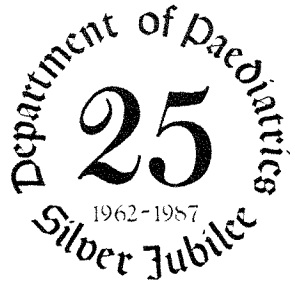
Silver Jubilee

25

1962-1987

Department of Paediatrics

University of Hong Kong



A Silver Jubilee always deserves some celebration especially when it coincides with another important occasion like the Centenary of the Medical School of the University of Hong Kong.

The Department of Paediatrics was established in 1962. It has grown from a two-member department to one with 64 members. There are a large number of people who have made major contributions to the development of the department, therefore it is impossible to acknowledge them individually in this article. However several past members deserve special mentioning. Professor C. Elaine Field who started the department, had also laid down a firm foundation for subsequent heads of the department to build on. Dr. Y.C. Tsao and Dr. Alice Chau were the key paediatricians who have made invaluable contributions during the formative years and they are still actively helping with our teaching programmes. Other important developments may be noted from the heads' reports on their respective periods.

The Department is responsible for: 1) teaching of paediatrics to under-graduates, post-graduates, and nursing staff; 2) serving the community as a resource and referral centre for various paediatric problems; 3) research in various fields of paediatrics; 4) acting as an advisory body in regard to paediatric care in Hong Kong. Since its inception, the Department has expanded into a 250 bed clinical unit. These beds are distributed in Queen Mary Hospital (the main teaching hospital) and several satellite centres, including the Paediatric Cardiac Unit in Grantham Hospital, the Child Assessment Centre in Duchess of Kent Children's Hospital, and the neonatal unit in Tsan Yuk Hospital.

We have attempted to include in this Silver Jubilee publication a list of all the staff and post-registration trainees who have worked in our department in the past 25 years. We have also tried to document all publications of the department up to June 1987. Unfortunately, relocation of offices and frequent changes of staff have rendered search for earlier department records very difficult. We hope that we may be forgiven for unintentional omission of important names and major projects.

We look forward to the department's continuing advance and progress in its firm commitment to the cause of child health and paediatric education in Hong Kong for years to come.

Chap-Yung Yeung,
Professor and Head,
Department of Paediatrics,
University of Hong Kong, 1987

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THE SILVER JUBILEE CELEBRATION

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Editors' note:

Material submitted by contributors outside the department has been printed as received.

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PROFESSOR C.E. FIELD

May 1962 – May 1971

In the Early Days –

The disease picture we saw in children in the outpatient department 25 years ago was gross, advanced and some of it preventable – advanced nutritional rickets for instance, tumours of all varieties and the late stages of blood or kidney disease. Before I left 9 years later the picture had completely changed.

I arrived in Hong Kong in May 1962 as the first Professor of Paediatrics within the Department of Medicine. The Chair of Pharmacology remained vacant so the University agreed to advertise it as a Chair of Paediatrics.

We started in a humble way. A single private ward in Queen Mary Hospital was our accommodation for Secretary, Technician with laboratory, Professor's office on the balcony and, with the bath removed, a records room and library! It taught me how you can manage on very limited accommodation and financially 'on a shoe-string'. What really mattered was the keenness of the few staff available and the interest of the medical students to be taught because therein lay the future of Paediatrics.

The Medical & Health Department drafted a few medical officers to Paediatrics and in this I was most fortunate to have really dedicated and loyal staff in Drs. Alice Chau, Tsao Yen Chow, Robert Johnson Lee, Luk Shing Chak, Paul Yue etc. With Miss Lenny Cheng joining us early on as secretary (and still the backbone of the department) we were set for development. With the co-operation and help of Dr. Hu Shih Cheong as Senior Paediatrician at the Kowloon Hospital I felt progress was inevitable – however we had our problems. We had no University lecturer, but with the generosity of the Li Shu Fan Medical Foundation we were able to establish the Li Shu Fan Lectureship in Paediatrics and Dr. Tsao Yen Chow was the first to be appointed to the post.

When starting anything new it is important to have guidelines. Our main objectives were care of the patients and teaching of the medical students. There were already two wards for children in Queen Mary Hospital and therefore adequate disease patterns for the students but what of the

broader aspect of child health which is the basis on which to understand disease? Much of this was lacking so it was necessary, with the co-operation of the various departments concerned, to develop a much wider field for student teaching in child health, infant welfare clinics, the neonatal service at Tsan Yuk Hospital and the handicapped children services. For the handicapped, care and facilities for the spastic child and mentally retarded child were lacking, so through voluntary organisations services for these children were developed. Shortly after I left, the Medical & Health Department established a much needed Child Assessment Clinic at Arran Street. Then finally, standards of growth and development of the normal child from birth to adolescence was a research project of the Paediatric Department largely organised and run by Dr. Flora Baber. We were widening our vistas but was the student interested? I regret to say not until we established a Paediatric Examination was the student really interested. It's just human nature after all!

The next important step was to convince the University authorities that we had reached maturity and should have a separate department of Paediatrics. Professors of Medicine rarely like to part with their Paediatric offspring but fortunately the Vice-Chancellor and Senate agreed we had come of age and the Paediatric Department was officially separated from the Department of Medicine on lst July 1966. A little later we were allocated a few rooms in the new block of offices for the Department of Medicine and shortly before I left plans were well advanced for a separate Paediatric floor.

A very important development for the raising of standards of treatment was the selection and training of medical staff. Most of them received overseas training returning with the M.R.C.P. and thus helped tremendously to broaden the outlook. At the same time we were able to draw in staff from the U.S.A. and Canada. Specialisation was just beginning with Haematology, Nephrology, Genetic Counselling, Neuro-psychiatry and Cardiology. Government medical staff numbers increased and a trainee lectureship was established. I do appreciate how much I owe to the wonderful staff I had throughout my 9 years. Student numbers also increased and may I give them due credit, I found them really interested and very stimulating to teach.

I have purposely omitted to mention the important liaison we had with the Hong Kong Paediatric Society and the private practitioners, because that is a separate story in itself. Many trained doctors from the department went into private practice and so helped to raise the standards in the town or left the department to staff the increasing number of Government children's wards in the new hospitals. Gross or advanced disease was now a rarity and services were rapidly developing for the handicapped and other specialties.

But what of the future? Many of our doctors have gone overseas already and seem to be doing good work. What about those who remain? I believe there is a wonderful opportunity for helping that vast continent of China to develop its Paediatric Services. I thrill to think that such a humble beginning in Queen Mary Hospital and the University is extending so far and just wish I had my life all over again to help you.

PROFESSOR G.M. KNEEBONE

May 1971 – June 1975

My tenancy in the Paediatric Department lasted for four years, a time which passed quickly, almost too quickly to be able to record any significant contribution that I could lay claim to as my own. It was, however, a period which gave me immense pleasure and enjoyment, and I have a real sense of pride in "being part of it all", albeit a brief association.

I inherited a Department which had been nurtured from its conception by my predecessor, Professor Elaine Field, and which in May 1971 was ready to take possession of the then newly constructed building. It seemed that this move symbolically represented another developmental stage in the Department's evolution.

The Department had already established an effective and efficient teaching and general clinical service role within the Queen Mary Hospital and the Medical School. Thus I saw my responsibility to expand the role that the Department should play within the under-graduate curriculum and in the broader community as a whole.

Paediatric Sub-specialisation

The development of sub-units of Paediatric sub-specialties would allow specialised clinical care and the opportunity for research and evaluation of the major child health problems in the colony. The contribution already made by Dr. Y.C. Tsao to the international study of the Nephrotic syndrome had initiated this process, and the subsequent appointment of Dr. Wai Kee-Ho, a cardiologist, and Drs. K.Y. Wong and Patrick Yuen, both haematologists, made this seem even more possible. The Department already had Dr. W.Y. Lui with training and expertise in neurology and child development, and thus the potential existed to create a degree of Paediatric specialisation which in turn would have encouraged an academic career structure within the Department. In the final outcome however the very great clinical demands for general Paediatric care and the overall teaching requirements combined to limit the opportunities for exclusive sub-specialty practice within the Department at that time. As a consequence the major function of the Department continued to be one of general Paediatrics with some areas of sub-specialty.

Community Child Health

Even in 1971 it was apparent that teaching students Paediatrics upon hospitalised children neglected the child health problems that existed in the community and which caused so much parental concern and so much medical attention in practice. The Paediatric curriculum was therefore extended to include home visits by the students after the child's discharge from hospital, and projects based on child health issues within the community. After consultation with the students, we planned to extend our "invasion" of the community by obtaining a double-decker bus which we planned would become our mobile base to travel around the districts giving child health advice to families. In retrospect the suspicion with which this was greeted by the Health Department was well founded, but at the time it seemed that our teaching would have been more appropriate and realistic had this development been possible.

The child health study begun by Elaine Field in Kowloon continued through the efforts of Flora Baber and emphasised the importance of the need to teach Child Health as well as Clinical Paediatrics within the Department.

The Children's Hospital

During this period there were several attempts to establish a definitive Children's Hospital, an initiative which would have provided a focus for the Department and for the further development of Paediatrics within the colony as a whole. Its ultimate failure to eventuate seemed at the time to be a significant blow to our aspirations for visible presence within community of Hong Kong. The subsequent pattern of including Paediatric units within general hospitals around the world has the benefit of allowing effective sharing of expensive equipment and resources, as well as the continuity of care from childhood through adolescence within one hospital. Thus our failure to achieve this goal might not have been the great disappointment it appeared at that time.

Other Hospitals

The Paediatric Department had responsibilities to a number of other hospitals during this period, and provided regular clinical service to the Grantham and Sandy Bay Hospitals, and in addition provided an over-view and consultant service to the Kwong Wah hospital in Kowloon. This then was a time when the Paediatric Department contributed quite widely to the general paediatric needs of the community. Following the visit of Dr. Kenneth Holt to Hong Kong at the request of the Health Department to evaluate the facilities for handicapped children, a regular clinic was set-up at Ruttonjee to survey the needs of handicapped children within the community. Dr. Lui Wai Ying continued to provide an assessment service to the John F. Kennedy Centre at Sandy Bay, to evaluate the outcome and progress of cerebrally palsied children under treatment at that Centre.

A considerable amount of the Department's activities at that time was therefore directed towards the provision of clinical service to the various hospitals and centres for children throughout Hong Kong.

Chinese University Medical School

Preliminary discussions began during this time to establish the second Medical School in Shatin with some misgivings that this would be feasible or possible. The subsequent successful achievement of this endeavour has effectively allowed the two Paediatric Departments to more reasonably share the responsibility of contributing to the total clinical care for children in Hong Kong.

"Significant" Events

Two "epidemics" of infective disease occurred during this period. Firstly, a carrier of Salmonella within the delivery suite at Queen Mary Hospital generated an overwhelming outbreak of post-natal septicaemia amongst the new-borns which led to the conversion of one paediatric ward to the total care of these infants. This epidemic was further complicated by the haemolytic jaundice induced by the surreptitious administration of "chuen lin" to the infants by the solicitous grandparents. The second involved the E. Coli septicaemia which resulted from the contamination of the feeding room preparation area at Tsan Yuk Hospital by the bed-pan cleaning amah.

The Paediatric Department invited Professor Wong Hock Boon as external examiner and during this visit discussions took place that led to the re-development of the South East Asian Association of Paediatric Societies which has grown in stature and significance ever since, and thus the Department played a part in initiating the re- establishment of this Organization.

Thus my association with the Paediatric Department during the four years between 1971 and 1975 was for me both fascinating and educational. The Department began the process of further development into the areas of sub-specialty and beyond the hospital and into the community but at that time these extensions were limited by the need to contribute to the immediate situation where Paediatric services were required at the various hospitals and centres throughout the colony. It was however a stage in the evolution of the Department where one could sense the excitement in preparing for the next phase of the Department's growth and development.

I am fortunate and proud of my association with the Department and the staff with whom I shared my involvement in its development.

DR. W.Y. LUI

June 1975 - August 1977

It was hardly enchanting when one was suddenly confronted with heading a "headless" department, on the return from one's year-long sabbatical abroad, with neither interest nor ambition for that position.

It was 1975.

There had been no professor for a few months. Clearly there was a need for direction and leadership. The medical staff was exhausted with excessive clinical service and teaching load. The laboratory staff was fully occupied with service routine due to the lack of microassays for paediatric patients in the hospital Biochemistry laboratory. The secretary had to shoulder all administrative, clerical and house-keeping responsibilities. In the paediatric wards, beds were closely packed. The equipment was inadequate and ineffective. The nurse-patient ratio was appallingly meager. A constant rapid turnover of nursing staff guaranteed against maintenance of high standard paediatric care.

At the University front, there were the medical undergraduate curriculum review and quadrennial planning. In a city like Hong Kong, where 20% of the population was under the age of 16 years, and where local access to continued education for medical graduates was not normally provided, it was striking how little emphasis had been put on paediatrics in the undergraduate medical curriculum. Yet, the need for quality comprehensive paediatric teaching in small groups in the phase of heavy patient service load and wide commitment of the department in community services put undue strain on the available number of University medical staff to the detriment of their own research pursuit.

It was not difficult to recognize tasks ahead. Problem solving was a different matter. Substandard paediatric care could hardly be a sound basis for teaching, training and research. Morale-lifting of departmental staff was essential. Effort was made to engender a sense of team spirit for a common cause. Free and clear communication among medical, nursing, and all supportive staff was emphasized. Trimming of clinical service load was attempted, with sacrifice of some community involvement of the department. In the process of fighting for more Governmental medical and nursing staff, new equipment, advocacy for paediatrics, and quadrennial planning for more teaching, technical and secretarial staff, the handicaps of a small "professor-less" department soon became obvious. It was fortunate that the Vice-Chancellor, Dr. Rayson Huang (to whom I am ever grateful) offered the department support and encouragement, among the political intricacy of overpowering large teaching departments and an unsympathetic Faculty.

It was only the hard work and dedication of all medical, nursing, technical, secretarial and supportive staff in those two plus years that gradually brought a different atmosphere to the department. There gradually emerged a family-like tie among all concerned. There were now laughs and tears instead of apathy. The harmonious work relationship between paediatric medical and nursing staff was noteworthy.

Clinical service load was as heavy but more up-to-date equipment for patient care became available. We had, however, a terrible loss of some medical and more experienced nursing staff to the new Princess Margaret Hospital. Two more lecturers were to be added to the department for the next quadrennium. A plan was initiated by the department in co-operation with Dr. H.J. Lin and Professor Gibson to develop microbiochemical techniques in the hospital biochemistry laboratory so as to partly free the paediatric departmental laboratory for research purposes. A training sabbatical in advanced medical technology in genetics was arranged for the senior technician. A clerical assistant was added to the department.

In teaching, a more structured paediatric program was organized, with emphasis on basic principles of child life, development, and health in the perspectives of a child's family and social environment. In co-operation with Professor Colbourne of Department of Community Medicine, and the paediatric social worker of Queen Mary Hospital, the medical-social case work format with home visits and conferences was established as the first teaching model in the Faculty. The close liaison between social workers and the department had been a notable tradition of the department throughout the years.

Little financial and human resources could be spared for research, however.

On the eve of Professor J.H. Hutchison's arrival to take up the Chair in Paediatrics in September 1977, the morale of the department was at its height. The question came up in my mind : "What have I done in the past two years?"

Instead of answers, I saw all the faces of friends and colleagues in medical, nursing, technical, secretarial and supportive fields. I still see them today at this moment.

PROFESSOR J.H. HUTCHISON

September 1977 - August 1980

Following a long career in the United Kingdom it was a great privilege to be offered the Chair of Paediatrics in Hong Kong on a 3-year contract. I look back on these years with much pleasure and some satisfaction. When I arrived in the department on lst September, 1977 it had been without a Professorial Head for almost three years. While the teaching and service functions had been maintained, largely due to the most praiseworthy efforts of two married ladies (Dr. W.Y. Lui, Acting Head of Department, and Dr. Anita Li), morale was not high and good quality young graduates were not being attracted to the department. My first challenge, therefore, was to encourage young doctors to join the department, some as lecturers and some as government medical officers, and to infuse into them enthusiasm for paediatrics. During the ensuing three years I believe that this was achieved. Period of study leave were arranged for each of them in sequence with friends and colleagues in the United Kingdom. A very high success rate in the MRCP(UK) allowed me to build up a team of young men and women who having completed their general professional training were ready for vocational training. This proceeded at a faster rate than is usual in the United Kingdom, and some members of the department were steered towards one of the paediatric subspecialties (neonatology, cardiology, oncology). We already had expertise in neurology (Dr. W.Y. Lui) and haematology (Dr. Anita Li).

A University department must regard research as a high priority and several projects were started. I was able to recruit Dr. Flora M. Baber (an expatriate paediatrician who had worked with Professor C. Elaine Field) to a vacant lectureship. In association with Dr. C.W. Chan, a government senior medical officer, Dr. Baber initiated a major project in the field of growth and development. Research in neonatology was developed at Tsan Yuk Hospital by Drs. Sam P. Lau and K.P. Fung, later joined by Dr. T.F. Fok. Dr. Patrick Yuen, trained in paediatrics in Canada, joined the department to its great benefit, and began his researches in oncology. From all these projects publications have appeared in scientific medical journals before and since my departure from Hong Kong. A further study, also published, involved a pilot screening programme to detect congenital hypothyroidism in babies born in Tsan Yuk and Queen Mary Hospitals. I understand that such a screening service has now been introduced to all hospitals in Hong Kong.

Modern paediatric practice demands the availability of chromosome analysis using banding techniques which permit precise chromosome identification. To this end I arranged that Mr. F.T. Lee, our graduate laboratory technician, spent six months in the Department of Medical Genetics in the University of Glasgow. He returned to us well trained in the latest techniques which added very considerably to our diagnostic and counselling facilities. On my advice the Vice-Chancellor also agreed to invite Professor M.A. Ferguson-Smith from the Department of Medical Genetics in Glasgow to visit and advise the University of Hong Kong on the feasibility of setting up a comprehensive clinical and laboratory genetic service. However, the financial and other implications

required government approval, and following Professor Ferguson-Smith's report the Medical and Health Department arranged for Professor Paul Polani from the Paediatric Research Unit in Guy's Hospital to visit and submit a further report. I have been informed that a consultant genetic service has now been established in Hong Kong but the opportunity to incorporate a strong University and research component seems to have been missed.

In any account of my time in the Chair of Paediatrics some mention should be made of my criticisms of the facilities for paediatric patient-care in Queen Mary Hospital. These were in contrast to the generous attitude towards the Department of Paediatrics by the University. In the overcrowded paediatric wards there was a marked lack of isolation facilities so that, for example, children on immunosuppressive drugs had to be nursed exposed to all manner of infections. There was no accommodation where mothers could be admitted with their young children, and there were no play areas for the children. My campaign for the provision of modern facilities in Queen Mary Hospital, or preferably in a separate children's hospital, attracted the attention of the public media, but I was given no reasons to think that it would achieve any of its objectives before I left Hong Kong.

Undergraduate teaching has received late attention in this short report, not because it is unimportant, but because it was the easiest and one of the most pleasant of the department's functions. Clinical teaching at the bedside was carried out not only by the staff of the University department but also by a few senior paediatricians who were in private practice in Hong Kong. This arrangement, which antedated my time in Hong Kong, allowed our students to see some of the problems of paediatric practice from a somewhat different perspective which I believed was valuable. However, teaching on growth and development, systematic paediatrics and neonatology was conducted entirely by the University and departmental staff. I found Chinese students to be hard working and eager to learn. It was a privilege to teach such fine young men and women.

Since leaving Hong Kong I have watched with great interest the progress of the men and women recruited during my time. Some continue to serve with my successor. Others have transferred to the department of paediatrics in the new medical school of the Chinese University of Hong Kong. Some have departed to private practice. I wish them every success and fulfilment in their future careers.

PROFESSOR C.Y. YEUNG

September 1980 – Present

This period of the department has been characterised by many changes and developments associated with the fluctuations of political mood and economic climate. The economic boom, before the British Prime Minister's historic visit to China in 1982, had brought about a tremendous public outcry to improve the various health care facilities. This resulted in more ready acceptance of our proposals to improve health care facilities for children.

The department was involved in planning 3 new building projects. A new child health centre at Queen Mary Hospital was proposed in late 1980. This plan was subsequently implemented and the building will be completed in 1988. A proposal for a new Child Assessment Centre was supported by the Society for the Relief of Disabled Children and a new building has just been completed at the Duchess of Kent Children's Hospital. We have also been associated with the recently established Cardiac Centre at Grantham Hospital and have assumed full responsibility for its paediatric division since early 1982.

Sub-specialty development has been a primary objective of the department. A neonatal intensive care program has been organised at Queen Mary Hospital and Tsan Yuk Hospital. Intensive care equipment and neonatal laboratory facilities were generously provided by the Government. A new, fully equipped neonatal laboratory was established in early 1982. General paediatric intensive care was introduced at Queen Mary Hospital in 1982. Cardiology has become an established discipline within the department at the Grantham Hospital. These clinical services have been increasingly utilized by colleagues in paediatrics and obstetrics and by other medical practitioners. These programs are also fully integrated into both the undergraduate and post-graduate educational programs of the department.

A new developmental paediatrics program has just started at the Duchess of Kent Children's Hospital; many para-medical professionals have already been recruited for this service. The other sub-specialties represented in the department, beside general paediatrics, include haematology/oncology, endocrinology, nephrology, respirology and infectious diseases. As at June this year, our medical staff consists of 14 University teachers, 14 Government SMOs/MOs, 8 subvented SMOs/MOs, 6 Interns and 2 Externs. There are also 12 technicians, 4 of whom are government staff seconded to our neonatal laboratory at Tsan Yuk Hospital. There are also 11 para-medical professionals in various clinical programs. We have, however, only 3 secretaries (including one at Grantham Hospital), supported by a small clerical and office staff.

Undergraduate teaching has been emphasized by the department since its inception. During the current period, a few additions and modifications were introduced. One or two students are now attached to a general paediatric team as 'residents' for a 2 week period to obtain resident paediatric training. This has proven to be an effective and well received educational endeavour. Teaching on common child health problems was added with sessions held at the Maternal and Child Centre and Kwun Tong Community Health Project. This provided a more balanced

education to medical students by de-emphasizing hospital-based paediatric teaching. Small groups of students are also sent, twice every 10 weeks, to the two major government hospitals for teaching by the Government paediatric consultants. This enriches the clinical exposure of the students. These additional teaching programs are made possible only with the tremendous effort and assistance from the respective heads of various institutions and our honorary lecturers. To all of them, I wish to express our deepest gratitude on behalf of my department.

The department is active in post-graduate education although there is no special funding or staff provided for the purpose. A residency rotation programme is designed for each clinical trainee for 3½-4 years. Most trainees obtain the MRCP (UK) before completion of the rotation. In the past 6½ years, 19 trainees have acquired the MRCP. Many of them have proceeded to additional training in a subspecialty for a further 2 years, including one year's training in an overseas centre. We also have 3 M.Phil. students, 2 of whom will be graduating in the next few months. We have had 9 clinical fellows, 5 of whom were supported by the China Medical Board. These fellows have come from neighbouring countries, viz Indonesia, Thailand, Philippines, China, or Pakistan to study neonatology, intensive care or cardiology. We continue to receive more applicants than our funding can afford.

