (b) To organize an exchange of information on matters of interest to the medical profession in Asia and Oceania.
- (c) To maintain the honor and protect the interest of the medical profession.
- (d) To study and report on the professional problems which confront the medical professions in Asia.
- (e) To assist all people in Asia and Oceania to attain the highest possible level of health.
- (f) To establish relations with and to present the views of the medical profession in Asia to the World Health Organization, World Medical Association, UNESCO, and other appropriate bodies.

III. MEMBERSHIP—The unit of membership shall be the national medical association of any country or medical association in any territory located in Asia and Oceania which can be recognized by the General Assembly as the representative of the Medical profession of that country or territory.

IV. TERMINATION—A member-association shall cease to be a member in any of the following ways:

- (a) By resignation, subject to the conditions prescribed by the By-Laws.
- (b) By default of payment of fees for membership as may be prescribed by the By-Laws.

V. FEES—Each member-association shall pay to the CMAAO an annual membership fee, the amount of which shall be prescribed by the General Assembly.

- VI. GENERAL ASSEMBLY, ITS POWERS—It shall be composed of the officers of the CMAAO, ex-officio, the members of the Council, and delegates from the Member association. It shall exercise general control and direction of the policy and affairs of the association.
- VII. DELEGATES AND ALTERNATES—Each Member-association shall be entitled to appoint two delegates who shall ipso facto be members of the General Assembly. Each Member-association shall also be entitled to appoint Alternate Delegates who may attend meeting of the General Assembly and may act as delegate, provided that a Member-association is not represented in the Assembly at any time by more than two speaking and voting delegates.
- VIII. MEETING—The General Assembly shall meet at least once in two years.
- IX. The MODE OF CONVENING meetings of the General Assembly and the proceedings thereat and relating thereto shall be such as may from time to time be prescribed by the By-Laws.
- X. DECISION—Resolutions carried in accordance with the following provisions shall be deemed to be decisions of the CMAAO.
- (a) Notice to submit to the General Assembly a resolution relating to any amendment of or addition to the Articles shall be given to the Secretariat not less than six months before the meeting at which it is to be considered. Such resolution shall be deemed a decision of the CMAAO if it is carried by a majority of not less than two-thirds of the votes given thereon in the manner prescribed by the By-Laws.
 - (b) Notice to submit to the General-Assembly a resolution relating to any amendment of or addition to the By-Laws shall be given to the Secretariat not less than one day before the meeting or session at which it is to be considered. Such a resolution shall be deemed of the confederation if carried by a simple majority of the votes given thereon in the manner prescribed by the By-Laws.
- XI. OFFICERS—There shall be the following officers of the Confederation: President, a President-Elect, an immediated Past President, a Chairman of Council, and a Treasurer. The officers shall be elected in such a manner and shall hold office for such term and shall have and enjoy such duties, powers, and privileges as may be determined from time to time by the By-Laws.
- XII. OFFICIALS—There shall be a Secretariat and such officials as may be determined by the General Assembly.

- XIII. COUNCIL—The General Assembly shall appoint at each regular meeting a Council which shall be composed of the President, the President-Elect, the Immediated Past President, and the Treasurer, all ex-officio and 4 members of the General Assembly elected in the manner and for the period prescribed in the By-Laws.
- XIV. It shall be the duty of the Council to carry into execution the resolutions passed by the General Assembly and to administer the affairs of the Association in accordance with the Articles and By-Laws.
- XV. COMMITTEES—Committees may be appointed in such manner and have such powers as may be prescribed by the By-Laws or as the General Assembly or the Council may think proper.
- XVI. LANGUAGE—The English language shall be the official language of the Confederation.
- XVII. JOURNAL—A Journal (or Bulletin) under the title of the Journal (or Bulletin) of the CMAAO may be published periodically.
- XVIII. ANNUAL AND FINANCIAL REPORTS—The Council shall publish and submit to the General Assembly when this body meets, for adoption and approval a report on the general state and proceedings of the Association for the interim period between meetings, a balance sheet and financial statement for the past year audited by a professional accountant, and an estimate of the probable income and expenditure of the Association for the coming year.
- XIX. DISSOLUTION—A decision to dissolve the Confederation shall require the consent of at least two-thirds of the Member-associations. It shall be taken at a meeting of the General Assembly specially called for the purpose. If two-thirds of the Member-associations are not represented at that meetings, a referendum of member-associations shall be taken on the question of dissolution and on the method of dealing with the funds of the Association in the event of dissolution.