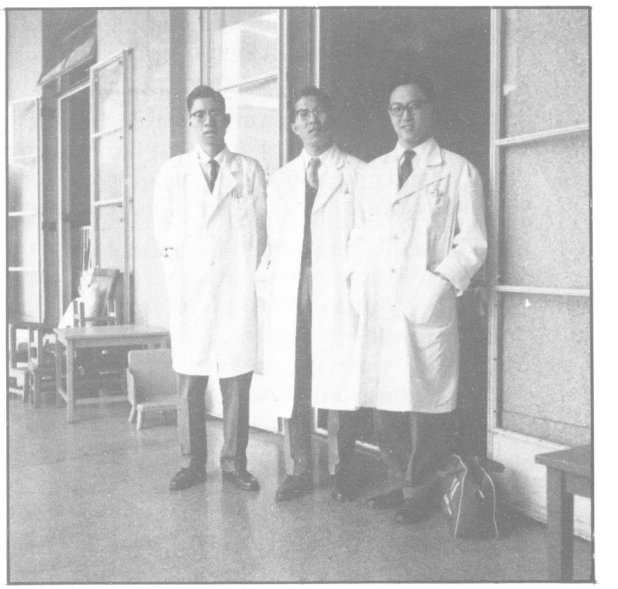
In 1982 we have started regular visiting professorships, inviting distinguished scientists and clinicians to teach in the department and to give public lectures. These were made possible only by the generous donations from various firms and charitable individuals, to whom we are extremely grateful. I wish to specially mention and thank Mr. Joseph Hotung for his most generous donation of 2.5 million dollars. This donation is to set up a Paediatric Research and Education Fund for our department and makes this Silver Jubilee Year a particularly joyous occasion.

I am most proud of all my staff, a highly dedicated and happy group of people. Our paediatric staff are not only providing a high level of medical care to our patients, they are also good teachers and research workers. Their research productivity is well evidenced in the publication list appended in this book . Of course, laboratory support is an indispensible part of good research projects. We are extremely lucky to have a laboratory which is well organised, efficient and of a high standard. The technical staff, in particular the senior laboratory superintendent, Mr. F.T. Lee are highly capable. Our department has historically provided the clinical laboratory services for our paediatric patients. I am grateful to the hospital laboratories for adapting to our needs and for taking over, since 1982, most of the micro-laboratory tests, so freeing some of our technician-time for research. Needless to say, the department could not go on without the extremely hard working and dedicated secretarial and clerical staff, to whom I am specially indebted.

The department has gone through some ups-and-downs during this period. We may experience further jitters during the next 10 years of 'count-down toward change of sovereignty' in Hong Kong. I am confident that the contributions and devotion by all the staff to the cause of child health and paediatric education will have lasting value for years to come, and that this will be unaffected by any change of government.

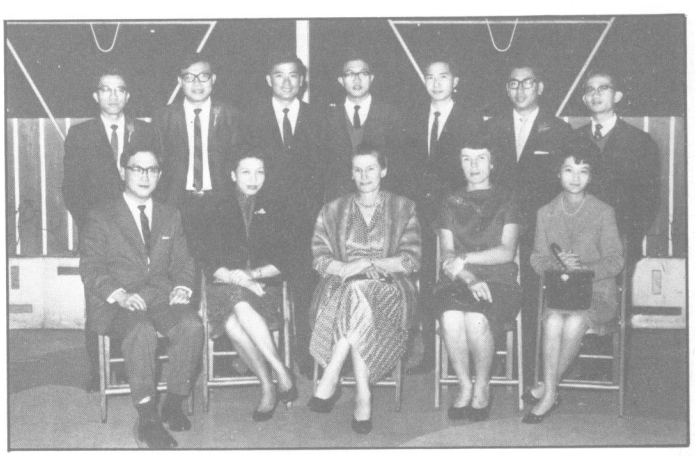
THE CONCEPTUAL YEARS

Paediatric Unit, Department of Medicine 1957
Back row : Dr. C.C.WONG, Sister MITCHEL, Sister CHOW, Dr. Frank HSU, Sister Phyllis WONG, Dr. W.Y. ORR, Dr. Doris CHAN, Dr. Alice LEE Dr. P.C. CHEUNG
Front row : Sister TSANG, Guest, Guest



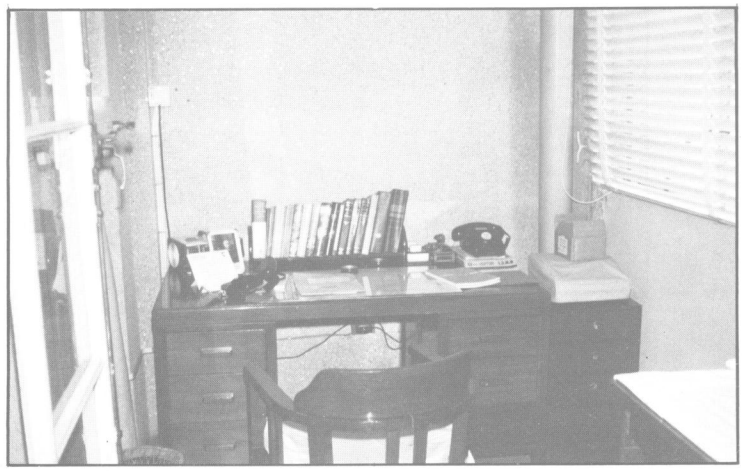
MacKenzie Ward

Staff of the new department:
Back row : S.K. WONG, Johnson LEE, Paul YUE, Michael LO, K.K. WONG, S.C. LUK, Y.T. TSANG, K. CHOW
Mid row : Alice CHAU, Elaine FIELD, S.C. HU*, Mrs. S.C. HU
Front row : W.Y. ORR, T.Y. CHAN
(*the first Government Paediatric Consultant)

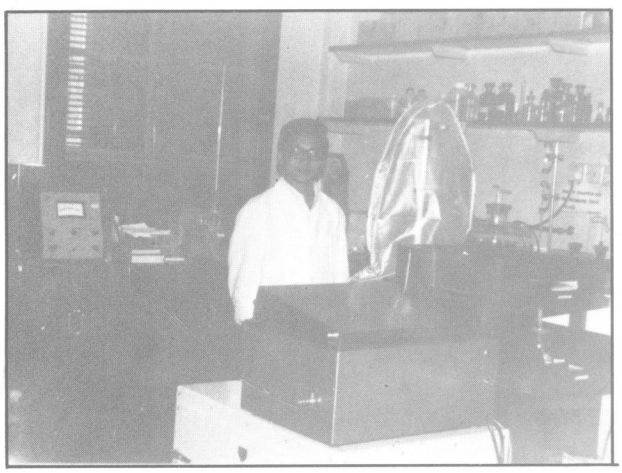


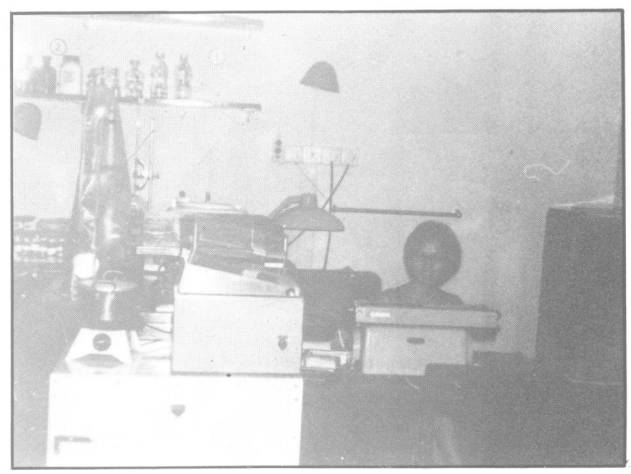
Y.C. TSAO and Alice CHAU (seated on the left):
the first two full-time faculty members

THE EARLY ACCOMMODATION

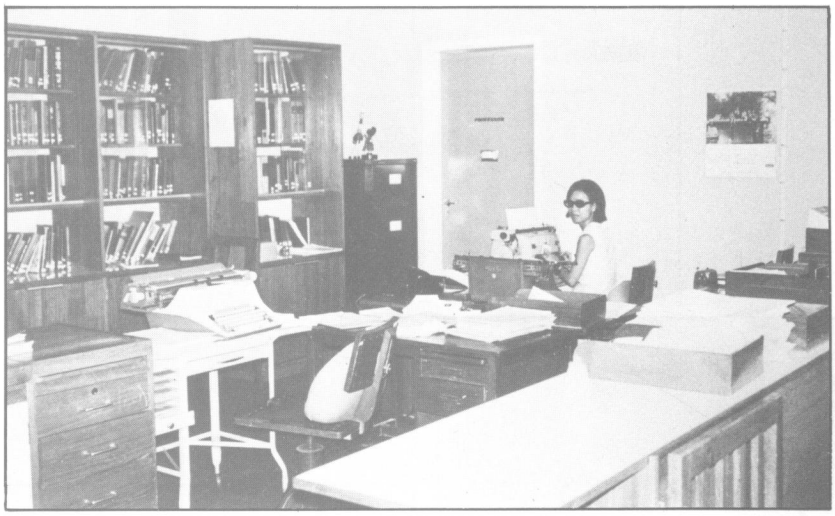


The Professor's office in the balcony

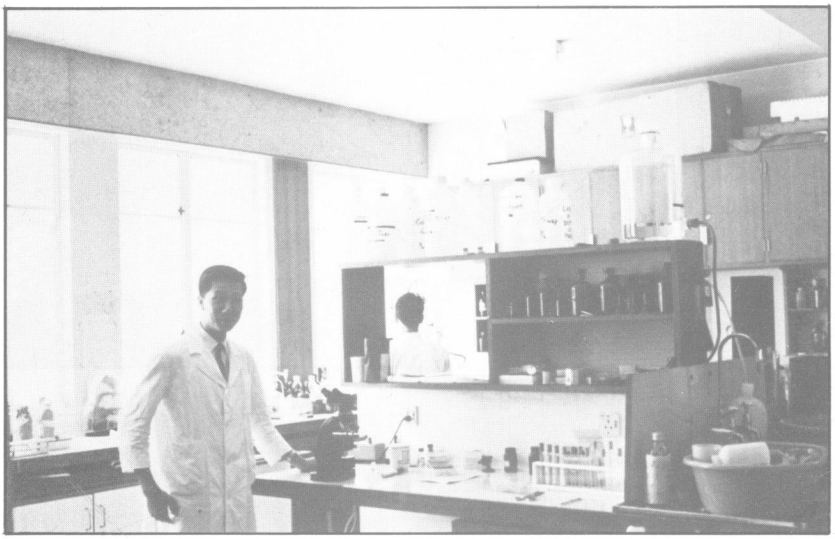




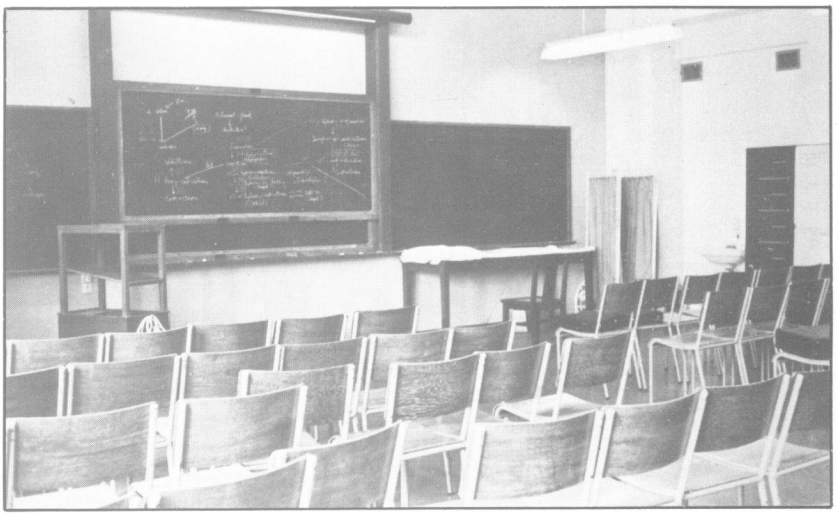
The laboratory and the office in one room



The new office 1966

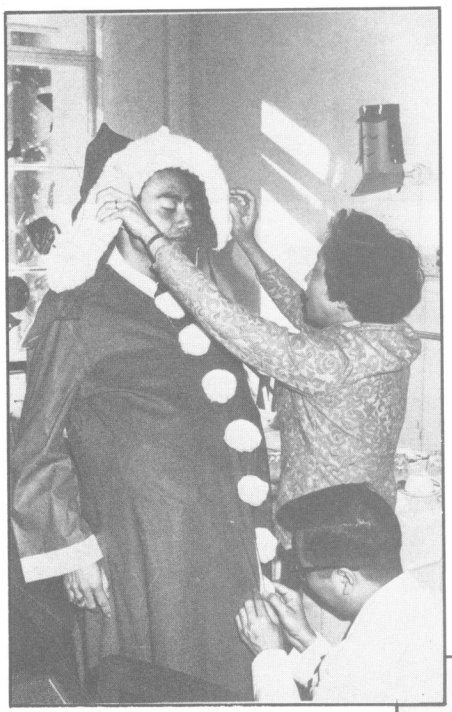


Laboratory 1966

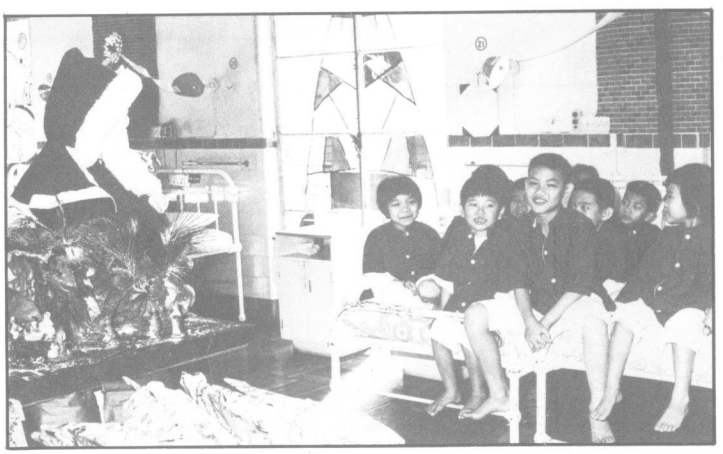


Lecture theatre shared with the Department of Medicine

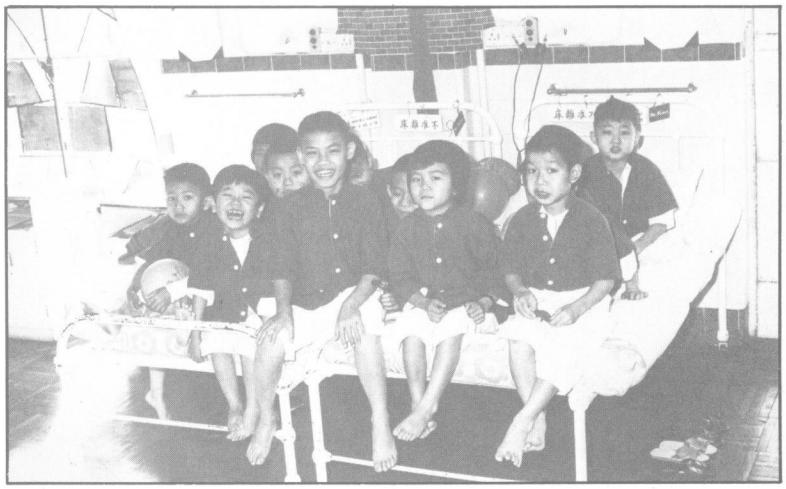
CHRISTMAS IN THE CHILDREN'S WARD



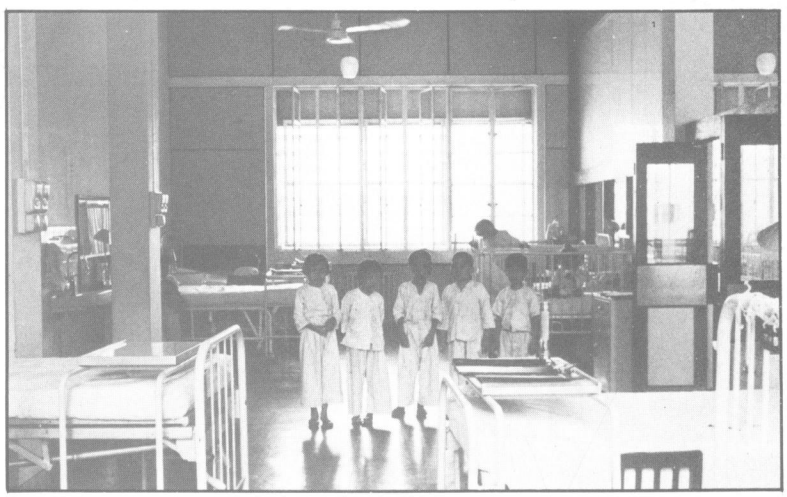
Our first allergist challenged by cotton wool



Children's Wing

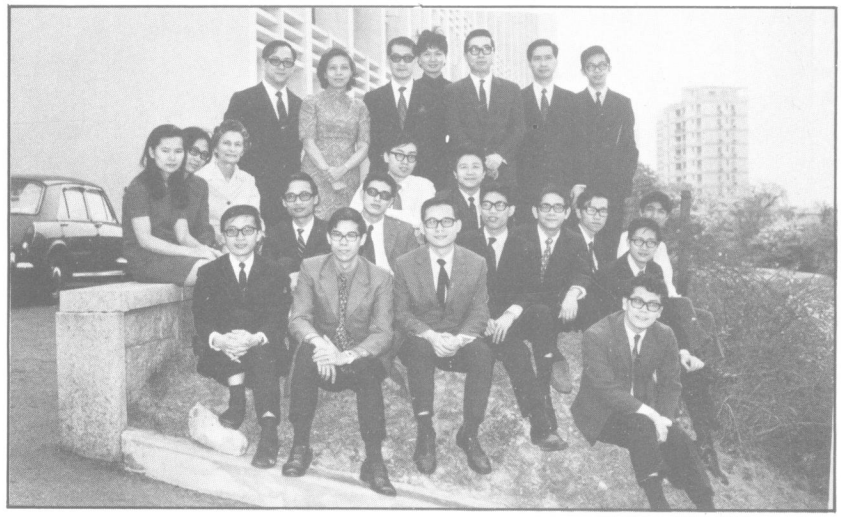


Head Ward 1968



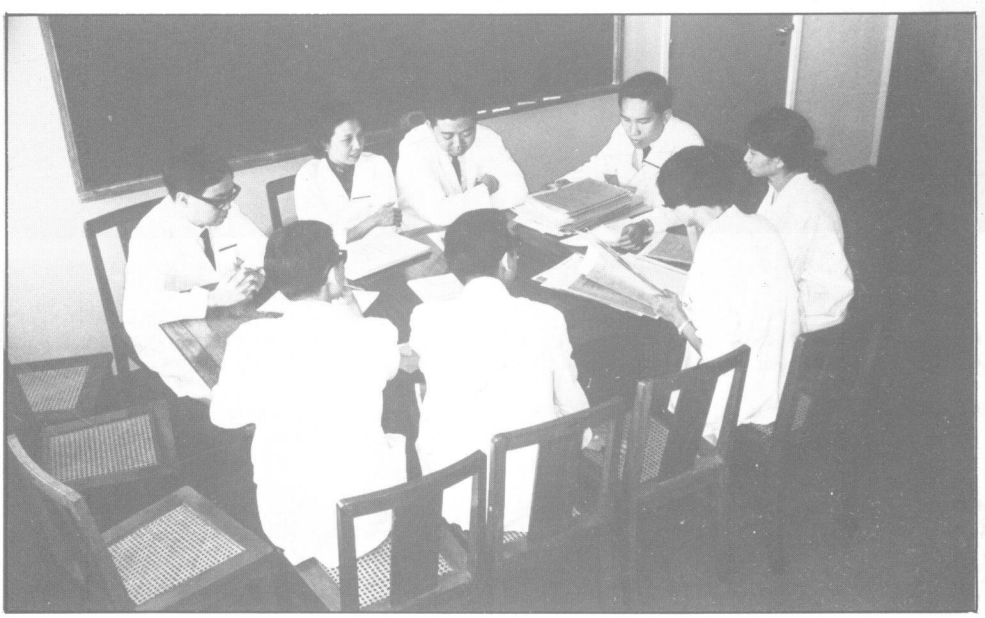
B6 Ward 1969

FAREWELL TO THE FOUNDATION PROFESSOR



STAFF MEMBERS AT WORK

Busy over Paediatric Society Dinner?



Journal Club

STAFF AND FRIENDS 1971

Back Row : Dr. C.K. Man, Dr. C.H. Sum, Dr. S. Lau, Dr. E. Lau, Dr. K.H. Lam, Dr. V. Yu, Dr. C.M. Chung, Dr. C.Y. Yeung, Dr. E. Chang, Dr. K.C. Poon, Dr. C.S. Cheung, Dr. Y.N. Chau, Dr. S.C. Hu, Dr. R.J. Lee, Dr. P. Yue, Dr. P. Lo, Mr. T.S. Chan.

4th Row : Mr. E.G. Baber, Dr. E. Chan, Dr. A. Li, Dr. C.W. Lam, Dr. Y.C. Tsao, Dr. K.C. Diu, Dr. W.Y. Chu, Dr. W.K. Sin, Dr. K.K. Tsang, Dr. S. Lim, Miss S. Liu, Mrs. J. Lee, Mrs. V. Yu, Mrs. C.M. Chung, Dr. T. Lee, Dr. K.K. Wong, Dr. P. Lam, Dr. Y.T. Fung, Dr. R. Fung, Dr. A. Cheung, Dr. K. Chau.

3rd Row : Mrs. M. Tsao, Dr. W.Y. Lui, Mrs. P. Yue, Miss L. Cheng, Mrs. A. Cheung, Dr. A. Chau, Dr. F.M. Baber, Mrs. S.C. Hu, Prof. C.E. Field Mrs. R.J. Lee, Mrs. R. Fung, Mrs. C.Y. Yeung, Mrs. K.C. Poon, Mrs. E. Chang, Mrs. K.K. Wong, Miss R. Li, Mrs. Y.T. Fung.

2nd Row : Mr. K.Y. Wong, Dr. P.F. Ip, Dr. Y.B. Au-Yeung, Mr. C.Y. Wong, Mr. F.T. Lee, Dr. N.K. Leung, Dr. L. Wong, Dr. T.W. Chiu, Mr. K.C. Nip, Dr. C.M. Yeung.

1st Row : Dr. J. Lee, Dr. R. Li, Dr. C.H. Yeung, Dr. C. Hsu, Dr. Y.H. Yung, Mr. M.C. Chan.