BY-LAWS

MEMBERSHIP

1. MEMBERSHIP APPLICATION—An association desiring to become a constituent member of the CMAAO shall apply for election in writing to the Council which, after appropriate inquiry, shall make recommenda-

tions for the admission or rejection of the application to the next meeting of the General Assembly.

2. Only one national medical association shall be recognized in each country or territory.
3. REGISTER—A register of Member-associations shall be maintained by the Council at the Association's Office.
4. OBLIGATIONS—Each Member-association shall:
 - (a) do all in its power to promote a knowledge of, and an active interest in, the objectives and work of the CMAAO.
 - (b) reply to all inquiries and questionnaire from the Council as quickly as possible or within the time limit specified by the Council.
 - (c) keep the Council informed of any events or developments in its country of interest to the CMAAO.
5. DUES—Dues or voluntary contributions as determined by the General Assembly shall be due within three months from the date of the last General Assembly, [REDACTED], [REDACTED], [REDACTED].
6. (Delete)
7. TERMINATION OF MEMBERSHIP—No Member-association shall except in the case of default in payment of dues, cease to be a member without having given six months' previous notice in writing of its intention to the Council and without having paid all arrears of subscription, if any, due from it.

GENERAL ASSEMBLY

8. GENERAL ASSEMBLY—The General Assembly shall determine the place and time for each succeeding meeting. The annual meeting shall as far as possible be held in a different country each year.

9. SPECIAL MEETINGS—A special meeting of the General Assembly shall be convened at any time by the President on the request of the Council or on the request of not less than 4 Member-associations or as subsequently determined by the General Assembly.
10. At least three months' notice of special meetings shall be given to the members of the General Assembly. The notice shall state the place and purpose of the meeting.
11. No business shall be dealt with by a special meeting of the General Assembly other than that for which it is specially convened.
12. OBSERVERS—Member-Associations shall have the right to send observers, without privileges of speaking or voting, to meetings of the General Assembly. The Council shall have power to invite at its discretion other organizations interested in the practice of medicine in Asia to send observers.
13. BUSINESS—The Business of the regular meeting of the General Assembly shall be:
 - (a) To elect or install a President of the Association;
 - (b) To elect a President-Elect, a Treasurer as by the Articles or the By-Laws may require to be elected;
 - (c) To appoint, when necessary, such officials of the Confederation as may be determined under Article XII of the Constitution and fix their remuneration;
 - (d) To elect members of the Council as required by By-Laws 29 and in accordance with procedure laid down in Standing Orders;
 - (e) To appoint a place and time at which the next regular meeting shall be held;
 - (f) To consider and determine application for membership;
 - (g) To fix the annual dues;
 - (h) To consider the Annual Financial Statement and the Balance Sheet presented by the Council and to arrange for such action to be taken thereon as may seem appropriate;
 - (i) To instruct the Council concerning investigations to be taken in the pursuit of the objects of the Association;
 - (j) To consider such resolutions as can properly be considered by the General Assembly having regard to the objectives of the Association and as have been submitted by Member-associations with the appropriate period of notice as laid down in Article 10;
 - (k) To appoint an auditor and to fix his remuneration.

14. **AGENDA**—The Agenda for the General Assembly shall be prepared by the Council, which has power to decide whether or not a resolution submitted by a Member-association fails within the objective of the CMAAO.
15. **NOTICE OF MOTIONS**—Resolutions requiring a period of notice as laid in Article 12 (a) and (b) shall be circulated by the Council before the meeting to all Member-associations for their consideration.
16. **PRESIDING OFFICER**—The president of the Association shall preside at meeting of the General Assembly. In the absence of the President the meeting shall appoint a presiding officer from its number.
17. **VOTING**—All members of the General Assembly shall be entitled to vote at meetings of the Assembly provided that, except in the election of officers, a member of the Council shall not be entitled to vote in the Assembly unless he is a Delegate. Each Delegate shall have one vote, provided that if a Member-association is represented in a meeting of the General Assembly by only one Delegate that the Delegate shall have two votes.
18. Voting shall be by show of hands, unless, before the vote is taken, 4 members present request that the vote be taken by secret ballot.
19. In speaking and voting upon any matter, the Delegate of a member-association shall have regard to the preponderance of opinion of the association he represents.
20. The presiding officer shall in the case of equality of voting have a vote, but shall not otherwise be entitled to vote.
21. **REFERENDUM**—If one-third of the Member-associations, whether represented or not at the meeting, request within two months of the date of the meeting that a decision of meeting of the General Assembly which was carried by a simple majority and less than two-thirds of the Member-association shall be submitted to a referendum of all Member-associations, the Council shall take steps to obtain by correspondence the votes of each Member-association.
22. A decision which is the subject of a referendum shall have no operation unless and until it shall have been approved on the referendum by at least three-quarters of the Member-associations who have answered the referendum, provided that in no case shall the operation of the decision be delayed for more than eight months from the date of the meeting.