THE DEPARTMENT 1972

Back row : Drs. K.H. KWAN, K.Y. CHEUNG, S. SHUM, C.W. CHAN, C.K. MAN, M.J. WATT, S.T. HWANG

Mid row : Drs. K.C. DIU, P.W. CHAN, L.K. WU, L. WONG, C.M. CHEUNG, Y.B. AU YEUNG, P.F. IP

Front row : Drs. J. LEE, W.K. SIN, W.Y. LUI, A. CHAU, Prof. G.M. KNEEBONE (Head), K.Y. WONG, A. LI, D. SUE, V. YU

THE DEPARTMENT 1975

Back row : Dr. L.S. WU, Dr. S.Y. LAM, Dr. A. TAM, Dr. R. CHING, Dr. C.W. CHAN, Dr. C.W. TSE, Dr. P. KO, Dr. T.C. TONG, Dr. K.W. NG, Dr. S.M. SIU, Dr. C.K. LEE

Front Row : Dr. K.H. WAI, Dr. H. IP, Dr. W.Y. LUI (Head), Professor J.H. HUTCHISON, Dr. A. LI, Dr. A BUCHANAN, Dr. P. YUEN, Dr. W.K. SIN


PROFESSOR C. ELAINE FIELD
H.K. PAEDIATRIC SOCIETY
REUNION DINNER
25-10-78


DEPARTMENTAL DINNER
IN HONOUR OF
PROF & MRS. J.H. HUTCHISON

OLD AND NEW STAFF 1980

Back row : Mr. F.T. LEE, Mr. M.C. CHAN, Mr. H.N. WONG, Mr. C.M. CHAE, Mr. C.M. WONG, Mr. C.L. CHEUNG, Sister S. TONG, Miss K.H. CHAN, Dr. S.P. CHOW, Miss S.N. LAU, Miss R. CHAN, Dr. S. LAU, Dr. C.Y. WU, Sister M. CHEUNG, Miss E. LIU, Dr. C.W. CHAN, Sister E. CHAN, Sister R. LAN, Mr. M.W. NIP, Dr. K.P. FUNG, Mr. CHAN, Mr. B.M. NG, Dr. S.C. CHAN, Dr. E. CHAN, Dr. Y.P. YIP Mr. F. LIU, Mr. L.H. KWOK

Mid row : Sister J. TSANG, Mrs. R. KO, Mrs. J. LI, Miss L.H. WU, Dr. O. CHOW, Mrs. S. LAU, Miss L. CHENG, Dr. W.Y. LUI, Prof. J.H. HUTCHISON, Mrs. J.H. HUTCHISON, Dr. A. LI, Mrs. A. CHAN, Mrs. A. TAM, Mrs. C.W. CHAN, Dr. A. TAM, Mrs. T.F. FOK, Mrs. P. KO, Sister G. LAU

Front row : Mr. C.W. HUI, Dr. S.Y. CHU, Dr. P. KO, Dr. T.C. TONG, Dr. K.H. KO, Dr. P. LEUNG, Dr. P. YUEN, Dr. A. CHAN, Dr. A. TAM, Dr. R. LO, Mr. A. WONG, Dr. T.F. FOK, Dr. E. YU

COMRADESHIP

Baby shower at
Tsan Yuk Hospital?

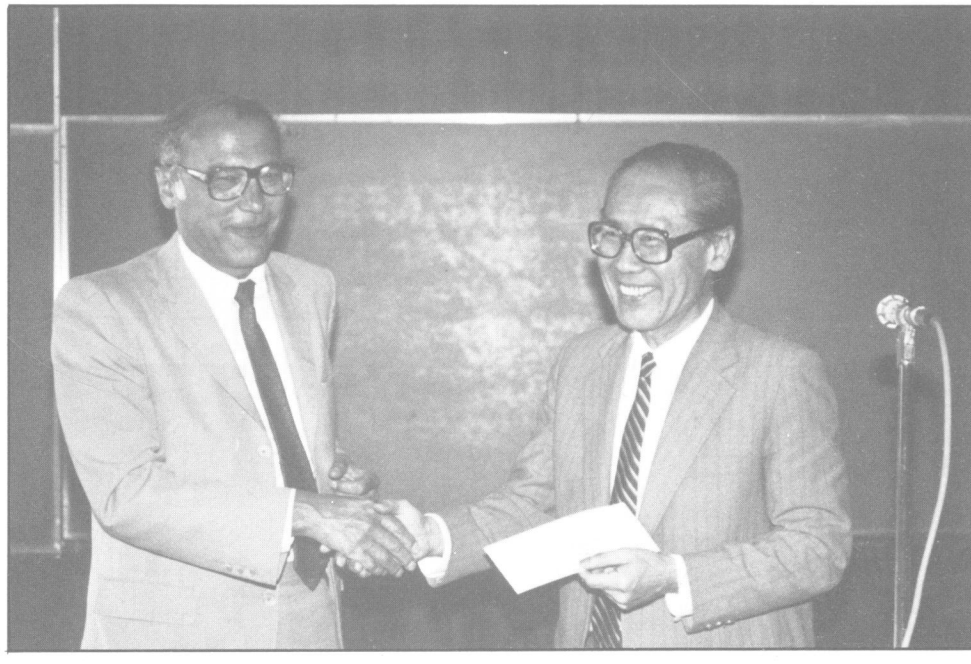
Floating membership

A colleague
(Dr. SY Chu at
far right) who
is sadly missed.

Seminars at Sun Yet-sen University of Medical Sciences, Guangzhou

Lectures at Beijing

MRCP examination comes to Hong Kong



2.5 million dollar donation from Mr. Joseph Hotung :
the Vice-chancellor, Dr. WANG Gungwu received the cheque for the department

THE CHILDREN'S NURSES

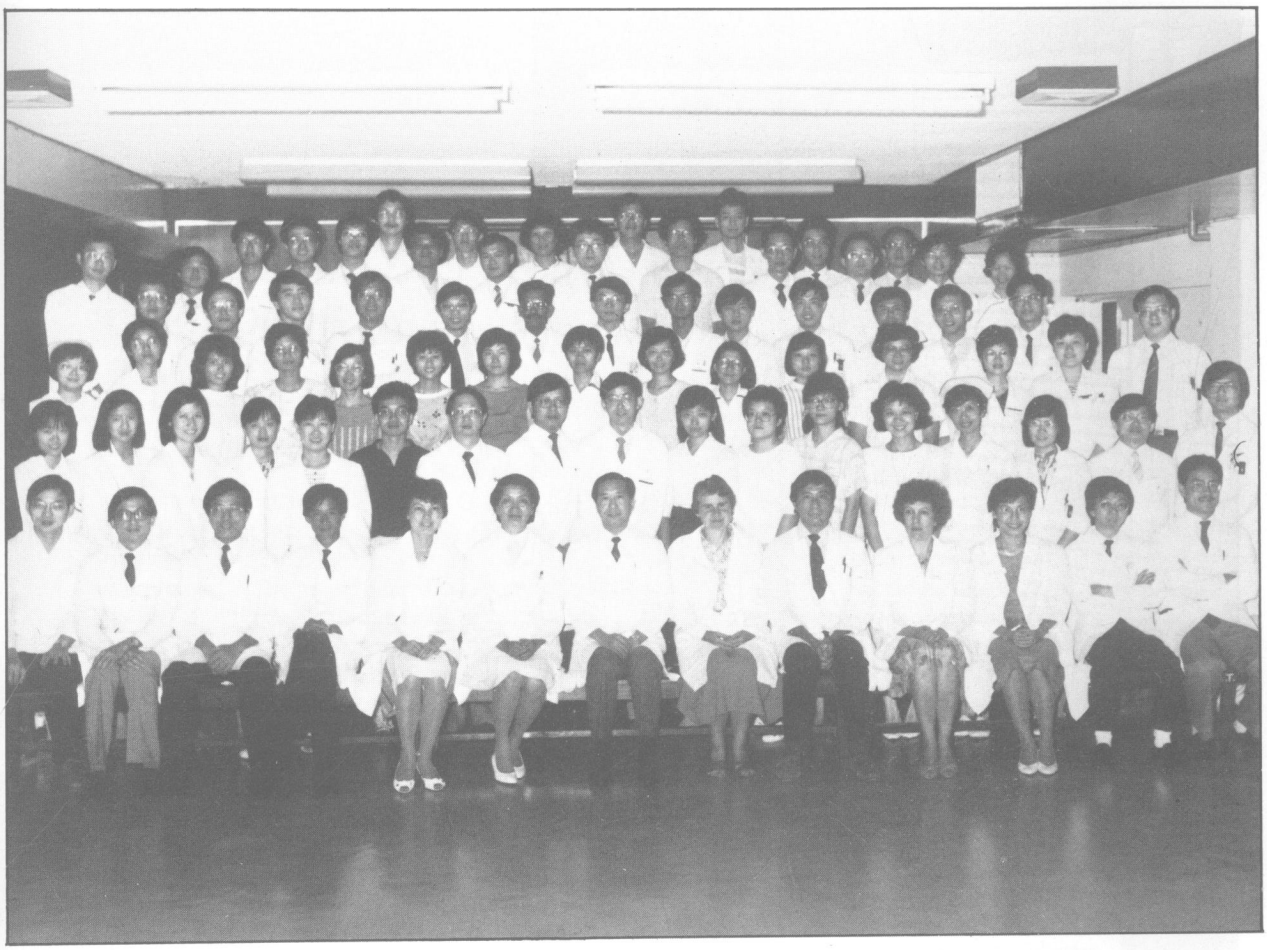
BEHIND THE SCENE

The Laboratory

Mr. FT Lee and colleague at work.

Ms Lenny Cheng and the office staff.

THE DEPARTMENT 1987



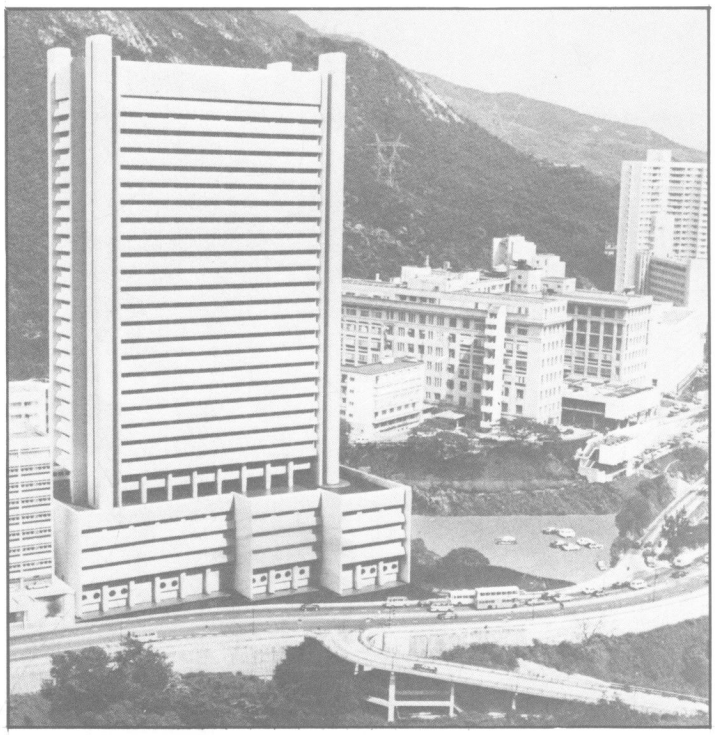
Back row : Mr. K.Y. HA, Mr. H.S. WONG, Mr. H.W. WONG, Mr. K.F. CHEUNG, Mr. P.S. SIU, Mr. C.M. WONG, Mr. Y.K. LUK, Mr. S.K. WAN, Miss K.H. CHAN, Dr. Y.W. SHIU, Mr. C.Y. LEE, Mr. B.M. NG, Dr. K.W. HUI, Dr. K.F. CHAU, Mr. A. YUEN, Dr. H. HUI, Mr. R. NG, Dr. W. CHIU, Miss S.N. LAU

4th row : Dr. C.H. LI, Dr. T.L. NG, Dr. T.S. TANG, Dr. K.H. TAM, Dr. P.S. TANG, Dr. T.L. KWOK, Dr. A. LAEEQ, Dr. K.C. CHAN, Mr. K.K. CHONG, Dr. S.K. KWAN, Dr. Y.T. WONG, Dr. D. MA, Dr. K.H. CHOW, Dr. C.W. LEE

3rd row : Miss S.L. NG, Dr. E. KWAN, Miss A. LO, Mrs. R. KO, Miss L. CHENG, Mrs. J. LI, Miss F. CHUNG, Miss V. CHAN, Miss K. CHAN, Miss B. CHAN, Miss C. CHEUNG, Sister S. NG, Miss L. CHOW, Miss H.M. HUI, Dr. R. SU

2nd row : Miss B. HUI, Miss C.E. CHUNG, Dr. S. FAN, Dr. L. CHOI, Dr. B. LAM, Mr. K.Y. CHUI, Mr. F.T. LEE, Mr. C.S. LEUNG, Mr. H.N. WONG, Miss M. CHEUNG, Miss R. CHUI, Miss M. CHEUNG, Mrs. C. YIM, Miss K.P. PUN, Dr. W.K. AU, Dr. P.T. CHEUNG, Dr. K.W. TSE

Front row : Dr. N.S. TSOI, Dr. M.Y. CHENG, Dr. E. YU, Dr. L. LOW, Dr. O. CHOW, Dr. A. LI, Prof. C.Y. YEUNG, Dr. R.E. DAY, Dr. K.C. LAU, Dr. G.M. SAMUDA, Dr. V. WONG, Dr. R. LO, Dr. P. LEUNG
Dr. Y. C. TAM (absent)

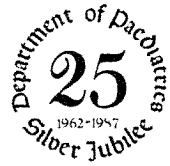


Future children's centre at Queen Mary Hospital

Child Assessment Centre at the
Duchess of Kent Children's Hospital

Neonatal Unit at Tsan Yuk Hospital

Cardiology Unit at the Grantham Hospital



CARDIOLOGY

Like paediatrics, paediatric cardiology began in the sixties as a branch of adult medicine. Children with heart diseases were referred to our physician colleagues for assessment and treatment. The increasing awareness of the very different needs of young infants and children coupled with the rapid world-wide growth in knowledge and practice of paediatric cardiology had revealed the inadequacy of this arrangement. The Department of Paediatrics began to take over the entire clinical activity of paediatric cardiology in early seventies and this marked the first developmental milestone of this sub-specialty. However the resources available at that time for programme development were unfortunately, extremely limited; for only one session a week could the paediatricians gain access to a catheterization laboratory in which was housed only prototype single-plane radiological equipment interphased with a video-tape recording system. Detailed anatomic delineation for more complex conditions was impossible, severely hampering the quality of investigations. Despite such adversity the Department strived to achieve an average of 100 to 150 cardiac investigations a year. Although we had difficulty coping with the clinical load, this experience laid the groundwork for subsequent development.

Following the decline in the prevalence of tuberculosis in Hong Kong, the bed-occupancy at the Grantham Hospital had dropped to an economically unacceptable rate. The Scadding and Fox recommendation in 1976 of converting the Grantham Hospital to a tertiary cardiothoracic institute for Hong Kong had added hope for a new dimension of cardiology service for the community. The timely and generous donation of Mr. Kwok Tak Sing made it possible to furnish the hospital with the most up-to-date investigatory (both invasive and non-invasive) and monitoring equipment, and cardio-pulmonary bypass facilities. The paediatric cardiac unit became operational in 1979, earlier than its adult counterpart, as an interim programme of the entire project. It soon became obvious that the staffing, laboratory, and other subspecialty support requirement of a tertiary paediatric cardiac center would be so immense as to necessitate the support and supervision of an experienced and sophisticated academic paediatric unit. The Department of Paediatrics was invited to join forces and to head the unit at the Grantham Hospital. The "marriage" took place in early 1982 and since then the development of a paediatric cardiology programme for the entire community of Hong Kong has rapidly developed.

The programme expanded rapidly to provide services not only to the entire community of Hong Kong but also to other surrounding countries. The number of admissions, investigations and cardiac surgical operations grew every year to a level which could match any world-class paediatric cardiac center. The growth was not in size but also in sophistication and quality. This was reflected in the rapid increase in admissions of the younger infants and newborns who

required much more sophisticated care than the older child or adult (40% of admissions in 1986 were below 12 months of age). The regular clinical activities of the unit included (i) 24 hour emergency catheterization service on top of daily elective cardiac catheterizations, (ii) daily non-invasive investigations including 2-dimensional and Doppler echocardiography, tread-mill exercise testing, 24 hours ECG monitoring, etc., (iii) primary care in a 10-bed post-operative cardiac intensive care unit and a 40 bed general cardiac ward, (iv) twice weekly out-patient service for cardiological referrals as well as pre-operative and post-operative ambulatory care.

Professor R.M. Freedom's visit in mid 1986 added a new dimension to the programme. Interventional cardiac catheterization was introduced. The paediatricians could now extend their activities in the catheterization laboratory from investigatory only to therapeutic as well. Balloon valvuloplasty now became the standard therapeutic option for stenotic arterial valves.

With 3 University staff cardiologists stationed full-time at the Grantham Hospital, this programme of the Department of Paediatrics not only functions as the only tertiary paediatric cardiac center for Hong Kong but also provides cardiological training to our local paediatricians- in-training as well as doctors from China and other Southeast Asian countries. Active academic activities in the various sub-sections like echocardiography, cardiac morphology, invasive cardiology and neonatal cardiology etc., serve to keep the unit at the frontier of paediatric cardiology.

What new developments will take place in the near future?
Interventional radiology will undoubtedly gain in popularity and further extend its applications. Non-invasive cardiology, which is already well developed, will become more sophisticated through incorporating new computer technology. Invasive investigatory catheterization will continue but, to a certain extent, lose popularity to the more non-invasive options. Cardiac electrophysiology will certainly develop in the next year or two to assume an important position in paediatric cardiology adding new academic and clinical activities to our programme. Research and clinical activities in neonatal cardiology, which constitute the major challenge to the clinicians, are presently receiving our prime attention.

K.C.L.

COMMUNITY PAEDIATRICS

An extensive longitudinal study covering broad aspects of child health was begun in 1967 by Professor C.E. Field and Dr. F.M. Baber. This work, which followed a cohort of children for almost 20 years, did much to identify child health needs during the early years of the department. At the same time a Child Development Centre was established at Yau Ma Tei and teaching at Maternal and Child Health Clinics (MCH) was included in the undergraduate curriculum.

Several studies have been conducted by Dr. F.M. Baber and Dr. A. Li to delineate the nutritional status of Hong Kong children.

In 1982 a 2 year longitudinal study to establish local epidemiology for gastroenteritis in the community was begun. This study was a joint project with the United Christian Community Service and the Department of Microbiology. Preliminary results for children less than 2 years show the average diarrhoeal rate to be 0.69 episodes per child per year; a comparable figure to those reported previously for developed countries.

In 1984 a study was conducted by Dr. C.L. Yu and Professor C.Y. Yeung to study the incidence of lead poisoning in Hong Kong school children. A follow-up study was done looking specifically at children of fishermen. These children were thought to be at greatest risk for lead poisoning. For this group data was collected concerning diet and the incidence of anaemia and lead poisoning.

Currently, in conjunction with the Department of Community Medicine, medico-social aspects of Paediatrics are taught during seminars following supervised home visits to an assigned family.

Rapid socioeconomic change in Hong Kong has resulted in a changing spectrum of child health needs and community problems. We now feel that our teaching of Community Paediatrics should be diversified, and included in plans for the Department's development is increased community based service and teaching.

Department staff are actively involved in the following community associations :—

Cooley's Anaemia Association

Federation of Chinese Canadian Professionals

Haemophilia Association of Hong Kong

Heep Hong Club

H.K. Association for the Mentally Handicapped

H.K. Association of Maternal & Neonatal Health

H.K. Society of Child Health & Development

H.K. Spastics Association

John F Kennedy Centre

Kwun Tong Community Health Project

Medical Council (H.K.) Licentiate Committee

Society for the Relief of Disabled Children

UNICEF (H.K.)

G.M.S.

DEVELOPMENTAL PAEDIATRICS AND NEUROLOGY

Interest in developmental paediatrics in the department dates back to 1967 when Professor C. Elaine Field first started a child developmental study centre to examine the physical and psycho-social development of Chinese children in Hong Kong. Subsequently Dr. F.M. Baber, a co-worker in this project, joined the department in 1978. She, in conjunction with Dr. C.W. Chan, had helped to develop a developmental screening program for Chinese children.

A Child Development and Rehabilitation Centre was proposed by Professor C.Y. Yeung in 1980, to provide a comprehensive assessment and therapeutic programme for children with various handicaps. The proposal was well received and supported by the Society for the Relief of Disabled Children. Plans to house this program at the Duchess of Kent Hospital materialised early this year(1987). We are extremely grateful to the Society for providing the entire fundi for this building project.

A temporary clinic was started at Duchess of Kent Hospital in 1982, with Drs. W.Y. Chan-Lui, F.M. Baber, L. Low and M.Y. Cheng participating in its various activities. From 1986, Dr. V. Wong has assumed major responsibilities at the clinic. The clinic, under a new name, 'Child Assessment Centre', is fully subvented by the Government as one of the several Assessment Centres as proposed by Professor K.S. Holt in 1973. We are lucky to have the support of the Society for the Relief of Disabled Children for other clinical and academic activities not subvented by the Government. We are now offering a full range of assessment for growth and development including psychological, behavioral, speech and hearing problems. We hope to develop into a regional resource for the various district child health services.

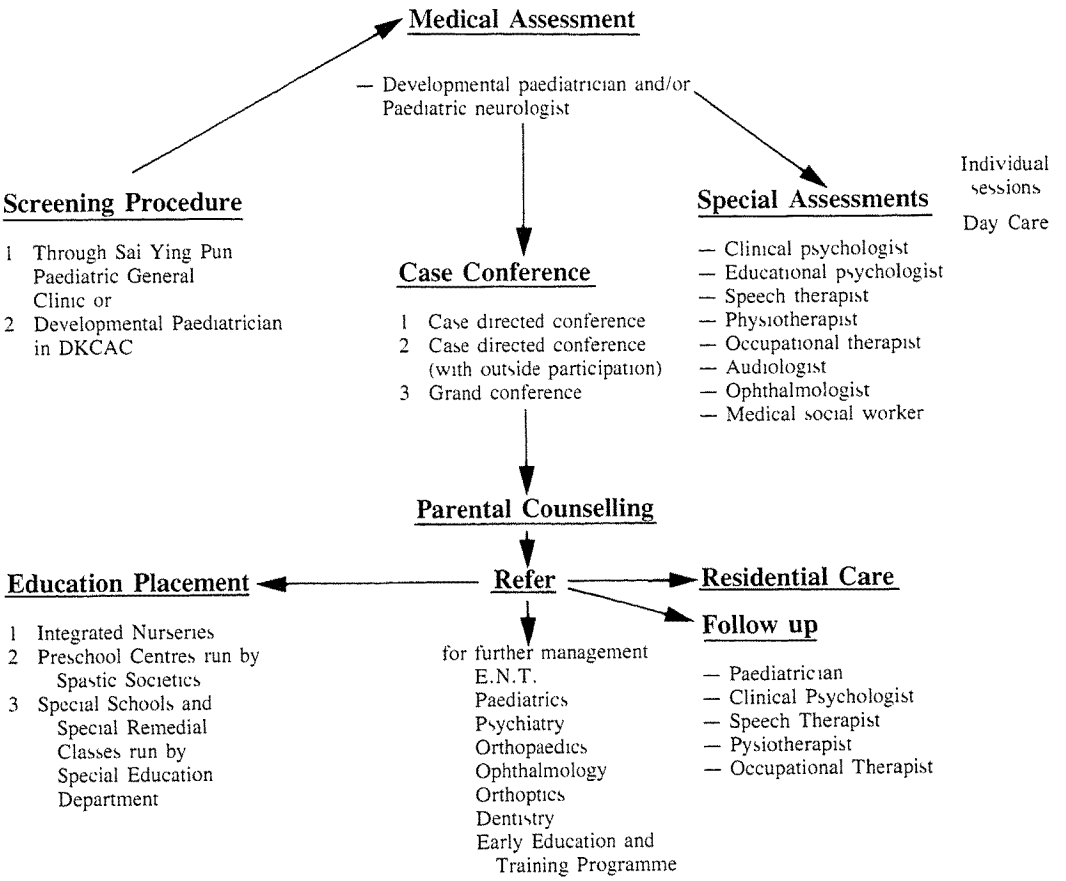
Child neurology began, when the department first started, as a specialty clinic held in Sai Ying Pun Jockey Club Clinic. Dr. W.Y. Lui was among the first to train in this discipline and subsequently helped to develop it further. A close liaison and working relationship is maintained with other health care professionals in David Trench Rehabilitation Centre, Wan Chai Polyclinic and Sai Ying Pun Jockey Club Clinic.

The current policy of the department is to combine neurology and developmental paediatrics into one clinical program. We have recruited a number of well qualified and interested health care professionals including nurses, psychologist, speech therapist, social worker, physio- therapist andccupational therapist for this service. Members of the department have also served as consultants and advisors to various associations and societies for the care and welfare of handicapped children in Hong Kong.

V.W.

DUCHESS OF KENT CHILDREN'S HOSPITAL

CHILD ASSESSMENT CENTRE



ENDOCRINOLOGY

The Paediatric Endocrine Clinic was set up in September 1982 in Queen Mary Hospital under the direction of Prof. C.Y. Yeung. This subspecialty is now run by a senior lecturer and a lecturer who also supervise the in-patient management and investigation of children with endocrine problems. Between twelve to fifteen children with a variety of endocrine disorders are seen in the Endocrine Clinic each week. The paediatric Endocrine team works closely with the endocrinologists of the University Department of Medicine, with joint clinical grand rounds and a joint research Growth and Pubertal Disorder Clinic held in the Metabolic Ward each week. Our current interest is in the use of growth hormone releasing hormone in children with short stature and LHRH-analogue in children with precocious puberty. The endocrine team also supervise the running of the neonatal screening program for congenital hypothyroidism in Queen Mary Hospital and Tsan Yuk Hospital.

L.C.K.L.

HAEMATOLOGY & ONCOLOGY

In the last 25 years, revolutionary changes have taken place in the field of haematology and oncology. The following review of what has happened within this department is to some extent a reflection of the rapidly changing international scene.

Severe Neonatal Jaundice

After the establishment of this department in 1962, the major haematology problem that surfaced was that of severe neonatal jaundice. Exchange transfusion, the only effective treatment then, was a "routine procedure", and negotiations for blood for this procedure demanded a great deal of effort. It was soon realized that most Chinese babies with severe neonatal jaundice were deficient for the enzyme erythrocyte glucose-6-phosphate dehydrogenase (G-6-P D). The incidence of severe neonatal jaundice fell progressively after the 1970s. Wide spread cord screening is now practised on almost all newborns and severe neonatal jaundice is much less common.

Cooley's Anaemia

For about ten years the prognosis for patients suffering from Cooley's anaemia was so poor that doctors felt that they were not worth treating. Blood transfusions were given to keep them barely alive. Later on as blood supply improved with an improved blood donation service, patients were kept at higher haemoglobin levels and had a slightly better quality of life. Nevertheless, hardly any reached adulthood, and almost all died of heart failure before puberty. In 1979, this department took the lead in Hong Kong in introducing home subcutaneous iron chelation therapy, but met staunch resistance from most parents. To tackle this difficult problem of non-compliance, the idea of forming a parents' group was first conceived. Miss Eva Liu, the hospital medical social worker, offered invaluable help to me in the formation of the Cooley's Anemia Association of Hong Kong, which was formally registered in 1982. The Association becomes the most effective venue through which parents and patients come to understand the disease and its treatment. In September 1986, a record number of thirteen patients were attending high school. Seven other patients had already left this department to join the adult unit. For the first time in medical history, these patients can look forward to a future with reasonable hope.

Haemophilia

Before 1970, in patients who had been suspected to suffer from haemophilia, clinicians had to spend many hours doing the tedious laboratory tests themselves in order to establish the diagnosis. It was ironical because after the diagnosis was made, no treatment could be offered. Young patients often died with intracranial haemorrhage at a tender age. In the mid 1970s, rapid technical advances in the manufacture of blood products made lyophilized factor VIII and factor IX concentrates commercially available. This has had a profound impact on the management of such patients. This department in 1975 was the first in Hong Kong to promote "home therapy" in such patients, and many practising doctors helped in giving the injections on an outpatient basis. This form of therapy reduced markedly both the length of hospital stay and the children's absence from schools, and is now operating in fourteen families in this department; nevertheless, it is not without its problems because there are no liaison personnel to bridge the gap between the clinician and the patients.

Since 1982, I have received generous help from Miss Helen Leung, Miss Anita W.L. Wong and Miss Anita Y.M. Wong, all medical social workers, who made significant contributions towards the eventual establishment of the Hong Kong Haemophilia Association. This association registered as a society in April 1985, and includes both adults and parents of affected children. It is hoped that, in due course, this organization can provide the help and support these patients need so badly at this difficult time when they are facing a hostile community.

Childhood Cancer

In the years before 1970, childhood cancer including acute leukaemia did not make much of an impression because patients died quickly and they soon disappeared from sight. Reports that acute leukaemia was a curable disease appeared in 1971 and this revolutionized their prognosis. The first leukaemic patient cured in this department confirmed the effectiveness of such a treatment regime. There are now many cured leukaemic patients and some are still returning to the follow up clinics. The updating of the hospital haematology laboratory service in recent years has been a welcome development making things slightly easier for the clinicians.

Reports on improved prognosis and cure for children with solid tumours followed that of acute leukaemia. It became obvious from overseas experience that an oncology team embracing multiple disciplines gave the best treatment results, but this was non-existent in this hospital. In 1973 the Paediatric Tumour Study Group was established with the aim to gather local data, and hopefully, to lead to the eventual formation of a similar team. This study group gave the first report on the types of childhood cancer seen in this locality. However, the establishment of a multi-disciplinary team for the treatment of childhood cancer remains elusive. A recent study within the department indicated other areas in patient management which require urgent improvement.

x x x

In the last two decades, medical knowledge has advanced at an astonishing pace, and haematology is an outstanding example. The field is now considered to be one of the widest in range, and it relates closely to laboratory medicine, immunology, cytogenetics, blood transfusion, and molecular biology. At the same time the emergence of the "total patient care" concept has brought about a fresh approach eliciting expert help from paramedical staff towards the care of such chronic patients. In the field of oncology, the evolution of a multi-disciplinary team in developed countries has given impressive results for the outcome of children treated for cancer, and this has come to be regarded as the most ideal approach.

In spite of many limitations, this department has strived strenuously to update the clinical care in patients with blood disease and cancer; this is obviously necessary for the teaching of undergraduates, if not for any other reason, but the manpower problem has shown little change over the years. Since care of general patients still commands the main effort of almost all staff members in the department, other developments are considered as a secondary priority. Further upgrading of patient care and research activities in this field depends largely on whether the existing difficulties, which have hitherto prevented staff members from devoting more time and effort to this specialty, can be successfully overcome.

A.M.C.L.

The following are staff members who have contributed to the development of this field in the department:
Dr. Brian Koon-Hung Luke, (1967-68)
Dr. Kwan-Yuen Wong, (1972-74)
Dr. Patrick Man-Pan Yuen (1974-84)
Dr. Anita Ming-Cheng Li (1974-present)
Dr. Garythe Samuda (1982-present)
Dr. Man-Yung Cheng (1984-present)

INTENSIVE CARE

Modern intensive care started off as a branch of anaesthesia with the advent of the ventilator. In the Department of Paediatrics, the story began with the purchase of the first "paediatric" ventilator in the late 1960s, the pressure-cycle Bennett PR-2; the neonates were ventilated by the Amsterdam ventilators. With limited resources and manpower, we endeavoured to ventilate critically ill children with respiratory failure in the wards. It soon became obvious that having the apparatus alone was not enough. One vital factor for the survival of these children was nursing care, and expertise in nursing critically ill children and babies could only be gained by concentrating both the nurses and the patients in one place. In the mid 1970s part of the children's private ward, E1, was converted into a neonatal unit and a small 3-bed room for treating very sick children.

As intensive care is a new and rapidly expanding branch of medicine, young medical and nursing staff often find themselves bewildered by new methods of treatment or unfamiliar equipment. Continual education therefore becomes an integral part of providing a satisfactory service. The department was instrumental in pioneering formal courses on paediatric intensive care for nurses in 1982. At the time of writing, some 30 nurses are attending the 3rd round of such a course. More is to come in the years ahead as paediatric intensive care nurses will be in great demand. To overcome the problem of a high turnover rate of nurses in the PICU and NICU, regular biweekly lectures on relevant topics are given by the staff physicians to the nurses and physiotherapists. In addition, an education subcommittee was formed by the nurses to oversee the training of new staff nurses coming to the PICU.

It was not until late 1982 that a better equipped 4-bed Paediatric Intensive Care Unit (PICU) was built in this area. Nursing service is now provided on a one to one basis in day time and one to two basis at night. The laboratory service has met the challenge of development well by providing an efficient 24-hour service for all major urgent laboratory tests. The only laboratory instrument in the PICU is a blood gas machine which is capable of providing a result within two minutes for the very urgent cases. The medical staff has also been re-organized so that there is a team of doctors responsible for paediatric and neonatal intensive care. This consists of 2 physicians, 2 registrars and a house officer. They share the responsibility for the PICU, the neonatal intensive care unit (NICU), and the neonatal nursery in E1. Several other second call registrars help out for the night coverage. Together with the doctors taking care of the Neonatal Service at Tsan Yuk Hospital and A7 at Queen Mary they make up the "ICU team", rotating among themselves to staff the PICU and NICU at the various hospitals. The physiotherapy service in the PICU and NICU at Queen Mary has a team of two or three members covering physiotherapy needs during office hours. Proposals have been made to expand such service throughout the day. Apart from the patients, the extensive medical equipment in the PICU and NICU also need attention. The Electric and Mechanical Services of the hospital have not been able to supply routine maintenance services; therefore a nurse is trained to do the job. She also teaches other staff the proper usage of equipment and liaises with the hospital on repairs and purchase of spare parts and consumables.

The medical staff, apart from gaining experience through managing critically ill patients, hold monthly academic meetings to elucidate management policies and to bring the group up to date on recent progress. A monthly statistics meeting is held in which cases of interest are reviewed with the help of the hospital pathologist. An infection round is also held weekly to discuss patients with infection in the PICU. This is a joint meeting with the hospital bacteriologists and virologist.

To further promote the spirit of cooperation, a ward committee has been formed with representatives from both doctors and nurses to discuss general issues and policies in the PICU and NICU. There is an honest exchange of ideas to improve services and engender cooperation.

With the very heavy work load imposed upon the team of doctors, research has not been given the attention it is due. However, the team has still tried to keep some work going. One of the on-going projects is the study of neutrophil function of normal and sick neonates. Our laboratory now utilizes about 2 ml of blood to assay neutrophil phagocytosis, candidacidal activity and chemotactic activity. Another project involves the study of transcutaneous blood gas measurements in normal and critically sick neonates and children. This allows an assessment of the patient's capacity for exercise (feeding) and ability to withstand other forms of stress, like weaning from the ventilator and tube feeding.

The intensive care service of the department is now in its infancy and there is ample room for improvement. With more medical and nursing staff obtaining relevant training abroad and locally, it is hoped that the standard and quality of service will be improved. Although more than two million dollars' worth of equipment has been procured in recent years, there are still a number of deficiencies. In the new Queen Mary K Block extension there will be a 16 bed PICU and 30 bed NICU. Hopefully the problem of equipment and space can then be solved. Research can be viewed as a long term investment and its vitality depends on the adequate provision of manpower. Last but not least, every effort should be made to ensure a stable team of nurses and doctors. Only in this way will expertise and experience accumulate and be further developed.

A.Y.C.T.

NEONATAL MEDICINE

Since its inception in 1962, the department has been involved in the management of neonates born in both Queen Mary Hospital and Tsan Yuk Hospital. We have also looked after cases referred from other hospitals. Currently, we have 2 senior staff and 2 junior medical officers working in the nursery at Tsan Yuk Hospital. There are also 1 senior staff and 1 junior medical officer at the Queen Mary Hospital nursery.

The first doctors of the department to specialise in neonatology were Dr. C.Y. Yeung (1966) and Dr. Victor Yu (1972). Other doctors who were sent overseas for specialised training in neonatology have included Dr. C.M. Chung (1974), Dr. S.P. Lau (1981), Dr. T.F. Fok (1982), and Dr. S.N. Wong (1986). Dr. Y.C. Tam (1984) and Dr. N.S. Tsoi (1986), who trained in intensive medicine, have also contributed to the neonatal programme together with several other senior staff.

In the past, newborn babies in Tsan Yuk Hospital were looked after by specially assigned doctors like Dr. June Leigh from the Obstetric Unit. There was only a part time paediatrician from our department available for consultations and ward rounds. Re-arrangement of the neonatal service in Tsan Yuk Hospital began around 1974 when our department became involved in the daily care of the sick neonates. More attention and resources were devoted to the specialty. A re-organisation was implemented in 1980 when two senior doctors were permanently assigned to the neonatal unit of Tsan Yuk Hospital to supervise the residents. This was followed by generous support from the government to purchase a large quantity of modern clinical equipment. A new neonatal laboratory was also established in 1982.

A similar re-organisation of the neonatal service was implemented at Queen Mary Hospital in 1980. Senior staff were specially assigned to supervise the neonatal work, resident teaching and the laboratory support was upgraded. It is gratifying to note the marked improvement in the neonatal survival results in recent years. At Queen Mary Hospital, the survival of very low birth weight infants has increased from 20% before implementation of the ICU to 66% after the ICU was implemented. At Tsan Yuk Hospital, similar improvement was observed with the overall neonatal mortality rate decreasing from 8.7 in 1974 to 4.7 in 1985. Many doctors, in particular Drs. H. Ip, C.M. Chung, S.P. Lau, and T.F. Fok have made significant contributions to the Tsan Yuk Hospital programme.

We have recently established a group of intensivists to provide the supervision and service for both the general paediatric ICU and the neonatal ICU / special care baby units at Queen Mary Hospital and Tsan Yuk Hospital. Drs. Y.C. Tam and N.S. Tsoi are providing a leading role in this programme. Since 1980, we have also increased the teaching of neonatal medicine to the clinical students studying obstetrics in Tsan Yuk Hospital. Regular in-service teaching has also been instituted to upgrade the knowledge and skills of the neonatal nurses.

There is also a plan to expand the Tsan Yuk Hospital nursery and to increase the intensive care beds to 8. With the completion of K block in Queen Mary Hospital in 1989, we shall be able to provide a modern and well equipped neonatal intensive care unit. We hope that we can then offer a much improved service to the community.

In the early days, research activities in neonatal medicine focussed mainly on erythrocyte Glucose-6-phosphate dehydrogenase (G6PD) deficiency, neonatal hypoglycaemia and neonatal jaundice. Pioneer work in these areas are still quoted by many authors. Although our research now includes other projects, these topics are still of major interest in the department today.

N.S.T.
C.Y.Y.

NEPHROLOGY

In 1958 there were many nephrotic patients and mortality was high. Dr. W.S. Choy and Dr. Y.C. Tsao started a follow-up clinic for these patients. This renal clinic was one of the first paediatric specialist clinics in Hong Kong. At this time the information from renal biopsies was not available for patient management. The study of renal biopsies was started in 1963 in collaboration with Dr. W.C. Chan. It was he who developed thin layer sectioning, special staining, immunofluorescence techniques, and later electron microscopy examination for use in Queen Mary Hospital. The results of a study presented in an international conference in 1967 aroused much interest and subsequently led to a joint study with the International Study of Kidney Diseases in Children. Laboratory facilities to study patients with tubular problems were also set up. Urological patients were taken care of mainly by surgeons, notably Dr. Paul C.K. Yue. Dr. Y.C. Tsao continued to take care of renal patients until 1973. During the next few years the renal clinic was, during different periods, subsequently run by Dr. John Lee, Dr. C.M. Chung, Dr. Paul Y.S. Ko and Dr. Edwin C.L. Yu. At times there was no coordinating figure.

The first attempt to treat children with end stage chronic renal failure was started in 1980. A girl was started on Continuous Ambulatory Peritoneal Dialysis (CAPD). Subsequently, after 1982, all children with end stage renal failure, where treatment was indicated, were maintained by dialysis and three such children were treated. The first child who received a cadaveric renal transplant has survived for 2½ years. All of these children are leading normal lives.

At present we have close liaison with other departments. Management of urological patients is assisted by the ready availability of radiological and isotopic imaging. Local ultrasonic standards for renal size have been obtained. Meetings with Paediatric surgeons are held to jointly manage and follow up urological patients. Systematic study of urinary tract infection is under way. Management of patients with renal problems is supported by a pathologist who is interested in ultrastructural pathology and immunological assessment. Patients with renal tubular problems have been studied in detail with the help of laboratory facilities. Joint meetings with the adult nephrology team are helpful in case management and study. Joint meetings with paediatricians interested in nephrology in other major hospitals are currently held to promote better collection of epidemiological statistics. A manual is in preparation, to act as a reference for paediatricians, on the current methods of management of children with renal disease.

C.L.Y.

PAEDIATRIC NURSING

Children and neonates require more nursing attention than adults. Nurses who are participating in paediatric care require special knowledge and skills.

A large number of senior nurses have helped to develop paediatric nursing in our hospital. Pioneer workers in the early days of the Department of Paediatrics who have devoted tremendous effort to the discipline include Sister P. Wong, Sister Kew and Sister Yue. Others, like Ms R. Lan, Ms S. Tong, Ms F.H. Chan, Ms P. Hong and many more have all continued with the good work.

Apart from the general paediatric wards, with isolation room and reverse-isolation room attached, areas of specialization were allocated in the Paediatric Unit in 1969. They were (a) the Neonatal Unit, (b) the Neonatal Surgical Unit, (c) the Premature Babies Special Care Unit, and (d) the Isolation Unit for children who require closer observation and special care. This room was enlarged and re-named the Paediatric ICU in late 1980.

The Premature Babies Special Care Unit was also converted to the Neonatal ICU in 1981. More new modern medical equipment has been introduced to the paediatric wards since then. The number of nurses assigned to work in the areas of specialization were gradually increased to more than double that of 1981.

To promote staff education, a series of lectures are given at regular intervals by the medical staff. Nurses working in the paediatric wards are enthusiastic to attend these to update their knowledge and skills. A number of nurses have gone overseas to obtain post registration training (RSCN, JBCNS and Paediatric Cardiac Thoracic Intensive Care) and have come back. They have been fully utilised in the paediatric wards in the past 15 years.

Assisted by the Department of Paediatrics, training courses on paediatric and neonatal intensive care were run by the nursing administration in 1983 and 1984 respectively. A total number of 60 nurses from the regional hospitals have completed the training course. The co-ordinators are Miss S. Chan, Miss C. So and Miss A. Yung. It is a pity that some of the nurses have left the service after the training. A further course was commenced in May this year.

Free visiting time to children, during day time, was recommended by Professor C.Y. Yeung and was put into effect in August 1983. Most of the parents are willing to participate in the care of their hospitalized child with instruction and supervision from the nurses. Children are benefited from this parent-professional partnership.

As knowledge and practice in paediatric nursing have expanded, continuing education programmes for nurses play an important part in optimal patient care. We need more trained and experienced nurses for care of children and neonates. We hope paediatric nursing may be recognised as a distinct specialty within the nursing profession to provide the best care needed for sick children.

P.H.

RESPIRATORY MEDICINE

The department has had a great interest in respiratory disorders since its inception, not only because of their prevalence but also because of Prof. C. Elaine Field's interest and established reputation in childhood bronchiectasis. Interest later focussed on asthma when members of the department started to study the effect of weather and emotional changes on the condition. Dr. Peter Lo, who spent 2 years with the department in the mid 1960s, helped to develop further interest in the study of various childhood allergies.

In the last decade, mortality from pneumonia has been reduced by more than half. This is no doubt due to many factors, including better laboratory support, new advances in pharmacology, and modernised equipment. Since the Government introduced BCG vaccination in 1950, tuberculosis, which was once common, is no longer a threat.

Allergic respiratory disease, however, remains the commonest cause of chronic illness in childhood. It is estimated that about 10% of our children suffer from it in different degrees of severity. The number of admissions to our department for asthmatic attacks has risen 4-fold in the past 15 years. The department has launched the following measures to tackle this problem:-

(1) Education of the public on the proper management of asthma: A slide talk on asthma is shown to the patient and family in the outpatient clinic before medical consultation. A Chinese pamphlet on how to prevent asthmatic attacks is distributed afterwards to reinforce the message. Every year,educational talks are held with the asthmatic family. Experts from different fields,including dietitians, paediatricians, psychiatrists, physiotherapists and medical social workers are invited to deliver talks and answer queries.

(2) To promote the general fitness of these children: Exercise is encouraged rather than restricted. A co-ordinated physiotherapy programme for asthmatic children was set up together with the Physiotherapy Department of David Trench Rehabilitation Centre in 1983, the proper breathing method was taught and faulty posture was corrected. Swimming classes are held every year in cooperation with the Physically Handicapped and Able Bodied Association. Last year, a physical conditioning programme was organised in the Jubilee Sports Centre with the help of Dr. Henrietta Ip. Children had 12 days of intensive indoor and outdoor exercise training followed by 12 weekly swimming drills.

Research in this discipline is active. The standard values of lung function for our local Chinese children have been established. Current research is mainly focussed on bronchial asthma and the lung function abnormalities of children with B-thalassaemia major.

O.C.

THE LABORATORY

The Laboratory of the Department of Paediatrics not only serves as a research laboratory but also provides clinical laboratory services such as haematology, clinical biochemistry, cytogenetics and other special laboratory tests for the paediatric patients. Paediatric laboratory technology differs greatly from general medical laboratory technology. A paediatric laboratory technologist takes great care of every sample of blood, urine and cerebrospinal fluid drawn from the tiny patients. Every technician takes pride in the accuracy of a test.

Routine Services

In the early 1970s, haematology and chemistry analysers for ultra micro or even micro measurement were not common.All tests in micro scale were done manually. The chemistry analysers in the Pathology Department, University of Hong Kong, required a considerable amount of blood. This procedure could not be applied to a paediatric patient, not to mention the small premature infant. Over 80% of the biochemical tests were done manually by micromethod in the Paediatric Laboratory. In addition to blood gas analysis, the tests included blood glucose, renal function tests, total calcium, magnesium and even some liver function tests. Blood cell counts for leukaemic children and small infants were performed mostly in the Paediatric Laboratory by direct capillary blood sampling from finger and heel pricks. This heavy work load continued till the 1980s when the laboratory was equipped with an automatic cell counter and some analysers. At the same time the ICU laboratory of the Pathology Department, University of Hong Kong, also became able to handle paediatric capillary samples.

In 1982, the neonatal laboratory in Tsan Yuk Hospital was established with positions for a number of technicians. Organization and management of this laboratory has since been the responsibility of the Laboratory Superintendent of the Paediatric Department. With financial support from the Government, we obtained the most sophisticated chemistry analyser (Roche Cobas Bio analyser) which can manage ultra micro serum samples. The Paediatric Laboratory is now properly equipped with the most up-to-date analysers and equipment to provide micro or ultra micro clinical laboratory services to the paediatric patients in both Queen Mary and Tsan Yuk Hospitals.

Research Laboratory Techniques

In the late 1960s Dr. Y. C. Tsao set up the dialysis and electrophoresis technique for serum and urinary proteins and haemoglobin, and an atomic absorption spectrophotometer for calcium and magnesium determination which was the most modern equipment at the time.

Following the interest of Prof. G. M. Kneebone in the early 1970s, the Paediatric Laboratory was geared to analyse the protein, fat, electrolytes and total caloric value in milk and food. The most up-date haemoglobin electrophoresis techniques on cellulose acetate and scanning quantitation by using the Helena electrophoresis system, column chromotography determination of haemoglobin A2 and clotting factor studies with the Clotek machine were introduced by Dr. A. Li in the mid-1970s.