23. LANGUAGE—The discussions at the meetings of the General Assembly shall be conducted in English and if a Delegate wishes to speak in the Assembly in any other language he shall be permitted to do so provided that he arrange for its immediate translation into English.
24. MINUTES—The Secretariat shall keep Minutes of each meeting of the General Assembly, which shall, after confirmation by the presiding officer, be transmitted to the Council.
25. ADJOURNMENT—The presiding officer shall have power to adjourn the meeting from time to time and from place to place.
26. COUNCIL—Each elected member of the Council appointed by the General Assembly shall hold office for four years and at the end of that term shall be eligible for re-election. Members shall retire in rotation. At the first annual meeting two members shall be elected for four years and two for two years.
27. Each term of office shall be calculated from the close of the annual meeting of the General Assembly at which the election is made.
28. CHAIRMAN AND VICE-CHAIRMAN—The Council shall elect a Chairman and a Vice-Chairman from its own number. The Chairman, or in his absence the Vice-Chairman shall preside over meetings of the Council. If the Chairman and Vice-Chairman are both absent the members of the Council shall elect one of their number to preside over the meeting.
29. MEETINGS—The Council shall meet at least once a year and at such other times as it may deem necessary. Meetings shall be held at such place and upon such notice as the Chairman may appoint.
30. QUORUM—No business shall be transacted at any meeting of the Council unless at least four members be present or as may be subsequently directed by the General Assembly.
31. BUSINESS BY CORRESPONDENCE—The Chairman shall have the power to decide what business may be conducted by correspondence and what by meetings of the Council.
32. SPECIAL MEETINGS—The Chairman of the Council may if he thinks fit and shall upon receiving a request signed by not less than four members of the Council and specifying the business for which a special meeting is required call together a special meeting of the Council. The place at which the special meeting shall be held and its purpose shall be specified in the notice calling the meeting. At least two months notice of such meetings shall be given to the members of the Council.

33. No business shall be transacted at the special meeting other than that for which such meeting is called.
34. VACANCIES—The Council shall have power to fill casual vacancies among its number until the next election of members of the Council.
35. VOTING—Voting shall be by show of hands, and a simple majority shall be sufficient to carry a resolution.
36. MINUTES—The Secretariat shall keep Minutes of the proceedings of each meeting of the Council.

DELEGATES

37. DELEGATES—Each Member-association shall notify the Secretariat of the names and addresses of its delegates and alternate delegates appointed in accordance with Article 9, and shall also notify any subsequent changes.
38. QUALIFICATIONS—A Delegate shall be a person who is medically qualified and a member of the association he represents.
39. DUTIES—On returning from a meeting of the General Assembly, Delegates shall report to their respective associations on the proceedings of the meeting. The Council may from time to time issue instructions to Delegates.
40. EXPENSES—The expenses of Delegates attending meetings of the General Assembly shall not be charged upon the funds of the CMAAO.
41. FINANCE—The Treasurer shall receive all moneys payable to the Association, discharge all accounts which have been ordered by the Council to be paid and keep accounts and submit them to the Council at each of its meetings.
42. The accounts of the Association shall be kept at the office of the Treasurer. Any Member-Association, through its Delegates, may inspect the accounts.
43. The financial year of the Association shall be the calendar year.

SECRETARIAT

44. SECRETARIAT—The expenses of the Secretariat shall be defrayed out of the general funds of the Association on the periodical production to the Treasurer of vouchers stating the expenses incurred.