A thyroid screening programme was started in 1977 by Prof. J. H. Hutchison and Dr. Henrietta Ip. The radioimmunoassay (RIA) technique is still being employed to determine T4, TSH, human growth hormone and ferritin levels. Cytogenetic techniques were started in 1964 in the Paediatric Laboratory by Dr. Alice Chau, the pioneer clinical geneticist, and improved with G-banding and other analysing methods in 1978 by Mr. F. T. Lee. In early 1982 Prof. C. Y. Yeung introduced the G6PD screening programme utilizing the automatic analyser to quantitate the enzyme activities. Recently, under the supervision of Prof. C. Y. Yeung, the following techniques were established:

Sephadex chromatography for unbound bilirubin
Horseradish peroxidase oxidation techniques for bilirubin protein binding
A rat model for foetal growth studies
The Gunn rat model for jaundice studies
Determination of lead and zinc levels in blood by flameless atomic absorption spectrophotometer
Enzyme-linked immunosorbent assay (ELIZA) technique for antibody screening
Candidacidal technique for leukocyte function
Gas chromatography technique for detection of carbon monoxide and hydrogen in the expired air of new-born babies

The Paediatric Laboratory also manages to run a small darkroom service to process slides and illustrative photographs for teaching and research purposes. However there is no professional photographer.

The multi-purpose Paediatric Laboratory has a total of 17 staff members, employed both by the University and the Government, to run the above mentioned comprehensive services. They consist of 5 technologists, 7 technicians and 5 laboratory assistants.

F.T.L.

Past medical staff

ABDURACHMAN S
China Medical Board Fellow 1981
Current Practice : Neonatalogist
Address : Indonesia.

ALVES MR R.M., L.M.& S. (Lisbon)
Temporary Lecturer 1974-75
Current Practice : Not known
Address : c/o Prof. G.M. Kneebone, Flinders Medical Centre, Bedford Park, South Australia 5042.

AU KK, Gary M.B.,B.S.(HK)
M.O. 1985
Current Practice : Private Practitioner
Address : 1B Amber Garden, 110 Blue Pool Road, Happy Valley, Hong Kong.

AU YEUNG YB M.B.,B.S.(HK)
M.O. 1971-73
Current Practice : Neonatologist
Address : Gainsberg, Florida, USA.

BABER FM M.B.,Ch.B., D.C.H., F.R.C.P.(Edin.)
Hon. Lecturer 1962-78;
Lecturer 1978-84;
Hon. Lecturer 1984-86
Current Practice : Retired
Address : 18 Cumnor Hill, Oxford OX2 9HA, England.

BUCHANAN JC, Andrew M.B.,B.S.(Melb.) M.R.A.C.P.
Lecturer 1974-77
Current Practice : Not known
Address : Not known

CHAN BW, Lily M.B.,B.S.(HK)
M.O. 1974-76
Current Practice : Private Practitioner
Address : Shop F, Mei Sun Lau B, 489 Queen's Road West, Hong Kong.

CHAN CW M.B.,B.S.(HK), M.R.C.P.(1977)*, F.R.C.P.(Edin)
M.O. 1972-78; S.M.O. 1978-81;
Hon. Lecturer 1981- present
Current Practice : Private Practitioner
Address : Room M-10 Hennessy Centre, 500 Hennessy Road, Hong Kong.

CHAN KH, Alex M.B.,B.S.(HK), M.R.C.P.(1980)*
M.O. 1976-82
Current Practice : Consultant, Caritas Medical Centre
Address : Caritas Medical Centre, 111 Wing Hong St., Sham Shui Po, Kowloon.

CHAN PH M.B.,B.S.(HK)
Clinical Assistant 1964
Current Practice : Private Practitioner
Address : 3/F Flat C, Po Wing Bldg., 114 Percival St., Hong Kong.

CHAN SP M.B.,B.S.(HK), M.R.C.P.(1986)*
M.O. 1984-87
Current Practice : Paediatric Resident
Address : c/o Mr. Donald Mak, 33 Palisades Ave., Creskill, N.J. 07626, U.S.A.

CHAN T, Eddie M.B.,B.S.(HK), M.R.C.P.(1982)*
M.O. 1978-82
Current Practice : Private Practitioner Hon. Clinical Tutor, Dept. of Community Medicine, Hong Kong Chinese University
Address : Shop 41 Chi Lok Fa Yuen, Tuen Mun, N.T.

CHAN TS M.B.,B.S.(H.K.)
M.O. 1981-85
Current Practice : M.O., Family Clinic
Address : Family Clinic, L Block, Queen Elizabeth Hospital, Kowloon.

CHAN-LUI WY M.R.C.P.(Edin.& Lond. 1968), M.D.(HK)(1980)*, F.R.C.P.(Edin.1978)* (Lond.1983)*(Glas.1985)
M.O. 1965-66; Lecturer 1969-76; Senior Lecturer 1976-84; Reader 1984-85
Address : Apartment 302, 75 Swindon Way, Tuxedo, Winnipeg, R3P OT8, Manitoba, Canada.

CHAN-YEUNG WL, Angeline M.B.,B.S.(HK), F.R.C.P.(C)
Assistant Lecturer 1968-69
Current Practice : Private Practitioner, Child Psychiatry
Address : 600 Sherbourne St., No. 604, Toronto, Ontario, Canada M4X 1W4.

CHAU SH, Alice M.B.,B.S.(HK), F.R.C.P.(Edin.) C.P.
M.O. 1962-69;
S.M.O. 1969-75;
Hon. Lecturer 1963 — present
Current Practice : Consultant
Address : Paediatric Unit A, Queen Elizabeth Hospital, Kowloon.

CHENG SK M.B.,B.S.(HK)
M.O. 1973
Current Practice : Private Practitioner
Address : Shop 6A, Level 5, Greenwood Terrace Commercial Complex, Tai Man Street, Chai Wan, Hong Kong.

CHEUNG KY, Andy M.B.,B.S.(HK)
M.O. 1973-75
Current Practice : Private Practitioner
Address : Shop 30, Wang Kwong Lau, 33, Ngau Tau Kok Road, Kowloon.

CHEUNG YK, Anson M.B.,B.S.(HK), M.R.C.P.(1984)*
M.O. 1980-86
Current Practice : Paediatrician
Address : Po Leung Kuk Joyce Kwok Paediatric Clinic, Units 5 & 6 Ground Floor, Wing Lok House Fuk Loi Estate, Tsuen Wan, N.T.

CHEUNG YW, Arnold M.B.,B.S.(HK), M.R.C.P.(1972)*
M.O. 1966-70; Hon. Lecturer 1971-74
Current Practice : Private Practitioner
Address : Room 905 Hang Seng Bank Bldg, 677 Nathan Road, Kowloon.

CHING ML, Rosa M.B.,B.S.(HK)
M.O. 1976
Current Practice : M.O., Outpatient Dept.
Address : Outpatient Dept., Kowloon Hospital

CHU SY M.B.,B.S.(HK), M.R.C.P.(1983)*
M.O. 1979-83
Deceased

CHUNG CM M.B.,B.S.(HK), M.R.C.P.(1977)*
M.O. 1970-76; S.M.O. 1977-78; Hon. Lecturer 1977-84
Current Practice : Private Practitioner
Address : 34 First Farm Drive, Castle Hill, NSW 2154, Australia.

CHUNG SH, Gabriel M.B.,B.S.(HK), M.R.C.P., F.R.C.P.(C
Assistant Lecturer 1966-68;
Hon. Lecturer 1975-76
Current Practice : Private Practitioner
Address : Room 609 Hang Shing Bldg, 363 Nathan Road, Kowloon.

DIU KC M.B.,B.S.(HK), M.R.C.P.(1974)*
M.O. 1970-74
Current Practice : Private Practitioner
Address : Stage 6 Podium F1, 2B Humbert St., Mei Foo Sun Chuen, Kowloon.

FIELD CE M.D., F.R.C.P.(Lond)
Professor 1962-71
Current Practice : Retired
Address : 77 Homelawn House, Brookfield Road, Bexhill-on-sea, E. Sussex, England TN40 1PN.

FOK TF M.B.,B.S.(HK), M.R.C.P.(1980)*
M.O. 1977-78; Lecturer 1978-84
Current Practice : Senior Lecturer
Address : Dept. of Paediatrics, Prince of Wales Hospital, Shatin, N.T.

FOK WG M.B.,B.Ch.(Wits), D.A.(SA)
M.O. 1984
Current Practice : Anaesthetic Registrar
Address : P.O. Box 389, Kelvin, 2054 South Africa.

FUNG HP, Robert B.A.(Harv.), M.D., C.M.(McGill), F.R.C.P.(C)
Lecturer 1967-68;
Hon. Lecturer 1968 — present
Current Practice : Private Practitioner
Address : 219 Holland House, 9 Ice House St., Hong Kong.

FUNG KP M.B.,B.S.(HK), M.R.C.P.(1981)*
M.O. 1979-87
Current Practice : Senior Lecturer
Address : Dept. of Paediatrics, National University of Singapore, South Kent Ridge, Singapore 0511.

FUNG KP, Charles M.B.,B.S.(HK), M.R.C.O.G.
M.O. 1974
Current Practice : Consultant, O & G Unit, Caritas Medical Centre
Hon. Lecturer, University O & G Unit, University of Hong Kong
Address : Obstetric Dept., Caritas Medical Centre,111 Wing Hong Street, Kowloon.

FUNG YK M.B.,B.S.
Temporary Assistant Lecturer 1968
Current Practice : Private Practitioner
Address : P.O. Box 212 Dural, 2158 New South Wales, Australia.

FUNG YT M.B.,B.S.(HK), F.R.C.P.(C)
M.O. 1963-64
Current Practice : Private Practitioner
Address : 180 Sherway Drive, No 8, West Mall Medical Bldg, Queensway General Hospital, Etobicoke Ont M9C

HOPKINS J MA
Hon. Lecturer 1968-69
Current Practice : Not known
Address : Not known

HOWARD JC M.B.,Ch.B.(Liv.), M.R.C.P.
Hon. Lecturer 1975-83
Current Practice : Private Practitioner
Address : Room 1007, Gammon House, 10/F 12 Harcourt Road, H.K.

HU FJ
China Medical Board Fellow 1985
Current Practice : Paediatrician
Address : Dept. of Paediatrics, Sun Yat-Sen University of Medical Sciences, Guangzhou, The People's Republic of China.

HU SC M.B.,B.S.(HK), F.R.C.P.(Edin)
Head, Govt. Paed. Unit, QMH. 1957-62;
Hon. Lecturer 1962-81
Current Practice : Private Practitioner
Address : Room 802 Central Bldg., Queen's Road Central, Hong Kong.

HUTCHISON JH
M.D., F.R.C.P.(Lond., Glas. & Edin), F.R.S.(Edin), F.A.C.P.(Hon.)
Professor 1977-80
Current Practice : Retired
Address : 3 Kelvin Court, Glasgow, G12 0AB, England.

IP MH, Henrietta O.B.E., J.P., M.B.,Ch.B.(Liv.), M.R.C.P.
Lecturer 1974-78
Current Practice : Private Practitioner
Address : 8/F, Duke Wellington House, 14-24 Wellington Street, Hong Kong.

IP PF M.B.,B.S.(HK), M.R.C.P.(1975)*
M.O. 1971-75
Current Practice : Private Practitioner
Address : 713B Champion Bldg., 301-309 Nathan Road, Kowloon.

KNEEBONE GM M.B.,B.S.(Adel.), M.Sc.(Pitt.), F.R.A.C.P.
Professor 1971-75
Current Practice : Professor
Address : Flinders Medical Centre, Bedford Park, South Australia 5042.

KO KH M.B.,B.S.(HK)
M.O. 1979-83
Current Practice : Private Practitioner
Address : Shop 97 Ngan Shing Commercial Centre, City One, Shatin, N.T.

KO SC M.B.,B.S.(HK), F.R.C.P.(Edin)
S.M.O. 1976-77
Current Practice : Private Practitioner
Address : 702 Hang Lung Centre, 2-20 Paterson Street, Hong Kong.

KO YS, Paul M.B.,B.S.(NSW), M.R.C.P.(1979)*
Lecturer 1975-82
Current Practice : Private Practitioner
Address : 801 Lansing House, 41-47 Queen's Road C, Hong Kong.

KWOK CW M.B.,B.S.(HK), M.R.C.P.
M.O. 1980-84
Current Practice : Private Practitioner
Address : Flat 131 1/F Commercial Complex, Richland Gardens, Kowloon Bay, Kowloon.

LAI PY, Michael M.B.,B.S.(HK)
M.O. 1965-66
Current Practice : Private Practitioner
Address : 6 Chung Wui St., G/F, Tai Kok Tsui, Kowloon.

LAM, Paul M.B.,B.S.(HK)
M.O. 1966-68
Current Practice : Private Practitioner
Address : 401 Castle Peak Road, 1st Floor, Kowloon.

LAM ,TK M.B.,B.S.(HK) M.R.C.P.(1984)*
M.O. 1981-85
Current Practice : Lecturer
Address : Dept. of Paediatrics, Prince of Wales Hospital, Shatin, N.T.

LAU, Anthony M.B.,B.S.(HK)
M.O. 1974-75
Deceased

LAU DY M.B.,B.S.(HK), M.R.C.P. (1976)
M.O. 1972-75
Current Practice : Private Practitioner
Address : Room 1305 Hang Shing Bldg., 363 Nathan Road, Kowloon.

LAU SP, Sam M.D.(Munich), M.R.C.P.
Lecturer 1978-85
Current Practice : Private Practitioner
Address : Room 1301 Champion Bldg., 301-309 Nathan Road, Kowloon.

LEE TY, Thomas M.B.,B.S.(HK), M.R.C.P.(1972)* Dip.Occ.Med. (CUHK)
Assistant Lecturer 1969-71;
Hon. Lecturer 1974 — present
Current Practice : Private Practitioner
Address : Drs. Anderson & Partners, 16A, United Centre, Queensway, Hong Kong.

LEE YK, John M.B.,B.S.(HK), M.R.C.P.(1975)*
M.O. 1970-75; Hon Lecturer 1976-78
Current Practice : Private Practitioner
Address : Suite 1-C 1/F, 521 Nathan Road, Kowloon.

LEE YS, Robert Johnson M.B.,B.Ch.(Rand.), M.R.C.P.(1964)*
M.O. 1959-65; S.M.O. 1965-66; Hon. Lecturer 1968-72
Current Practice : Private Practitioner
Address : Rm 501 Edward Mansion, 141 Prince Edward Road W., Kowloon.

LEUNG CK M.B.,B.S.(HK), F.R.C.S.(Edin.)
M.O. 1963-64
Current Practice : Private Practitioner
Address : 9/F, Kam Sek Commercial Bldg., 17 Waterloo Road, Kowloon.

LEUNG NK M.B.,B.S., M.R.C.P.(1972)*, F.R.C.P.(Edin)
M.O. 1968-71; Hon. Lecturer 1976–present
Current Practice : Consultant
Address : Paediatric 'A' Unit, Princess Margaret Hospital, Kowloon.

LEUNG SN, Kevin M.B.,B.S.(HK)
M.O. 1965-67
Current Practice : Private Practitioner
Address : 955 Queen St. E., Suite 40, Sault Ste. Marie, Ont. P6A 2C3 Vancouver, Canada.

LI MC, Anita M.B.B.S.(HK), M.R.C.P.(1969)*, F.R.C.P.(Edin. 1979)*
M.O. 1964-70; Lecturer 1970-80; Senior Lecturer 1980-81
Current Practice : Senior Lecturer
Address : Dept. of Paediatrics, University of Hong Kong.

LI-GREEN, Jeffery M.B.,B.S.(SA)
M.O. 1986
Current Practice : Paediatric Registrar
Address : c/o G. Pilkington, 43 Burnley Terrace, Mt. Eden, Auckland 3, New Zealand.

LO KF, Peter M.B.,B.S.(HK)
Li Shu Fan Lectureship 1967-68
Current Practice : Private Practitioner
Address : 727 Nathan Road, 5/F Kowloon.

LUK SC M.B.,B.S., F.R.C.P.(C)
M.O. 1961-63
Current Practice : Consultant Pathologist
Address : Mount Sinai Hospital, Toronto, Canada.

LUKE KH, Brian M.B.,B.S.(HK), F.R.C.P.(C)
Li Shu Fan Lectureship 1966-68
Current Practice : Director, Haematology/Oncology
Chairman, Dept. of Laboratory Medicine
Head, Division of Haematology/Blood Bank
Address : Children's Hospital Eastern Ontario, University of Ottawa, 401 Smyth Rd., Ottawa, Ontario, Canada.

LUN KW, Raymond M.B.,B.S.(HK), M.R.C.P.(1986)*
M.O. 1982-87
Current Practice : Private Practitioner
Address : Shop B2, 2/F Shopping Mall, Sheraton Hotel, 20 Nathan Road, Tsimshatsui, Kowloon.

MAN CK, Gregory M.B.,B.S.(HK), F.R.C.P.(C)
M.O. 1972-73
Current Practice : Paediatrician
Address : York Central Hospital, 10168 Yonge St., Ste 203, Richmond Hill, Ont., L4C 1T6, Canada.

MATTHEWS R M.B.,B.S., F.R.A.C.P., M.R.C.P.
Research Assistant 1966
Current Practice : Haematologist
Address : Melbourne Children's Hospital.

NG WC M.B.,B.S.(HK), M.R.C.P.(1983)*
M.O. 1979-83
Current Practice : Private Practitioner
Address : Lower Wong Tai Sin Estate, S29B Wong Tai Sin Shopping Centre, Kowloon.

ORR WY M.B.,B.S.(HK),
F.P.Q.C.(Australia)
M.O. 1959-62
Current Practice : Private Practitioner
Address : Room G, 2/F
King's Centre, 193-203
King's Road, Hong
Kong.

PERALTA ML
China Medical Board Fellow 1986-87
Current Practice : Paediatrician
Address : 3451 M. Villamor St.,
Lourdes Subdivision,
Sta. Mesa, Manila,
Philippines.

SALVANI-BAUTISTA M
China Medical Board Fellow 1983
Current Practice : Paediatrician
Address : Phillipines Cardiac
Centre, Manila,
Philippines.

SHIU SM, Evelyn M.B.,B.S., M.R.C.O.G.
M.O. 1973
Current Practice : Private Practitioner
Address : 85 Waterloo Rd., 6/F
Kowloon.

SHUM S M.B.,B.S.
M.O. 1972-73
Current Practice : Private Practitioner
Address : 3720 Farwell Drive,
Amarillo TX 79109,
Texas, U.S.A.

SIN WK M.B.,B.S.(Sydney),
M.R.C.P.(1973)*
M.O. 1969-76; Hon. Lecturer 1976-85
Current Practice : Private Practitioner
Address : 177 Aberdeen Main
Road, 1/F
Aberdeen, Hong Kong.

SO KY M.B.,B.S.(HK)
M.O. 1973-74
Current Practice : Private Practitioner
Address : 154 Victoria Avenue,
Remuera, Auckland,
New Zealand.

SUE WK, David B.Sc., M.D.(McGill),
F.R.C.P.(C)
Lecturer 1972-74
Current Practice : Private Practitioner
Address : 3A Hang Seng Bank
Bldg., Causeway Bay
Branch, 28-30 Yee Woo
Street, Hong Kong.

SZETO J M.B.,B.S.(HK)
M.O. 1966
Deceased

TAM CW M.B.,B.S.(HK), M.R.C.P.
Hon. Lecturer 1974-77
Current Practice : Private Practitioner
Address : Room 7ll, Hang Shing
Bldg., 363 Nathan Road,
Kowloon.

TAM LS, John Alex M.B.,B.S.(HK)
Assistant Lecturer 1968; M.O. 1969-70
Current Practice : Private Practitioner
Address : 1201 Winter Hunt Road,
Mclean, North Virginia,
Virginia 22101, U.S.A.

TAM MY, Angela M.B.,B.S.(HK),
M.R.C.P.(1979)*
M.O. 1976-82
Current Practice : Private Practitioner
Address : Po Shan Mansion, Shop
No. P10, Tai Koo Shing
Stage 3, Hong Kong.

TAO SK, Billy M.B.,B.S.(HK),
M.R.A.C.P.
M.O. 1964
Current Practice : Private Practitioner
Address : Adelaide, Australia.

TAY KC M.B.,B.S.(HK)
M.O. 1964
Current Practice : Private Practitioner
Address : 74 Stevens Road
#02-01, Singapore 1025.

TEAINBOON P
China Medical Board Fellowship 1981-82
Current Practice : Private Practitioner
Address : Sydney, Australia

TO KS M.B.,B.S.(HK), M.R.C.P.(1971)*
M.O. 1967-70
Current Practice : Private Practitioner
Address : 211 Tung Ying Bldg., 100 Nathan Road, Kowloon.

TO WH M.B.,B.S.(HK), M.R.C.O.G.
M.O. 1978
Current Practice : Private Practitioner
Address : 79C Waterloo Rd., B5 Yee On Court, 15th Floor, Kowloon.

TONG CY M.B.,B.S.(HK), M.R.C.P.(1986)*
M.O. 1984-86
Current Practice : M.O., Virus Unit
Address : Virus Unit, Queen Mary Hospital

TONG TC M.B.,B.S.(HK), M.R.C.P.(1979)*
M.O. 1975-79
Current Practice : Private Practitioner
Address : M07 Hennessy Centre, 500 Hennessy Road, Hong Kong.

TSANG RC M.B.,B.S.(HK)
M.O. 1966
Current Practice : Director, Division of Neonatology Director, Perinatal Research, Institute
Address : Professor of Pediatrics, Obstetrics & Gynecology, University of Cincinnati Medical Center, 231 Bethesda Avenue, Cincinnati Ohio 45267-0541, U.S.A.

TSAO YC M.B.,B.S.(HK), M.R.C.P., F.R.C.P.(Edin)
Li Shu Fan Lectureship 1963-65; Lecturer 1965-68; Senior Lecturer 1968-71; Hon. Lecturer 1971 — present
Current Practice : Private Practitioner
Address : 1611 Wing On Centre, 111 Connaught Road C, Hong Kong.

TSE LY M.B.,B.S.(HK)
M.O. 1978-79
Current Practice : Acting S.M.O.
Address : Family Health Service, Medical & Health Dept., Hong Kong.

TSUI YS, William M.B.,B.S.(HK)
M.O. 1986-87
Current Practice : Private Practitioner
Address : 35 Shung Yau St., G/F, Kwun Tong, Kowloon.

USMAN A
China Medical Board Fellow 1984
Current Practice : Not known
Address : Indonesia.

WAI KH, Gregory M.B.,B.S.(HK), F.R.C.P.(C)
Lecturer 1973-78;
Hon. Lecturer 1978 — present
Current Practice : Private Practitioner
Address : Hong Kong Adventist Hospital, 40 Stubbs Road, Hong Kong.

WALDMAN E. M.B.,Ch.B, D.T.M. & H.
Research Assistant 1968-72
Current Practice : Community Physician
Address : The Richmond Fellowship, 8 Addison Road, London W14 8DL.

WAROUW SM
China Medical Board Fellow 1980
Current Practice : Not known
Address : Not known

WEI TH, PATRICK M.B.,B.S. (Hons.) (HK) M.R.C.P., F.R.C.P.(C)
Hon. Lecturer 1972-73
Current Practice : Private Practitioner
Address : 15/F, Hang Lung Bank Bldg., Hysan Avenue, Causeway Bay, H.K.

WONG CF, Paul M.B.,B.S.(HK), M.R.C.O.G.
M.O. 1963
Current Practice : Private Practitioner
Address : Room 903 Loke Yew Bldg., 50 Queen's Road C., Hong Kong.

WONG KK, Leo M.B.,B.S.(HK), M.R.C.P.(Lond), F.R.C.P.(Edin.& Glas)
M.O. 1963
Current Practice : Private Practitioner
Address : Room 301 Tung Ying Bldg., 100 Nathan Road, Kowloon.

WONG KK, Simon M.B.,B.S.(HK), M.R.C.P.(1966)*
M.O. 1962-68
Current Practice : Private Practitioner
Address : 4/F Room 402 Prince Commercial Bldg., 150 Prince Edward Road, Kowloon.

WONG KY M.B.,B.S.(HK)
Lecturer 1972-74
Current Practice : Associate Professor of Paediatrics
Address : Children's Hospital Medical Center, The Children's Hospital Research Foundation, Elland and Bethesda Avenue, Cincinnnati Ohio 45229, U.S.A.

WONG SF, Dora M.B.,B.S.(HK)
M.O. 1973
Current Practice : Private Practitioner
Address : 705 Union Bank Bldg., Central, Hong Kong.

WONG TK, Lawrence M.B.,B.S.(HK), F.R.C.P.(C)
M.O. 1970-74
Current Practice : Associate Director, Biochemical Disease Clinical Service,
Address : B.C. Children's Hospital, 4480 Oak Street, Vancouver, B.C. Canada V6H 3V4.

WONG YK M.D.(Birm.), F.R.C.P.(Edin)
Hon. Lecturer : 1983-81
Current Practice : Private Practitioner
Address : Room 503, 4/F, Winner House, 310, King's Road, North Point, H.K.

WONG YS M.B.,B.S.
Research Assistant 1968-69
Current Practice : Not known
Address : Not known

YEUNG CH M.B.,B.S.(HK), M.R.C.P.(1973)*
M.O. 1968-71
Current Practice : Private Practitioner
Address : 86 Starkey St. Forestville, NSW 2087, Australia.

YEUNG CY M.B.,B.S.(HK), M.R.C.P.(1967)*, F.R.C.P. (Edin., Glas. & Can.) 1963-68 Hon. C.R.C.P.(C),
M.O. 1963-6
Acting S.M.O. 1968-69
Hon. Lecturer 1968-72
Current Practice : Professor of Paediatrics
Address : Dept. of Paediatrics, University of Hong Kong, Queen Mary Hospital.

YIP YP, Kenneth M.B.,B.S.,(HK), M.R.C.P.(1983)*
M.O. 1980-84
Current Practice : Private Practitioner
Address : 12 Wu Pak Street, G/F Aberdeen, Hong Kong.

YU YH, Victor M.D., B.S. MSc(Oxon), M.R.C.P.(1973)*, F.R.A.C.P., F.R.C.P.(Lond)
Assistant Lecturer 1969-72
Current Practice : Director of Neonatal Intensive Care
Address : Queen Victoria Medical Center, 172 Lonsdale St., Melbourne Victoria 3000, Australia.

YUE CK, Paul M.B.,B.S.(HK), F.R.C.S., F.R.C.S.(Edin), F.R.A.C.S., F.A.C.S.
M.O. 1962-63
Current Practice : Private Practitioner
Address : 712-3 Melbourne Plaza, 23 Queen's Road C., Hong Kong.

YUEN MP, Patrick M.D.(Sask), F.R.C.P.(C)
Lecturer 1974-80; Senior Lecturer 1980-84
Current Practice : Senior Lecturer
Address : Department of Paediatrics, Prince of Wales Hospital, Shatin, N.T.

* Higher degree obtained while in the Department of Paediatrics, University of Hong Kong

As far as possible, the above information has been confirmed; the editors apologise for any possible errors.

Present staff

Medical Staff

CHAN SC M.B.,B.S.
M.O. 1982

CHENG MY M.B.,B.S.(HK), M.R.C.P.
S.M.O. 1982
Subspecialty : Haematology, Oncology, Developmental Paediatrics

CHEUNG PT M.B.,B.S.(HK), M.R.C.P.(1985)*
M.O. 1982-86; Lecturer 1986
Subspecialty : Endocrinology

CHISHTY A. Laeeq M.B.,B.S.(Punjab)
Clinical Fellow 1987

CHOI L L.M.C.(HK)
M.O. 1987

CHOW KH M.B.,B.S.(HK)
M.O. 1985

CHOW KW, Olivia M.B.,B.S.(HK), M.R.C.P.(1979)*
M.O. 1976-77; Lecturer 1977
Subspecialty : Respirology

FAN YS, Susan M.B.,B.S.(HK)
M.O. 1987

GOH HS, Winnie M.B.,B.S.(Sing.) M. Med. (Paed.) (1987)*
M.O. 1985

HUI KW M.B.,B.S.(HK)
M.O. 1987

KWAN YW, Elaine M.B.,B.S.(HK)
M.O. 1987

KWOK TL, Osmond M.B.,B.S.(HK)
M.O. 1983

KWONG NS M.B.,B.S.(HK)
M.O. 1987

LAM CC, Barbara M.B.,B.S.(HK), M.R.C.P.(1985)*
M.O. 1982-85; Lecturer 1985
Subspecialty : Intensive Care

LAU KC M.B.,B.S.(HK), M.R.C.P., F.R.C.P.(Edin.1986)
S.M.O. 1980-82; Senior Lecturer 1982
Subspecialty : Cardiology

LEE CW M.B.,B.S.(HK)
M.O. 1987

LEUNG P, Maurice M.B.,B.S.(HK), M.R.C.P.(1982)*
Lecturer 1980
Subspecialty : Cardiology

LI CH M.B.,B.S.(HK), M.R.C.P.(1986)*
M.O. 1982
Subspecialty : Infectious Diseases

LI MC, Anita M.B.,B.S(HK), M.R.C.P.(1969)*, F.R.C.P.(Edin,1979)*
Senior Lecturer 1984
Subspecialty . Haematology, Oncology & Nutrition

LO NS, Roxy M.B.,B.S.(HK), M.R.C.P.(1981)*
M.O. 1976-78; Lecturer 1978
Subspecialty : Cardiology

LOW CK, Louis B. Sc.(Hons.), M.B., Ch.B.(Glas.) M.R.C.P.
Lecturer 1982-85; Senior Lecturer 1985
Subspecialty : Endocrinology

MA PK, Danny M.B.,B.S.(Sydney)
M.O. 1986

NG TL M.B.,B.S.(HK)
M.O. 1986

ONG OL, Catherine M.B.,B.S.(HK)
M.O. 1987

SAMUDA Garythe B.Sc.(Otago), M.B.,Ch.B.(Otago), F.R.A.C.P.
M.O. 1982-83; S.M.O. 1983
Subspecialty : Oncology, Community Paediatrics

SU CW, Robin M.B.,B.S.(Lond)
M.O. 1987

TAM KH M.B.,B.S.(HK)
M.O. 1985

TAM YC, Alfred M.B.,B.S.(HK), M.R.C.P.(1982)*
M.O. 1982-84; Lecturer 1984
Subspecialty: Intensive care

TANG PS M.B.,B.S.(HK)
M.O. 1985

TANG TS, Allen M.B.,B.S.(HK)
M.O. 1985

TSE KW M.B.,B.S.(HK)
M.O. 1984

TSOI NS M.B.,B.S.(HK), M.R.C.P.(1985)*
M.O. 1981
Subspecialty : Intensive care

WONG CN, Virginia M.B.,B.S.(HK), M.R.C.P.(1984)*
Lecturer 1980
Subspecialty: Neurology & Developmental Paediatrics

WONG SN M.B.,B.S.(HK), M.R.C.P.(1984)*
M.O. 1981
Subspecialty : Intensive care

WONG YT L.M.C.(HK)
M.O. 1987

YEUNG CY M.B.,B.S.(HK), F.R.C.P.(Edin.,Glas.& Can.) (Hon.) C.R.C.P.(C)
Professor 1980
Subspecialty: Neonatology

YU CL, Edwin M.B.,B.S.(HK), M.R.C.P.(1980)*
M.O. 1976-84; Lecturer 1984
Subspecialty: Nephrology

YUNG TC M.B.,B.S.(HK)
M.O. 1987

Honorary Lecturers

CHAN CW	1981	M.B.,B.S.(HK), F.R.C.P.(Edin.& Ire.)
CHAU ASH	1974	M.B.,B.S.(HK), F.R.C.P.(Edin.)
CHENG MY	1983	M.B.,B.S.(HK), M.R.C.P.
FUNG HP, Robert	1968	M.D.,C.M.(McGill), F.R.C.P.(C)
IP MH, Henrietta	1978	O.B.E., J.P., M.B.,Ch.B.(Liv.), M.R.C.P.

KO YY, Lillian	1976	M.B.,B.S.(HK), F.R.C.P.(Edin)
LAI YF, Pansy	1981	M.B.,B.S.(HK), M.P.H. (Calif.)
LEE TY, Thomas	1974	M.B.,B.S.(HK), M.R.C.P., Dip. Occ. Med.(CUHK)
LEUNG NK	1976	M.B.,B.S.(HK), F.R.C.P.(Edin)
SAMUDA GM	1983	B.Sc., M.B.,Ch.B.(Otago), F.R.A.C.P.
TAM SY, Anita	1981	M.B.,B.S.(HK), M.R.C.P.
TSAO YC	1971	M.B.,B.S.(HK), F.R.C.P.(Edin)
WAI KH, Gregory	1978	M.B.,B.S.(HK), F.R.C.P.(C)

Office Staff

CHENG K, Lenny
Senior Personal Secretary
KO-SIU YP, Rebecca
Personal Secretary I
MOK YK, Donna — Secretary (GH)
CHEUNG, Mona — Stenographer (CAC)
CHUNG MC, Fanny — Clerk II
LI-TSANG LH, Juanna — Typist
LO YC, Alice — Typist
CHEUNG, Christina
Clerical Assistant (CAC)
CHEUNG, Martha
Clerical Assistant (CAC)
LIU YW, Kelly
Clerical Assistant (GH)
CHUI, Rebecca
Office Assistant (CAC)
CHUI KY — Office Assistant
NG BM — Office Assistant
SO-LAU SN — Workman

Postgraduate Students (M.Phil.)

HO KS
WONG HW, Francis

Laboratory Technical Staff

LEE FT
Senior Laboratory Superintendent
WONG HN
Laborattory Superintendent
CHOW NT, Lorna
Medical Technologist (TYH)
LEUNG CS
Senior Technician
LEE CY, Norman
Senior Technician
CHEUNG KF, Ronald
Technician (TYH)
HA KY, Philip — Technician (TYH)
HUI HM, Bonnie — Technician
HUI WK — Technician (TYH)
LUK YK — Technician
SUNG WK — Technician
WAN SK, Thomas — Technician
CHONG KK — Laboratory Assistant
SIU PS — Laboratory Assistant
WONG HS, Wilfred
Laboratory Assistant
LIU LC — Laboratory Attendent (TYH)
WONG CM — Laboratory Attendent
CHAN YL — Workman (TYH)
NG-CHAN KH — Workman

Other Health Professionals

CHAN, Betty
Occupational Therapist (CAC)
CHAN, Karen
Speech Therapist (CAC)
CHAN, Vivian — Physiotherapist (CAC)
NG, Richard
Clinical Psychologist (CAC)
YIM, Candy
Medical Social Worker (CAC)
NG, Shirley — Sister (CAC)
NG SL — R/N (CAC)
YUEN CM, Anders
R/N, Electrodiagnosis
LEE YW, Sharfa — E/N, QMH Clinic
PUN KP — E/N (CAC)

VISITING PROFESSORS AND CONSULTANTS

VISITOR	YEAR	PURPOSE
Dr. H. Miller Consultant Paediatrician, University of New Castle, New-Castle-Upon-Tyne, U.K.	1965	Academic visitor To advise on child development studies.
Prof. P.E. Polani Professor of Paediatric Research, Guy's Hospital, Medical School, University of London, U.K.	1972	IUC Visitor To advise on teaching & research in genetics.
Prof. I.C. Lewis Professor of Child Health, Department of Child Health, University of Tasmania, Australia.	1973	Honorary Visiting Professor To teach students.
Dr. T.P. Eddy Senior Lecturer, London School of Hygiene & Tropical Medicine, U.K.	1975	IUC Visitor To advise on human nutrition and preventive medicine to Departments of Community Medicine & Paediatrics.
Prof. J.H. Hutchison Professor of Paediatrics Royal Hospital for Sick Children, University of Glasgow , U.K.	1977	IUC Visitor To advise on general development in the Paediatrics Department.
Prof. M.A. Ferguson-Smith Professor of Medical Genetics, University of Glasgow, Glasgow, U.K.	1978	IUC Visitor To advise on cytogenetic service.

Prof. J.C. Sinclair Professor of Pediatrics, McMaster University, Ontario, Canada.	1982	Honorary Visiting Professor To teach & advise on research activities.
Dr. A. Duncan Director, Intensive Care Unit, Royal Children's Hospital, Melbourne, Australia.	1983	Honorary Lecturer To participate in & teach intensive care medicine.
Prof. R. Tsang Professor of Pediatrics, Obstetrics & Gynecology, Director, Division of Neonatology, University of Cincinnati, U.S.A.	1983	Wyeth Visiting Professor To teach neonatology and hold open scientific meetings.
Prof. O.H. Wolff Nuffield Professor of Child Health, Institute of Child Health, London, U.K.	1983	Croucher Visitor To advise on academic & service activities of the department.
Prof. R.J. Robinson Professor of Paediatrics, Guy's Hospital, University of London, U.K.	1984	CICHE Visitor To advise on developmental paediatrics & neurology programme.
Prof. A. Zipursky Director, Division of Haematology/ Oncology, The Hospital for Sick Children, Ontario, Canada.	1984	Wyeth Visiting Professor To teach haematology and to hold open scientific meetings.
Prof. G.H. McCracken Professor of Pediatrics, The University of Texas, Health Science Center at Dallas Southwestern, Medical School, U.S.A.	1985	Wyeth Visiting Professor To teach infectious disease and to hold open scientific meetings.
Dr. K. Brown Consultant Paediatric Neurologist, Hospital for Sick Children, Edinburgh, U.K.	1986	Honorary Senior Lecturer To teach child neurology.
Prof. R.M. Freedom Professor of Paediatrics (Cardiology), The Hospital for Sick Children, Toronto, Canada.	1986	Wyeth Visiting Professor To teach cardiology and to hold open scientific meetings.

Prof. M.M. Grumback Edward B. Shaw Professor of Pediatrics & Chairman of Dept., University of California, San Francisco, U.S.A.	1986	Visiting Professor To teach endocrinology and to hold open scientific meetings.
Prof. W. Oh Professor of Medical Sciences in Pediatrics & Obstetrics, Brown University Program in Medicine, Pediatrician-in-chief, Women & Infants Hospital of Rhode Island, U.S.A.	1986	Wyeth Visiting Professor To teach neonatology and to hold open scientific meetings.
Dr. R.E. Day Consultant Paediatrician, Royal Hospital for Sick Children, Glasgow, U.K.	1987	Honorary Senior Lecturer To participate in and teach developmental paediatrics.

This list is comprised of only those invited distinguished visitors who have spend varying periods working in the department.
We also acknowledge with thanks the great many distinguished visitors who passed through Hong Kong and accepted our invitation to deliver lectures in the department.

EXTERNAL EXAMINERS

Prof. V.L. Collins Professor of Child Health, University of Melbourne, Australia.	1965
Sir A. Moncrieff Nuffield Professor of Child Health, Institute of Child Health, University of London, U.K.	1966
Prof. W. Gaisford Professor of Child Health, St. Mary's Hospital, Manchester, U.K.	1967
Prof. D. Hubble Professor of Paediatrics, University of Birmingham, U.K.	1968

Prof. O.H. Wolff 1969
Nuffield Professor of Child Health,
University of London, U.K.

Prof. J.P.M. Tizard 1970
Professor of Child Health,
Institute of Child Health,
Hammersmith Hospital, U.K.

Prof. J.A. Davis 1971
Professor of Child Health,
St. Mary's Hospital,
Manchester, U.K.

Prof. W.B. MacDonald 1972
Professor of Child Health,
University of Western Australia,
Australia.

Prof. H.B. Wong 1973
Professor of Paediatrics,
University of Singapore,
Singapore.

Prof. R.G. Mitchell 1974
Professor of Child Health,
University of Dundee, U.K.

Prof. G.M. Maxwell, 1975
Professor of Paediatrics,
University of Adelaide,
Australia.

Prof. J.H. Hutchison, 1976
Samson Gemmell Chair of Child Health,
University of Glasgow, U.K.

Prof. J.H. Hutchison, 1977
Samson Gemmell Chair of Child Health,
University of Glasgow, U.K.

Prof. O.P. Gray, 1978
Professor of Child Health,
University Hospital of Wales,
Cardiff, U.K.

Prof. R.W. Smithells, 1979
Professor of Child Health,
University of Leeds, U.K.

Prof. G. Arneil, (Licentiate Examination), 1979
Leonard Gow Lecturer,
University of Glasgow, U.K.

Prof. J.O. Forfar 1980
Professor of Child Health,
University of Edinburgh, U.K.

Prof. V. Dubowitz 1981
Professor of Child Health
Hammersmith Hospital,
University of London, U.K.

Prof. D. Hull, 1982
Professor of Child Health,
University of Nottingham, U.K.

Prof. J.A. Davis, (Licentiate Examination), 1982
Professor of Child Health,
Addenbrooke's Hospital,
Cambridge, U.K.

Prof. A.L. Clark, 1983
Professor of Paediatrics,
Monash University,
Melbourne, Australia.

Prof. F. Cockburn, 1984
Professor of Child Health,
Royal Hospital for Sick Children,
Glasgow, U.K.

Prof. C Chantler, 1985
Professor of Paediatric Nephrology,
Guy's Hospital,
University of London, U.K.

Prof. J. Lloyd, (Licentiate Examination), 1985
Nuffield Professor,
Institute of Child Health,
University of London, U.K.

Prof. D. Barltrop, 1986
Professor of Child Health,
Charing Cross & Westminster Medical School,
University of London, U.K.

Prof. J. Lloyd, 1987
Nuffield Professor of Child Health,
Great Ormond Street Children's Hospital,
University of London, U.K.

Publications

1962

Field CE. The development of paediatrics in Singapore. J Singapore Paediatr Soc 1962;3(1):1-12.

Field CE. The changing pattern of disease in children — an inaugural lecture from the chair of paediatrics, University of Hong Kong Supplement to the Gazette 1962;10(2):1-5.

1965

Tsao YC, Fung YT. A comparative study of the Mantoux, Heaf and Tine skin tests for tuberculosis. Far East Med J 1965;1(6):216-220.

Yue PCK, Park JM. Medulloblastoma simulating tuberculous meningitis. J Singapore Paediatr Soc 1965;7(1).

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Programme of the Silver Jubilee Celebration

SILVER JUBILEE BANQUET **September 12, 1987**

SCIENTIFIC PROGRAMME **September 13, 1987**

Neonatology Update

(A programme of the Centennial Conference, Faculty of Medicine, University of Hong Kong)

1. Non-Invasive Investigation of Newborns with Congenital Heart Disease	K.C. Lau
2. Neonatal Hypocalcaemia and Rickets of Prematurity	Reginald C. Tsang
3. Late Outcome of Extremely Low Birth Weight Infants	Victor Y.H. Yu
4. Bilirubin Metabolism in Chinese Newborn Infants	C.Y. Yeung

Paediatric Forum

Keynote Lectures

1. The Maternal and Child Health Model Counties in China	Carl E. Taylor
2. The Infant of the Diabetic Mother	Reginald C. Tsang
3. Recent Advances in the Prenatal Diagnosis of Biochemical Disorders	Paul W.K. Wong

Free Paper Presentations

Posters

FREE PAPERS — ORAL PRESENTATION

A1 Observation of the Blood Sugar Pattern and Hypoglycaemia in Neonates
X.J. Guan, G.Z. Li, S.J. Yu.

A2 Neonatal Septicaemia — A Five Year Review
A.Y.C. Tam, E.Y.W. Kwan, R.W.H. Yung.

A3 Impact of a Neonatal Intensive Care Programme on Neonatal Mortality of Very-Low-Birth-Weight Babies
P.T. Cheung, C.Y. Yeung, A.Y.C. Tam.

A4 Developmental Changes of Thyroid Function in Infants Born Extremely Preterm
V.Y.H. Yu, M. Mercadeo.

A5 Necrotizing Enterocolitis in Neonates with Symptomatic Congenital Heart Disease
M.P. Leung, K.T. Chau, P.W. Hui, A.Y.C. Tam, F.L. Chan, C.Y. Yeung.

A6 Congenital Heart Disease in the Neonate
R.N.S. Lo, K.C. Lau, M.P. Leung, C.Y. Yeung.

A7 Seizure Susceptibility and the Neurochemical Changes in the Genetic Seizure-Prone Versus Seizure-Resistant Rat's Brain
X.R. Wu, P.S. Lin, Y. Hua, A.H. Zhang, Q.H. Zuo.

A8 Early Prognostication of Locomotion in Cerebral Palsy
M.J. Watt, C.M.T. Robertson.

A9 Neonatal Behavioural Assessment of Chinese Babies in Hong Kong
L.Y. Ko-Yang, P.K.B. Miu-Lee.

B1 Experience of Clinical Genetic Service in Hong Kong
A.S. Chau.

B2 Approaches in Genetic Metabolic Diseases — Local Experience
S.T.S. Lam.

B3 Treatment of HBsAg Associated Membranous Nephropathy
E.C.L. Yu, C.B. Chow.

B4 Anaemia in Hong Kong Adolescents
A.M.C. Li, M.Y. Cheng, A.M.C. Yu.

B5 Bone Marrow Transplantation (BMT) in Paediatric Patients
K.W. Chan, P.C.J. Rogers, S.L. Pritchard, C.J.H. Fryer.

B6 A Follow-Up Study on 140 Cases of Histiocytosis X
Y.M. Hu, S.Y. Yang.

B7 Coumadin Therapy in Children
M.Y. Cheng, V. Blanchette.

B8 Physical Conditioning Programme for Asthmatic Children — its Effect on Cardiorespiratory Function
O.K.W. Chow, R.N.S. Lo, M.H.H. Ip.

C1 Local Experience of Hepatitis B Vaccination in Newborns of HBsAg Carrier Mothers — the Optimal Dose Schedule and Timing
B.W.Y. Young.

C2 The Effect of Recombinant 2 Interferon in Chinese HBsAg Carrier Children
C.L. Lai, A.S.F. Lok, H.J. Lin, E.K. Yeoh, C.Y. Yeung.

C3 Endemic Giardiasis in a Hong Kong Nursery
G.M. Samuda, C.Y. Yeung, F.T. Lee.

C4 Nosocomial Gastroenteritis in Paediatric Patients
B. Lam, J. Tam, M.H. Ng, C.Y. Yeung.

C5 Factors Affecting Fatty Acids Absorption from Infant Formulae
G.M. Kneebone.

C6 Acute Viral Hepatitis in Children
T.T.Y. Lau, C.B. Chow, N.K. Leung.

C7 Childhood Tuberculosis in Hong Kong in the 1980s
M.K. Sham, M.J. Humphries, M. Gabriel.