45. OFFICERS: President, President-Elect,—The President of the Association shall be elected at the regular meeting of the general assembly and shall enter upon the duties of his office at the next regular meeting, and until then shall bear title of President-Elect.
46. CHAIRMAN OF COUNCIL—The Chairman of the Council elected under By-Laws 13 shall be eligible for re-election from year to year. For one year after the end of his period of office he shall be a member of the Council ex-officio.
47. TREASURER—The Treasurer shall be elected at the regular meeting of the General Assembly. He shall hold office for 4 years and be eligible for re-election. During his term of office he shall be member of the Council ex-officio.
48. VACANCIES—In the event of the death or resignation of an officer during his term of office the Council shall make such appointment or other provision as it may deem expedient for the discharge of the duties of the concerned until the next regular meeting of the General Assembly.
49. OFFICIALS—Officials of the Association appointed by the General Assembly under Article 12 of the Constitution shall be medically qualified. They shall hold office for such periods and receive such remuneration as the General Assembly may from time to time determine and may be dismissed by the General Assembly.

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President: Prof. N. Shimada, Keio Univ.

August 26-28, 1963 Hakone, Japan

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3. BLOOD PROTEINS and THEIR BIOCHEMICAL and CLINICAL PROBLEMS
4. PRESERVATION OF BLOOD and BLOOD TRANSFUSION SERVICE

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August 26: Registration, Inaugural Session and Seminar, and Reception Party

August 27: Seminar and Recreation

August 28: Seminar, Closed Session and Farewell Party

The opening time of all seminars and sessions will be announced later.

Presentation of Papers:

Those wishing to present papers at the Congress must send abstracts within 500 words in English of all papers with a application form to the Program Committee on or before May 1, 1963. The time to be allowed for each presentation will not exceed 10 minutes at the tentative plan.

Congress Fees:

- a. Member of the Asian Congress of Blood Transfusion
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- b. Family
5,500 Yen (ca. 15 US Dollars)

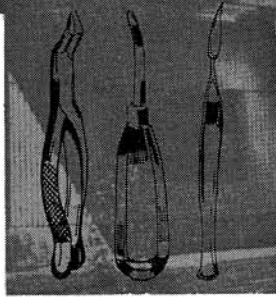
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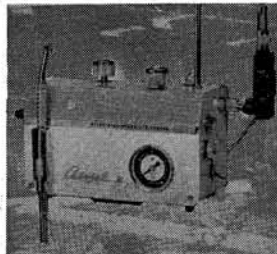
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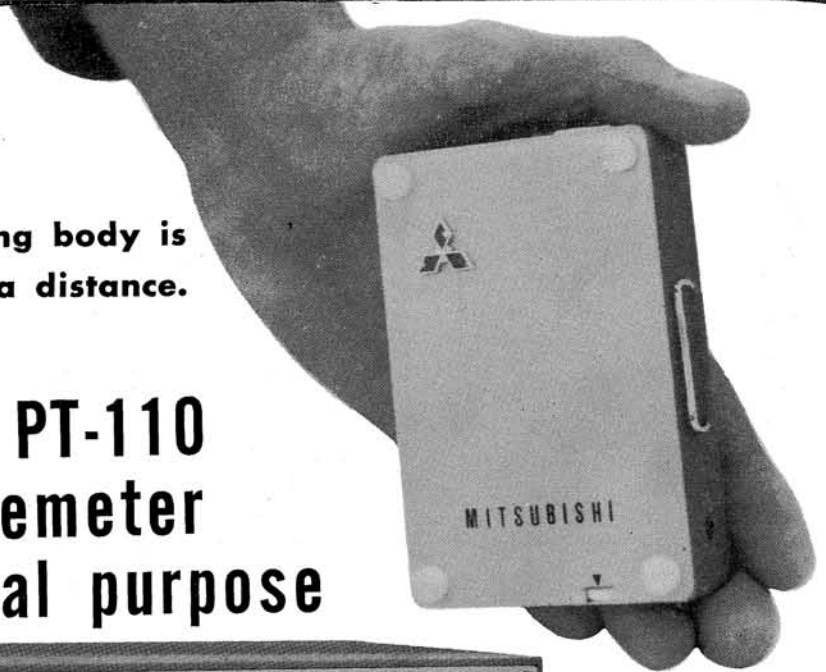
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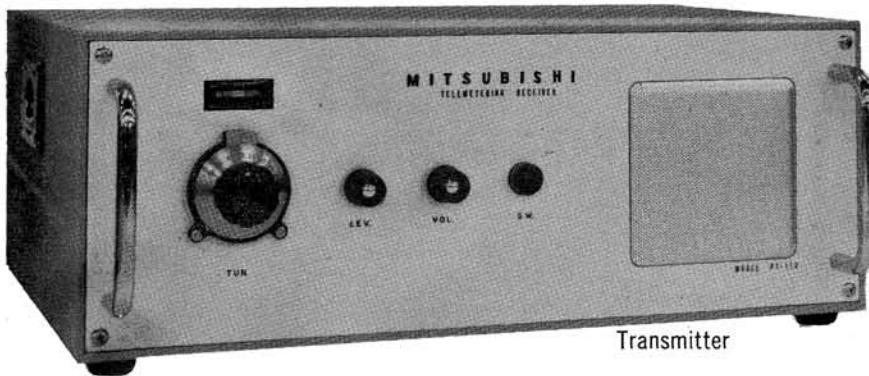