C8 Parallel Defect in Type I IGF and Insulin Receptors in Two Patients with Acanthosis Nigricans (AN) and Insulin Resistance Type A
L.C.K. Low, M.A. Sperling.

C9 Prevalence and Significance of Mild Bleeding Disorders In Children with Recurrent Epistaxis
K. H. Luke

FREE PAPERS — POSTERS

P1 Congenital Mesoblastic Nephroma — A Benign Tumour That Usually Does Not Require Chemotherapy
M.Y. Cheng, H.S.L. Chan.

P2 A Review of the Clinical Presentation of 111 Children with Acute Lymphoblastic Leukaemia in Hong Kong
M.Y. Cheng, A.M.C. Li, G.M. Samuda.

P3 A 3 1/2 Year Old with Wilson's Disease–The Youngest Recorded in the English Literature
M.Y. Cheng, J.M. Seidman.

P4 Typhoid Fever in Hong Kong Children
C.B. Chow, P.S. Wang, N.K. Leung.

P5 The Prevalence of Small-For-Gestational-Age (SGA) Infant in South China. Part I: The Incidence
Z.K. Feng.

P6 The Relationship Between Platelet Function, Vitamin E, Vascular Reactivity in Diabetic Children
G.M. Kneebone.

P7 Mongolian Spot in Chinese Children
A.K.C. Leung.

P8 Double-Blind, Randomized, Multi-Centre Trial on the Efficacy and Safety of Two Oral Solutions as Maintenance Therapy of Acute Diarrhea
A.K.C. Leung, L. Geoffroy, P.G. Taylor, P. Darling.

P9 Echocardiographic Assessment of Neonates with Pulmonary Atresia and an Intact Ventricular Septum (PA + IVS)
M.P. Leung, P.W. Hui, C.K. Mok, E. Tam, K.C. Lau, R.N.S. Lo.

P10 Patterns of Chromosomal Abnormalities in Hong Kong
A.M.C. Li, F.T. Lee.

P11 Childhood Cancer: Its Impact on the Families (Local Report)
A.M.C. Li, T.L. Que, G.S.C. Chui, G.M. Samuda.

P12 The Pattern of Paediatric Bacterial Meningitis in Queen Mary Hospital
C.H. Li.

P13 The Trend of Postinfectious Nephritis
C.H. Li, E.C.L. Yu.

P14 Oral Rehydration Therapy Using Rice Powder — Electrolyte Solution
G.M. Samuda, B. Lam, C.Y. Yeung.

P15 Arterial Blood Pressure Measurement in Critically Ill Preterm Neonates
A.Y.C. Tam.

P16 Neonatal Leukocyte Function Studies
A.Y.C. Tam, S.K. Wan, C.Y. Yeung.

P17 Lead Poisoning in Infancy
K.H. Tam, E.C.L. Yu.

P18 Fallot's Tetralogy: 10 Years' Experience in Hong Kong
P.S. Tang, K.C. Lau, C.K. Mok, R.N.S. Lo, M.P. Leung.

P19 Haemorrhagic Shock Encephalopathy in Chinese Children
T.S. Tang, A.Y.C. Tam.

P20 Retrospective Survey of Neonatal Infections in Tsan Yuk Hospital
N.S. Tsoi, C.Y. Yeung.

P21 Myasthenia Gravis in Chinese Children
V. Wong, B.R. Hawkins, Y.L. Yu, C.Y. Yeung.

P22 Acute Viral Encephalitis in Children
V. Wong, C.Y. Yeung.

P23 Ataxia Telangiectasia in Chinese Children–A Clinical and Electrophysiological Study
V. Wong, Y.L. Yu, W.Y. Chan-Lui, E. Woo, C.Y. Yeung.

P24 Effect of Serum pH on Bilirubin-Protein Binding
C.Y. Yeung, F.T. Lee, H.N. Wong.

P25 Intra-Uterine Growth Promoting Effect of a Chinese Herb
C.Y. Yeung, C.S. Leung, F.T. Lee.

P26 Renal Size Measured by Ultrasonography in Children
E.C.L. Yu, E.M.T. Chau, L.L.Y. Leong, K.P. Fung.

SILVER JUBILEE

Department of Paediatrics
University of Hong Kong

September 12-13, 1987


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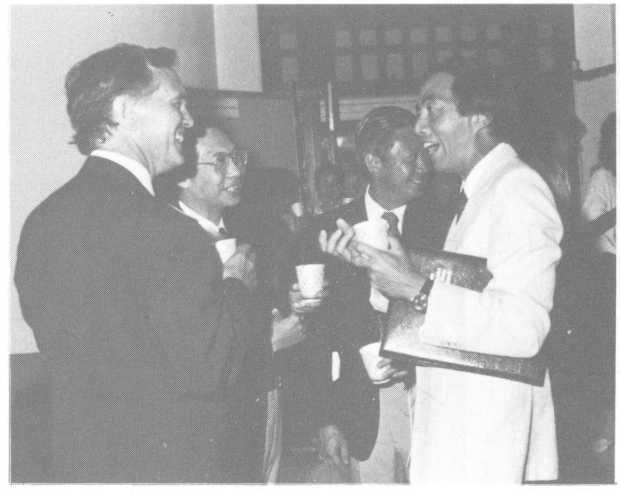

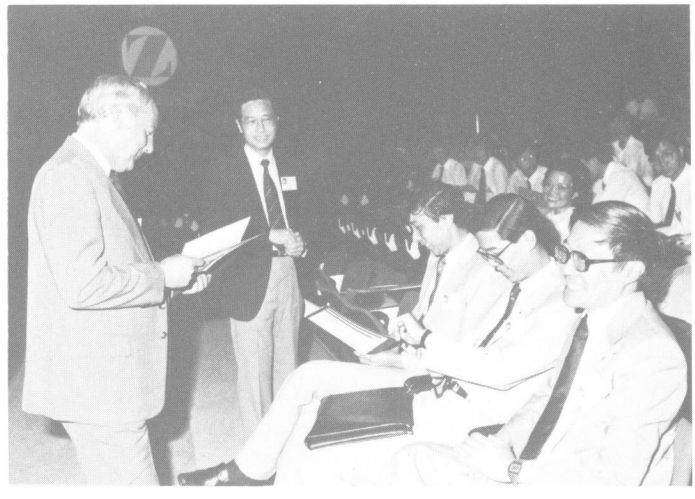

INVESTIGATION OF NEWBORNS WITH SUSPECTED CONGENITAL HEART DISEASE BY NON-INVASIVE METHODS

KC LAU, CK MOK[1], MP LEUNG, RNS LO, CY YEUNG
Department of Paediatrics and Surgery[1],
University of Hong Kong.

When the newborn with congenital heart disease becomes symptomatic the defect is severe and the prognosis is usually poor. If left untreated, most would die within the neonatal period. These patients are critically ill. Any unnecessary intervention or investigation will unfavourably influence their outcome. Although cardiac catheterization and angiography is an important investigatory method for patients with heart diseases it carries a considerable risk when the patient is a fragile newborn. With the introduction of cross-sectional echocardiography (CSE) and pulse doppler echocardiography (PDE), many varieties of congenital heart lesions can be diagnosed with a high degree of accuracy. By using cardiac catheterization or post-mortem examination as the diagnostic reference we have demonstrated a high diagnostic sensitivity (95.5%) and a high specificity (98%) with combined CSE and PDE in a group of 96 newborns suspected to have significant congenital heart disease [1]. Our results suggested that cardiac catheterization, an investigation not without risk in the sick newborns, may be replaced by CSE and PDE as a preoperative imaging method. We have therefore undertaken another study to find the actual outcome of using non-invasive tests alone as the definitive investigatory tool.

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PATIENTS AND METHODS

From August, 1983 to March, 1985, 110 consecutive newborns were admitted to the paediatric cardiac unit; 53 were females and 57 were males. They were admitted because of cyanosis (43%), heart failure or cardiogenic shock (51%), cardiac arrhythmia or conduction defects (2%), or other physical signs suggestive of significant heart diseases (eg. upper limb hypertension and absence of lower limb pulses) (4%). Every case was included and each had a complete history taken, physical examination, chest radiography, electrocardiogram, blood gas sampling and other laboratory tests as indicated (eg. sepsis work-up, etc.) A systematic CSE and PDE examination was performed with precordial long axis and short axis left and right ventricular cuts, scanning from the base to the apex of the heart. The apical four-chamber cut was then performed followed by the subcostal four chamber, short axis and long axis scans. The great arteries were evaluated by employing the suprasternal views [2]. The anatomical diagnosis was worked out segmentally and sequentially [3]. Then haemodynamic status and valvular functions were studied by PDE. When the diagnosis was still not conclusive, cardiac catheterization was performed to establish the diagnosis. The patients were grouped according to the results of non-invasive tests.

Group 1 consisted of patients in whom adequate anatomical and haemodynamic data could be obtained confidently from non-invasive evaluations alone, enabling formulation of a management plan. Management would thus be executed without further catheterization even when surgery was one of the modes of treatment.

Group 2 consisted of patients in whom the diagnosis obtained was either incomplete or inconclusive such that catheterization was deemed necessary to establish the diagnosis.

Group 3 consisted of patients in whom the diagnosis was confidently established after non-invasive tests. The diagnosis was subsequently proven to be inaccurate either by cardiac catheterization which would be carried out if the clinical course deviated from expected, or from surgical or post-mortem findings.

The modes of management include :-

(i) Surgical:-
   a) Total repair, eg. total anomalous pulmonary venous connection, etc.
   b) Limitation of pulmonary flow with or without repair of coarctation of aorta or ligation of ductus arteriosus.
   c) Enhancement of pulmonary flow by systemic-pulmonary shunting.
   d) Valvotomy: aortic or pulmonary; with or without cardio-pulmonary bypass.

(ii) Non-surgical:-
   a) Interventional cardiac catheterization: eg. atrial septostomy in transposition of great arteries, pacing, etc.
   b) Medical: antifailure medications, conservative treatment, etc.

TABLE 1. Case with adequate non-invasive studies.

	Total no.	Diagnosis confident and accurate	Percentage
TGA	18	17	94
HLH Sx	12	12	100
PA+IVS	13	8	62
Coarctation	8	6	75
TAPVD	7	4	57
PDA(isolated)	6	5	83
PA=VSD	4	2	50
Fallot's tetrology	6	6	100
Situs solitus + UVH	5	3	60
Ebstein's anomaly	4	4	100
VSD (isolated)	4	4	100
Tricuspid atresia	3	3	100
Hemitruncus	2	1	50
AV defect	2	2	100
NSH disease	6	6	100
MS (supravalvar membrane)	1	1	100
Cardiac tumour	1	1	100
	102	85	

TGA = transposition of great arteries; HLH SX = hypoplastic left heart syndrome; PA+IVS =pulmonry atresia and intact ventricular septum; TAPVD= total anomalous pulmonary venous drainage; PDA = persistent ductus arteriosus; UVH = univentricular heart; VSD = ventricular septal defect; AV = atrioventricular; NSH = non-structural heart disease; MS = mitral stenosis; PA+VSD = pulmonary atresia and ventricular septal defect.

RESULTS

Group 1. 86 patients (80%)

The cardiac anomalies in these patient are summarised in Table 1. Anomalies allowing 100% diagnostic sensitivity and specificity were:
hypoplastic left heart syndrome (n=12),
Fallot's tetralogy (n=6),
Ebstein's anomaly (n=4),
isolated ventricular septal defect (n=4),
tricuspid atresia (n=3),
atrioventricular septal defect (n=2),
cardiac tumour (n=1),
supramitral valvular membrane (n=1).

As a result, all of the 6 patients with "non-structural heart disease" were diagnosed correctly by non-invasive methods but one with infra-diaphragmatic type total anomalous pulmonary connection was initially misdiagnosed as non-structural heart disease. In 17 of 18 cases with transposition of great artery, the diagnosis was made accurately. In the eighteenth, a small associated ventricular septal defect was not detected. This however did not alter the management regime for this patient.

Group 2. 20 patients (18%)

Table 2 summarises the anomalies in this group. The majority of this group had ambiguous situs which were associated with total anomalous pulmonary venous drainage (n=8). When the pulmonary flow was diminished in the presence of severe pulmonary obstruction, the pulmonary veins were small and their drainage sites could not be imaged with certainty. All required cardiac catheterization to determine the site of pulmonary venous drainage. 2 of the 6 (33%) patients with isolated pulmonary venous connection and situs solitus had severe pulmonary venous obstruction, severe pulmonary hypertension and low pulmonary flow. The anomaly, though suspected on clinical grounds, could not be diagnosed with certainty by CSE and PDE. Cardiac catheterization confirmed obstructive total anomalous pulmonary venous drainage. 2 of the 4 (50%) newborn with pulmonary atresia and ventricular septal defect had extremely dimunitive pulmonary arteries. Angiography was necessary to demonstrate them and the collateral vessels before any shunting operation could be performed. 2 of the 8 patients (25%) with coarctation of aorta required catheterization before surgery because of inconclusive echo findings.

Group 3. In 5 patients (4.5%) an incorrect anatomical diagnosis was made initially (Table 3). In 2 patients with pulmonary atresia and intact ventricular septum, significant myocardial sinusoids running

TABLE 3. Incorrect Diagnosis

PA+IVS	2 (myocardial sinusoids not seen)
PDA+TR	1 (PDA missed)
TAPVD	1 (diagnosed as PFC)
TGA	1 (a small VSD missed)

Abbreviations refer to Table 1.

TABLE 2. Cases with inconclusive diagnosis.

	Total no.	Uncertain diagnosis	Percentage
Situs ambiguus (? PV drainage)	8	8	100
TAPVD	7	2	28.5
Coarctation	8	2	25
PA+IVS	13	3	23
PA+VSD	4	2	50
Situs solitus+ UVH	5	2	40
Hemitruncus	2	1	50
	47	20	18.5

PV = pulmonary vein; others refer to Table 1

across the right ventricular outflow tract were not imaged by CSE but were found during surgery. In one baby with tricuspid regurgitation and a large ductus arteriosus, the ductus was missed during the initial CSE and PDE examination. His heart failure was resistant to conservative treatment and a subsequent catheterization revealed the true diagnosis. One patient had total anomalous pulmonary venous connection and pulmonary venous obstruction. Initially he was thought to have persistent foetal circulation. He did not improve on conservative treatment for 2 days and the diagnosis was made by cardiac catheterization. He succumbed after an attempted surgical repair.

38 (35%) required surgery within the first month of life (Table 4). 31 (79%) had surgery without prior catheterization while only 8 (21%) needed catheterization and angiography to confirm or to establish the diagnosis before surgery.

DISCUSSION

In this study 77.5% of newborns admitted because of suspected heart diseases were judiciously managed without cardiac catheterization. 18% still required invasive tests, mainly to identify the pulmonary veins and arteries when they were narrowed and the blood flow in these vessels was sluggish. With better resolution instruments and improved Doppler technology, this group of patients may be expected to diminish.

Newborns with myocardial failure from any cause tolerate stresses like cardiac catheterization poorly. If cardiac catheterization could be avoided surgical results might be improved. In fact, our surgical mortality for neonatal coarctation of aorta did drop from a previous rate of 35% to 20% during the present period ($p < 0.05$). The reasons leading to this improvement are multiple, and cannot be attributed

TABLE 4. Surgery in the newborns.

	Surgery without catheterization	with catheterization
IAA repair	4	0
Coarctation repair +/– PA banding	3	1
PDA ligation	5	1
TAPVD repair	4	2
Hemitruncus	1	0
Systemic-pulmonary shunt	5	1
Closed pulmonary valvotomy	8	3
Mustard's operation	1	0
	31 (79%)	8 (21%)
Other Interventions:		
Atrial balloon septostomy under echo control:–	19	

Abbreviations refer to Table 1.

solely to our present policy. Apparently improvement of surgical techniques is the major factor but undoubtedly a contributory one is the presentation to the surgeons of patients in better shape. Our results strongly support the policy of avoiding catheterization and using echocardiographic imaging as the sole preoperative diagnostic method in newborns suspected to be suffering from congenital cardiac lesions. Even if the effect of employing non-invasive tests alone on final surgical statistics is only minimal, such a practice should continue and be encouraged. Not only is the morbidity of catheterization avoided, surgical or other therapeutic regimes can be significantly speeded up. Additionally this is certainly the most cost-effective way of treating patients with heart diseases.

To subject the sick newborn to surgery with a wrong diagnosis is the most important hazard of this policy. In 2 of the 4 cases of misdiagnosis, the surgeons were able to make appropriate changes in two. In one case of infradiaphragmatic type of total anomalous pulmonary venous connection, surgical correction was delayed. This was one of the factors contributing to his dismal outcome. In the fourth case, the delayed diagnosis of a large ductus arteriosus did not lead to any morbidity or mortality. Thus the misdiagnosis rate was very low which should, by no means, discourage further application of this policy. After all, in only one case was the outcome of the patient adversely affected. The advantages certainly outweigh the disadvantages. Additionally, one can predict that mis-diagnosis will soon be rare and may disappear.

In conclusion, our study confirms the feasibility and safety of using non-invasive tests as the definitive preoperative diagnostic methods in newborns with suspected heart disease. Only in a minority is cardiac catheterization required, mainly in evaluating the great artery anomalies or the sites of pulmonary venous drainage. Misdiagnosis is acceptably infrequent and, perhaps, will eventually disappear.

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DIABETES IN PREGNANCY: METABOLIC CONTROL AT SPECIFIC PREGNANCY PERIODS

F. MIMOUNI R.C. TSANG
The Department of Paediatrics, Division of Neonatology,
Univeristy of Cincinnati, College of Medicine,
Cincinnati, Ohio, U.S.A.

INTRODUCTION

In the diabetic pregnancy there are very few studies that examine the impact of management at specific times during pregnancy on outcome. It is instructive to view the impact of management at different stages of pregnancy, since the influences in mother or fetus theoretically would be different at each stage.

PERIOD		COMPLICATION
1	Periconception	Abortions
2	First Trimester	Malformations
3	Second Trimester	Preeclampsia
		Premature labor
4	Third Trimester	Neonatal Macrosomia
		Hypoglycemia
5.	Labor	Perinatal Asphyxia
		Neonatal Hypoglycemia

CONTROL IN THE PERICONCEPTIONAL PERIOD

Periconceptional adverse influences theoretically may precipitate embryonic losses[1]. We demonstrated that diabetic women are at a significantly greater risk for early fetal losses/abortions than the general population[2-3]. Diabetic women with abortions had higher glycosylated hemoglobin contents at 8-9 weeks of gestation than those without abortions. However glycosylated protein concentrations were similar between groups[4]. Glycosylated total protein content reflects control during the previous 1-2 weeks before measurment, whereas, glycosylated hemoglobin reflects control in the previous 4-8 weeks[4]. Therefore it appeared that poor glucose control in the periconceptional period, rather than just before the abortion increases the risk of early fetal losses.

CONTROL IN THE FIRST TRIMESTER

The major fetal organs develop predominantly in the first 9 weeks of gestation[1]. Major congenital malformations appear to correlate with elevated maternal blood hemoglobin A1C concentrations determined prior to the 14th week of gestation[5]. These findings support the thesis that first trimester glycemic control may be a significant factor in the risk for congenital malformations.

CONTROL IN THE SECOND TRIMESTER

We reported that preeclampsia, and premature labor occur at a high rate in insulin dependent diabetic women. These complications generally occur by the end of the second trimester or early third trimester[6,7]. We therefore hypothesized that these complications may correlate with poor second trimester

glycemic control. Pregnancy associated hypertension correlated significantly with poor late first trimester and second trimester glycemic control[6]. The mean first trimester mean maternal blood glucose concentration was significantly elevated in women who developed hypertension; hemoglobin A1 also was significantly elevated by 14 weeks and at 20 weeks.

Factors predisposing to premature labor and delivery in the diabetic pregnancy have been poorly studied and the relationship of diabetic control and prematurity has not been examined prospectively. Earlier studies of prematurity in the diabetic pregnancy could not separate out the influence of "iatrogenic" prematurity, since previous management techniques involved deliberate early delivery of fetus based on the assumption of higher fetal loss in infants carried to term. Recent management of diabetic pregnancy involves a more "tolerant" approach towards non-preterm delivery, which provides a new opportunity to examine the spontaneous prematurity rate. In a study of diabetic pregnancies, we determined that the rate of spontaneous premature labor was 31.1%, which was more than 3 times the rates for the general population (7-10%)[7]. Factors found to be significantly associated with premature labor were premature rupture of membrane, previous history of premature delivery, urogenital infection, and poor glycemic control during the second trimester of pregnancy as assessed by maternal hemoglobin A1 concentrations at 21 weeks gestation. Surprisingly, polyhydramnios was not significantly associated with premature labor. From these 2 studies it appears that strict glycemic control at the end of the first and during the second trimester will decrease the rates of pregnancy associated hypertension, and of spontaneous premature labor[4].

CONTROL IN THE THIRD TRIMESTER

It has been suggested that fetal macrosomia in diabetic pregnancy is related to fetal hyperinsulinisim[8-10]. Beta-cell hyperplasia may be observed in fetuses of diabetic mothers between the 16th[10] and the 34th week of gestation[11], but not earlier. On theoretic grounds therefore it would appear that first and second trimester glycemic control probably do not affect insulin-related macrosomia; however poor third trimester glycemic control theorectically may stimulate hyperinsulinism and its two major consequences: macrosomia and neonatal hypoglycemia.

In our study, of infants of diabetic mothers[12], 43% were large for gestational age (LGA). However, maternal blood hemoglobin A1 at but not in the first 2 trimesters, was significantly higher in the LGA compared to the appropriate weight (AGA) group. Statistically analyses showed that hemoglobin A1 measurement at delivery was an even stronger predictor of macrosomia than White class.

In another study reporting insulin dependent diabetic pregnancies[13], the rate of neonatal hypoglycemia (serum glucose 30 mg/dl in the first 4 hours of life) was significantly related to third trimester glycemic control. There was a higher maternal blood hemoglobin A1 at delivery in infants who develped neonatal hypoglycemia compared with infants of diabetic mothers who did not develop neonatal hypoglycemia.

From these two studies it appears that neonatal macrosomia and hypoglycemia, two major indicators of neonatal hyperinsulinism, are associated with poor trimester glycemic control.

GLYCEMIC CONTROL IN LABOR

A hyperglycemic environment during the few hours preceding delivery might theoretically stimulate future fetal hyperinsulinemia and secondarily cause neonatal hypoglycemia[14]. In a study of insulin-dependent diabetic pregnancies[15], maternal serum glucose concentration in labor was targetted to be maintained between 70-100 mg/dl by infusion of glucose and/or insulin. The lowest infant

plasma glucose concentration in the first 4 hours of life correlated with the highest maternal capillary glucose concentration within 4 hours before delivery. A maximum maternal blood glucose concentration above 90 mg/dl during the last 4 hours before delivery resulted in a 47% rate of neonatal hypoglycemia; in mothers with blood glucose less than 90 mg/dl, their infants had a 14% rate of hypoglycemia. It appears that elevation of maternal blood glucose during labor will lead to increased insulin release and subsequent neonatal hypoglycemia.

In the diabetic pregnancy reduced uteroplacental blood flow correlates with acutely elevated maternal blood glucose concentration[16]. In a study of diabetic pregnancies, we found that 26% of the infants of diabetic mothers developed perinatal asphyxia; perinatal asphyxia was significantly correlated with maternal hyperglycemia prior to delivery. A blood glucose concentration of 150 mg/dl appeared to be the cut-off point for increased risk of perinatal asphyxia: 48% of mothers who had infants with perinatal asphyxia had at least one blood glucose value above 150 mg/dl compared to 25% of mothers of the infants without asphyxia. Perinatal asphyxia was not associated with maternal hypoglycemia. From this study it appears possible that hyperglycemia within the 6 hours prior to delivery may lead to fetal hypoxemia by decreasing uteroplacental blood flow.