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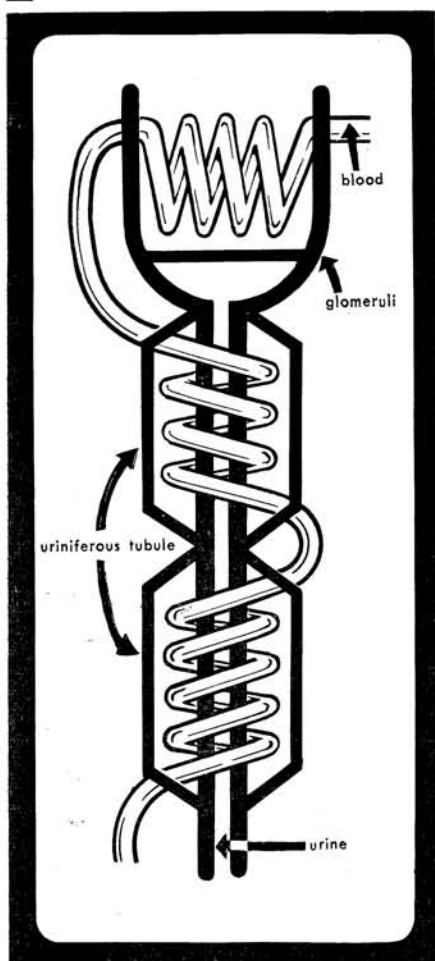
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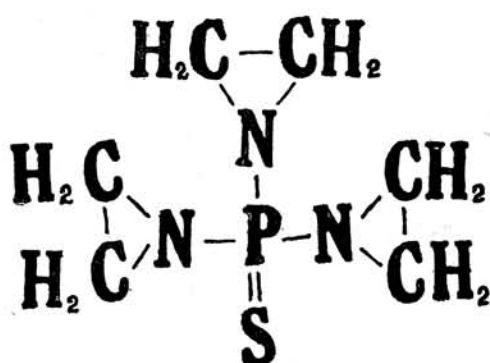
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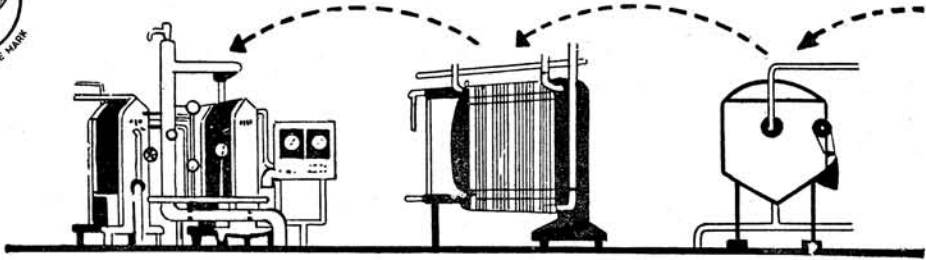
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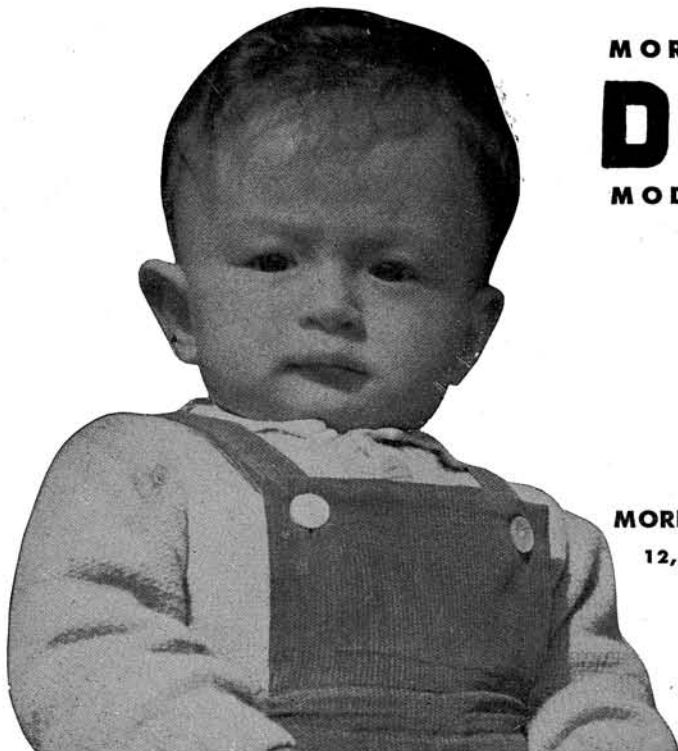
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Acid-stable Digestive Multienzyme.

With Proteolytic, Amylolytic Activities.

Containing also Lipase, Cellulase, Maltase

Inulase, Pectinase, Lecithinase, etc.

Normally Employed in Digestive Aids.

Less Hygroscopic



TAISHO PHARMACEUTICAL CO., LTD.

TOSHIMAKU TOKYO JAPAN

An Antitumor Antibiotic

Mitomycin

Mitomycin C is a purple crystalline antitumor antibiotic produced by a new species of streptomyces named Streptomyces caespitosus.

It is strongly active in inhibiting the nuclear divisions, and in destructing cancer cells.

CHARACTERISTICS

1. Broad Antitumor Spectrum

According to the experimental data ever published, Mitomycin C has the most wide antitumor spectrum and most powerful destructive activities against cancer cells among the antitumor substances which have ever been reported.

2. Excellent Therapeutic Results

As anticipated from the above-mentioned results with experimental tumors of animal, Mitomycin C was proved to have potent and wide-ranged activities in clinical studies, too. Remarkable objective effects as followings were observed in many cases: diminution or disappearance of tumors, metamorphosis or disappearance of tumor cells in pleural or ascitic fluid, diminution or disappearance of metastatic lesions, etc

Subjective effects such as improvement of appetite, sense of wellbeing and complete or partial relief of pain were often observed. It is also expected that Mitomycin C will be effective even in those patients who have become resistant to other chemotherapeutics and radiation therapy.

3. Less Side Effects

Mitomycin C can conveniently be used as it causes subjective side effects such as nausea, vomiting and poor appetite only in limited cases.

Decrease in number of leucocytes and blood platelets are subjective side effect of this medicine, but the decrease is usually recovered rapidly by discontinuing the administration.

4. Prevention of Recurrence After Operation

Excellent follow-up results are being indicated in the postoperative recurrence.

INDICATIONS

Mitomycin C is indicated for improvement of subjective and objective symptoms of following diseases:

Carcinoma: Stomach cancer, uterine cancer, cancerous peritonitis, breast cancer, liver cancer, lung cancer, pancreas cancer, intestinal cancer, maxillary cancer, skin cancer.

Sarcoma: Reticulosarcoma, lymphosarcoma, melanosarcoma.

Leukemia: Acute leukemia, chronic leukemia

Hodgkin's disease;

Cholionepithelioma malignum

PACKAGE

Ten ampules each containing 2mg(potency)



KYOWA HAKKO KOGYO CO., LTD.

OHTEMACHI BLDG., OHTE-MACHI, CHIYODA-KU, TOKYO, JAPAN



TRPD ALINAMIN

Thiamine Propyl Disulfide

New thiamine derivative with wide range of utilization compared with common vitamin B₁.

CHARACTERISTICS

- Absorption from intestines not limited ; oral therapy effective as injection.
- Transferred and stored in various organs in high level and retained over a long period.
- Easily converted into cocarboxylase.
- Not destroyed by thiaminase.

INDICATIONS

- Thiamine deficiency.
- All types of neural disturbances, neuritis, neuralgia and rheumatism.
- As adjuvant in all marasmic diseases.
- Auditory disturbance due to streptomycin.

Supply:

Tablets	5 mg./tab.	100's & 1,000's
Injection	5 mg./1 cc.	10 & 50 amps.
	10 mg./2 cc.	10 & 50 amps.

TAKEDA CHEMICAL INDUSTRIES, LTD.

OSAKA JAPAN