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LATE OUTCOME OF EXTREMELY LOW BIRTHWEIGHT INFANTS

V.Y.H. YU
Department of Paediatrics, Monash University,
Melbourne, Victoria, Australia

Both mortality and morbidity of preterm births have improved with modern perinatal care. Extremely low birthweight (ELBW, <1000 g) infants continue to have relatively high mortality and incidence of increased neurodevelopmental and continuing medical morbidity. Although less than 1% of all births are ELBW, they are responsible for one-third perinatal deaths. Twelve published ELBW series are reviewed. Reported survival rates ranged from 12-67% and impariment rates from 13-35% (cerebral palsy: median 12%, range 4-25%, blindness: median 3%, range 0-25%; sensorineural deafness: median 2%, range 0-5% developmental delay: median 15%, range 3-35%). Problems involved in the interpretation and comparison of published data are analysed. A review of the literature for survival and disability data reported by week of gestation at birth up to 28 weeks indicates that there are only three such reports. This information is nevertheless important to the obstetrician who has to make critical decisions on the care of the mother and fetus in the presence of extremely preterm labour. A review is also made on studies which identified perinatal risk factors associated with death or impairment in ELBW infants. Lastly, lesser but continuing morbidity in the first 2 years, such as health problems that require medical or surgical treatment, rehospitalisation patterns, suboptimal physical growth and behavioural problems, are described. Based on these data, we can begin to answer the philosophical ethical, legal and economical questions raised in the provision of perinatal intensive care to such infants, many of whom were previously considered nonviable.

In the State of Victoria, Australia, which had 61176 births in 1985, 326 (0.4%) of all births were extremely low birthweight (ELBW, <1000 g). However, these ELBW births were responsible for 34% of stillbirths and 36% of neonatal deaths (Table 1). The overall perinatal mortality rate, for all infants born weighing 500 g or more and including deaths up to 28 days of birth, was 12.1 per 1000 births. The perinatal mortality rate reported for ELBW infants was 66 times that figure. Consequently, when infants born below 1000 g were excluded from the statistics and only deaths up to 7 days of birth were included (World Health Organisation definition used for international comparison) the perinatal mortality rate for Victoria in 1985 was reduced to 6.9 (stillbirth rate 4.3, neonatal death rate 2.6). Not only do ELBW infants have a high mortality, the neurodevelopmental and continuing medical morbidity rates among ELBW survivors remain relatively high.

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TABLE I. Birthweight and perinatal mortality rate

Birthweight (g)	All births	Stillbirths	Neonatal deaths	Perinatal Mortality rate
500-999	326 (0.5%)	136 (34%)	124 (36%)	797.6
1000-1499	336 (0.5%)	50 (13%)	57 (17%)	318.5
1500-1999	662 (1.1%)	40 (10%)	28 (8%)	102.7
2000-2499	2059 (3.4%)	45 (11%)	24 (7%)	33.5
>2500	57717 (94.3%)	114 (29%)	102 (30%)	3.7
Not known	76 (0.1%)	13 (3%)	8 (2%)	276.3
Total	61176	398	343	12.1

HOSPITAL-BASED STUDIES

Twelve published studies[1-12] on the survival and impairment rates of ELBW infants are summarised in Table 2. Although it is tempting to compare the outcome between hospitals and trends over the past two decades, there are many problems involved in the interpretation and comparison of published data.

The most obvious discrepancy between the reports is the proportion of the study population who were outborn. One study had an exclusively inborn population[10] and another an exclusively outborn population.[4] Except for two study populations from our own hospital with a 16% outborn group,[9,12] the remaining reports had 43-82% of the study population who were outborn. Not all ELBW births outside major tertiary centres with neonatal intensive care facilities are referred for treatment. Therefore, a profound selection bias is known to exist for outborn infants. Since the survival and impairment rates of inborn and outborn ELBW infants are significantly different, it is mandatory to report outcome for those born with a perinatal centre separate from those outborn and referred in for neonatal intensive care.

ELBW cohorts, by virtue of the fact that subjects were weight-selected, might include varying proportions of infants who are relatively mature but small-for-gestational age. Except for two study populations from our own hospital which had a 7% incidence of fetal growth retardation,[9,12] the other published studies had 25-40% of infants with a birthweight below the tenth percentile for gestational age. It is therefore impossible to interpret differences in outcome of these ELBW cohorts which do not have comparable gestations.

Two studies[4,12] clearly indicated that livebirths who died in the delivery room, which account for up to one-third of ELBW deaths, were included in the statistics. The remaining reports referred to infants who had been admitted to the neonatal intensive care unit, which might suggest that livebirths who died prior to admission had been excluded. The neonatal (28-day) mortality rate was reported in most studies. However, longer term studies have shown that there was a significant number of post-neonatal deaths before or after hospital discharge during infancy.[13] From our own inborn data, the survival rate of ELBW infants would have increased from 47% to 57% if delivery room deaths had been

excluded and to 62% if postneonatal deaths had also been ignored.

Differences in the definition for impairment or disability and the age at follow-up of the survivors probably accounted for the wide range (13-35%) of neurodevelopmental impairment reported among ELBW survivors (Table 2). It had been reported that over half the children diagnosed to have cerebral palsy at 1 year did not have a motor deficit on longer follow-up. The pooling of data from children of widely different ages, small numbers of survivors, failure to separate inborn and outborn survivors, and a high attrition rate are some of the major criticisms of published studies. Children lost to follow-up are likely to have a higher impairment rate than those who return for assessment.

The term impairment commonly refers to adverse neuro-developmental outcome which includes cerebral palsy of all types of severity, blindness, sensorineural deafness and developmental delay defined as a score on psychometric testing which exceeds two standard deviations below the mean for the normal population. The literature review referred to in Table 2 shows that the median incidence of cerebral palsy was 12% (range 4-25%), blindess 3% (0-25%), sensorineural deafness 2% (0-5%) and developmental delay 15% (3-35%). The term disability is a measure of interference with function due to the presence of the above impairments. The criteria for majority disability involves value judgements but between half to two-thirds of ELBW children with an impairment were diagnosed to have a major disability.

TABLE 2. Literature review on ELBW infants

Reference	Year of birth	No. of infants	% survival	No. survivors followed up	% abnormal
1	1965-70	161	12%	20	35%
2	1968-72	98	28%	27	30%
3	1966-75	148	26%	27	22%
4	1974	97	47%	44	23%
5	1973-76	69	35%	23	35%
6	1674-76	100	23%	16	25%
7	1976-78	134	30%	35	26%
8	1977-78	134	30%	38	26%
9	1977-80	107	55%	59	25%
10	1979-80	106	67%	69	13%
11	1980	56	52%	29	28%
12	1977-83	261	46%	108	29%

REGIONAL-BASED STUDIES

The efficacy of perinatal/neonatal intensive care programmes in improving outcome for ELBW infants within the entire population can only be ascertained in regional-based studies. Comparison of a regional cohort of ELBW livebirths in 1973-76 with that in 1977-80 has shown that their survival has doubled without any increase in the incidence of impairment among survivors.[14] We have also published a geographically- defined ELBW study based on all ELBW livebirths during 1979-80 in the State of Victoria.[15-17] Our findings showed that the survival rate of ELBW infants born in perinatal centres within the State was significantly higher than those born elsewhere. Furthermore, the rate of severe and moderate functional disability in outborn survivors was over three times that of inborn survivors. Suboptimal care prior to the arrival of the Newborn Emergency Transport Service, which was identified in over 70% of the outborn survivors, probably accounted for the significantly worse quality of survival.

The importance of place of birth on perinatal outcome is also shown in Table 3 which summarised the data for 1294 ELBW infants born in the State of Victoria in the years 1982-85. It shows that 63% of ELBW births were born in tertiary units where about a quarter of all livebirths within the State were delivered. Their perinatal mortality and stillbirth rates for inborn infants were significantly lower than those born outside perinatal centres. Significantly more outborn infants were not offered resuscitation at birth and only 35% of outborn infants were subsequently referred for neonatal intensive care. Consequently the neonatal survival rate of outborn ELBW infant was significantly lower than those born in the more optimal environment of a perinatal centre.

TABLE 3. Place of birth and perinatal outcome

	Perinatal centres	Born elsewhere
No. of births	816 (63%)	478 (37%)
Perinatal mortality rate/1000 births	718	931
No. of stillbirths	292 (51%)	284 (49%)
Stillbirth rate/1000 births	358	594
No. of live births	524 (73%)	194 (27%)
No. of resuscitated	389 (74%)	90 (46%)
No. of referred to perinatal centre	---	68 (35%)
Neonatal survival rate		
Overall	230 (44%)	35 (18%)
Referred	---	33 (51%)
Not referred	---	2 (2%)

EXTREMELY PRETERM INFANTS

Two perinatal centres besides ourselves have published their experience on survival and impairment rates by week of gestation in large inborn cohorts born at 24-28 weeks (Table 4).[18-20] The results are relatively comparable except in one where the low survival rate in those less than 27 weeks was likely to be due to the fact that 27% of their deaths occurred after a decision was made not to offer ventilator care because of extreme prematurity.[19] Of the 460 extremely preterm survivors from the 3 studies, 84 (18%) were reported to have an impairment. Obstetrical decision-making in the management of extremely preterm labour has become increasingly complex with advances in perinatal intensive care. Every perinatal centre providing this type of care is encouraged to develop its own outcome predictions for use in its own clinical service.

PERINATAL RISK FACTORS

The three perinatal centres which have reported outcome according to gestation have also reported their findings on perinatal factors associated with death or impairment in ELBW or extremely preterm infants.[19,21,22] Factors associated with increased mortality included antepartum haemorrhage, multiple pregnancy, breech presentation, delivery without preceding labour, absence of antenatal steroid therapy, male sex, perinatal asphyxia, hypothermia, hypotension, hyaline membrane disease, persistent pulmonary hypertension, severe respiratory failure, infection, intraventricular haemorrhage and delayed onset of diuresis. Factors associated with increased impairment included antepartum haemorrhage, absence of antenatal steroid therapy, male sex, severe respiratory failure, intraventricular haemorrhage and delay in regaining birthweight. Stepwise multiple discriminant function analysis has shown that intraventricular haemorrhage was the most important predictor of death.[22] The introduction of routine cerebral ultrasonography has enabled accurate diagnosis of intracerebral haemorrhage and periventricular leukomalacia which we have shown to correlate with increased mortality and impairment.[23,24]

Other studies have reported perinatal events associated with increased risk of impairment which included bronchopulmonary dysplasia,[25] abnormal neurodevelopmental examination at term,[26] prolonged initial hospitalisation,[27] poor postnatal head growth,[28] and postnatal growth failure.[29] The optimal route of delivery for ELBW infants remains controversial.[30] We found no evidence to support the use of caesarean section in extremely preterm infants with vertex presentation, except for

TABLE 4. Survival and impairment rates of extremely preterm infants

Year of birth	Reference 18 1979-82	Reference 19 1977-82	Reference 20 1977-84
Survival rate by gestation			
24 weeks	9/23 (39%)	2/27 (7%)	13/40 (33%)
25 weeks	28/44 (64%)	11/54 (20%)	11/44 (25%)
26 weeks	34/45 (76%)	36/80 (45%)	36/62 (58%)
27 weeks	45/60 (75%)	47/67 (70%)	63/87 (72%)
28 weeks	71/88 (81%)	76/98 (78%)	70/95 (74%)
No. of survivors with impairment	33/158 (21%)	15/111 (14%)	35/190 (18%)

recognised maternal or fetal indications. Our findings in the non-vertex group of a lower impairment rate in caesarean births compared to vaginal births indicated that there is a definite need for a randomised clinical trial to investigate the possible benefits of caesarean delivery in extremely preterm breech infants.[31]

CONTINUING MEDICAL MORBIDITY

The broader spectrum of lesser morbidity in ELBW survivors which includes continuing health problems requiring medial or surgical intervention, rehospitalisation, suboptimal growth and behavioural problems, has rarely been documented. In a series of 119 ELBW survivors, we have found that 61% required rehospitalisation in the first 2 years of age, with respiratory tract disorders and surgical procedures accounting for most admissions (Table 5). This compared with a rehospitalisation rate of 18% for children of normal birthweight in Melbourne, thus giving a relative risk of 3.4 in ELBW children. Otitis media (55%), wheezing episodes (48%) and lower respiratory tract infection (29%) were the three most common medical disorders in ELBW children. This compares with prevalence rates of 45%, 8% and 16% respectively for normal birthweight children in Melbourne. Temper tantrums (45%), colic (19%) and excessive vomiting (12%) were found in ELBW children compared to prevalence

TABLE 5. Continuing morbidity in ELBW survivors

	500-799g (n=36)	800-999g (n=83)
No. of children rehospitalized	21 (58%)	51 (61%)
Mean no. of rehospitalization	2.3	2.5
Medical disorders		
Otitis media	24 (66%)	42 (51%) *
Wheezing	15 (43%)	42 (51%)
Lower respiratory tract infection	8 (22%)	24 (29%)
Gastroenteritis	11 (31%)	20 (24%)
Surgical procedures	15 (42%)	28 (34%)
Aural ventilation tubes	8 (22%)	4 (5%) *
Inguinal herniorrhaphy	3 (8%)	8 (10%)
Miscellaneous	4 (12%)	16 (19%)
Children below 10th percentile		
Weight	17 (47%)	35 (43%)
Height	16 (44%)	37 (46%)
Head circumference	4 (11%)	10 (13%)

* $p < 0.05$

rates of 16%, 22% and 2% respectively for normal birthweight children in Melbourne. By 2 years corrected age, 44% remained below the tenth percentile for weight, 45% below the tenth percentile for height and 12% below the tenth percentile for head circumference. It was nevertheless encouraging that, except for otitis media and the increased rate of aural ventilation tube insertion, survivors of 500-799g birthweight did not have significantly higher rate of rehospitalisation, health problems, suboptimal growth or behavioural problems, compared to those of 800-999 g birthweight.

CONCLUSION

Because of the enormous financial and human resources as well as psychoemotional burden required to provide neonatal intensive care to ELBW infants and their increased mortality, impairment and continued morbidity rates, there is persistent questioning and doubt concerning such treatment. In the face of limited medical resources, the ethical issues involved in treating these ELBW infants need to be continually reviewed.[32-34] The poor reproductive history of the maternal population from which ELBW infants are derived and the unfavourable subsequent obstetric experience of these mothers[35] emphasise the importance of research towards prevention of extreme prematurity. Based on the review data in this paper, we can begin to answer the philosophical, moral, legal and economical questions raised in the provision of perinatal intensive care to such infants, many of whom were previously considered non-viable.

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BILIRUBIN METABOLISM IN CHINESE NEWBORN INFANTS

C.Y. YEUNG
Department of Paediatrics,
University of Hong Kong

INTRODUCTION

The serum bilirubin in the newborn infant comes from three sources, namely the breakdown of red blood cells, ineffective erythropoeisis and the tissue haem[1,2]. Red blood cell breakdown normally constitutes 75% of the total bilirubin generated in the infant. In the adult, however, 95% of the bilirubin is derived from red blood cells, and tissue haem plays a much less important role. As bilirubin is not water soluble, it is transported in the circulation in miscible form bound to serum proteins, mainly albumin. The bilirubin of this albumin-bilirubin complex is selectively taken up by the liver which is the main organ for its excretion.

Within the hepatocyte, a new protein complex is formed with the ligands, Y and Z proteins. These transport proteins nullify the cytotoxic effects of bilirubin and enhance its transfer to the microsomes where conjugation to bilirubin-glucuronides takes place, catalysed by glucuronyl-transferase. These glucuronides are water soluble and are excreted via the bile-duct canaliculi into the intestine.

Normally the bilirubin glucuronides are rapidly reduced by the bacteria present in the intestine to stercobilinogen and stercobilin which are excreted in the faeces. In the newborn, the relative scarcity of bacteria allows the bilirubin glucuronides to come under the action of ß-glucuronidase enzyme present in the upper gastro-intestinal tract. The glucuronides are freed and the original bilirubin molecules regenerated. The bilirubin is absorbed via the fat absorption pathway by the lymphatics to the liver. This entero-hepatic re-circulation of bilirubin is unique in the newborn and is believed to be an important factor in the physiologic jaundice[3] in these infants.

NEONATAL HYPERBILIRUBINAEMIA

The following three main mechanisms may contribute to hyperbilirubinaemia in the newborn; (1) increased production of bilirubin, (2) impaired liver function and (3) increased entero-hepatic re-circulation.

(1) Increased Production of Bilirubin

(A) Increased red cell breakdown

(a) Rhesus iso-immunization

Rhesus iso-immunization is a classical disorder of excessive red blood cell breakdown resulting in hydrops foetalis, severe jaundice, anaemia and often deaths in the newborn. The disease is not a major problem in Southern Chinese as 99.9% of the population are Rh+.[4] Among the Caucasians the condition has also been declining since the introduction of routine immunoglobulin therapy in early 1970s.[5]

(b) ABO incompatibility

ABO incompatibility is the commonest associated condition of neonatal hyper-bilirubinaemia identified in the Southern Chinese infants.[6,7] However overt features of haemolysis is present in only about 1/3 of the infants with severe jaundice[7]. The classical infant appears relatively normal at birth but develops jaundice within the first 24 hours. Among those who show signs of haemolysis, it is not uncommon to find normal

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haematologic results at birth although their mothers may demonstrate high anti-A or anti-B titres. Hydrops foetalis is rarely due to ABO disease. The post-natal haemolytic phenomenon appears to be triggered by events occurring around parturition. Sepsis and maternal-foetal exchanges of circulation during labour appear to be contributing factors.[8]

(c) Erythrocyte G6PD deficiency

Erythroctye glucose-6-phosphate dehydrogenase (G6PD) deficiency occurs in 4.42% of the Chinese male infants.[9] This condition predisposes the infants to a 4-fold increased risk[10] of hyperbilirubinaemia necessitating hospital care. The most acute and severe form of jaundice is also encountered in the G6PD deficient babies.[7,10] The typical infant boy is normal at birth and discharged from the hospital in healthy and good condition. He is brought to medical attention a few days (1-21 days) later because of sudden severe jaundice, with or without associated features of encephalopathy. Detailed history often reveals an antecedent event such as exposure to naphthalene moth balls (blanket or clothings), taking herbal tea especically Chuen Lin, or signs of infection.

Although homozygous deficiency state is much less common in girls, severe hyperbilirubinaemia in the early neonatal period is not a rare occurrence. Recent enzymatic survey has shown that the frequency of phenotypic expression of severe enzyme deficiency in girls is 0.45%.[7,9] In theory therefore, one should expect to see the sex ratio of female to male with severe bilirubinaemia to be 1:10. In practice, however, the incidence is much higher,[10] suggesting that many heterozygous deficient females manifest the clinical problems also.

The defective X-linked gene appears to protect the red blood cells against infection by malaria. This explains its high prevalence in Southern Chinese and its rarity among the Northerners; just like the Mediterranean people are affected and not the other Caucasians in Northern Europe.

(d) ∝-Thalassaemia

∝-Thalassaemia is the commonest cause of hydrops foetalis[11] in our population. This occurs in the homozygous infant who presents with severe hydrops, profound anaemia and jaundice at birth. He either dies in-utero or shortly after birth. There are occasional infants who are not hydropic but are born with anaemia and rapidly increasing jaundice requiring exchange transfusions within the first day or two. They demonstrate Haemoglobin Bart's and H granules in their peripheral blood like the hydropic infants, but their clinical course resembles Thalassaemia intermedia as they grow up.

(e) Concealed haemorrhage

Bruises and cephalhaematoma are well known causes for hyperbilirubinaemia due to excessive breakdown of red blood cells in extravasated sites. In Chinese, there is a relatively high frequency of sub-aponeurotic haemorrhages occurring even in infants not delivered by vaccuum extraction (personal observation). Jaundice is a common sequalae.

(B) Tissue heme breakdown

Hypoxia is a potent stimulus for haem-oxygenase activities enhancing the breakdown of tissue haem and other haem products. Thus infants born with severe birth asphyxia and those preterm infants developing severe hyaline membrane disease often develop intense jaundice.

(2) Impaired liver function

(A) Non-specific neonatal hyperbilirubinaemia

Studies conducted in our department have indicated that healthy infants develop hyperbilirubinaemia [8, 12, 13] which differs from the classical physiologic jaundice [14] in three areas. Firstly the jaundice is much more intense, secondly it peaks 1 day later and thirdly it lasts much longer. No evidence of haemolysis is demonstrated in these infants, and the problem appears to be due to impaired liver function. Earlier work has shown that such impairment of liver function could be corrected readily by enhancement with phenobarbitone[6,12,15]. By 24 hours of therapy a significant beneficial effect has been observed, in the excretion of bilirubin[6,12] and BSP[15]. This form of therapy was first introduced to Hong Kong in 1968[6] and had resulted in a significant reduction of the need for exchange transfusions for the ensuing few years[12], before phototherapy became more widely available.

Some workers[16] have suggested that this kind of hyperbilirubinaemia in Chinese is due to an ethnic or genetic immaturity of the liver. An observation was made in 1969[7](table 1) that there was a significantly different frequency of hyperbilirubinaemia between the Chinese infants born in two government hospitals of Hong Kong. The hospital admitting mothers from a much lower socio-economic stratum showed a significantly higher incidence of severe neonatal jaundice compared to the other hospital with parents coming from a more privileged background. Such observation indicated that severe neonatal jaundice was probably not due to ethnic but due to environmental reasons. Many factors were thought to be related; they were over-crowding, cross infections, over colonization, herbal teas, poor maternal nutrition, inadequate education and early immunization. It was predicted at the time[7] that as Hong Kong's economic and social programmes continued to improve, there would be progressive decline of severe neonatal jaundice also. On re-examining the jaundice problem in the recent few years,[8] it is most interesting to note a marked decrease of severe neonatal jaundice with gradual disappearance of kernicterus also. Marked social and environmental changes have occurred during the

TABLE I. PEAK SERUM BILIRUBIN IN CHINESE NEWBORN (FIRST WEEK OF LIFE)

Hospital	Infant Number	Bilirubin in mg/dl		
		>10	>15	>20
A1	45	25	12	3
A2	44	20	6	0
B	45	18	4	0

Socio-economic status : A << B
A1 & B with control infants;
A2 with phenobarbitone-treated infants*
Infants: healthy,>37 wk.>2.5 Kg, Apgars ≥8,
Bl Gr O, Rh D+, G6PD+, no bruises (Pediat 1971; 48:372)

past 1 1/2 decade. These have included proper housing for over 2 1/2 million under privileged people, compulsory education up to age 15, increase of individual earning, and improvement in health facilities etc. Hong Kong has now become the third largest financial centre in the world, and people's life-style has become more sophisticated. The prediction on neonatal jaundice has come true.

(B) Immaturity of liver

This is a unique problem of the infants born too early and it also plays a role in the genesis of jaundice in the infants of the diabetic mothers. Chinese infants are just as prone to develop this problem as other non-Chinese. Insufficient Y and Z proteins and immature glucuronyl transferase activities are responsible. Enzyme induction therapy with phenobarbitone does not improve the condition.[6]

(C) Infections

Infections of all kinds have been reported to be associated with neonatal jaundice partly due to increased haemolysis and partly due to suppression of liver function. In a preliminary study[17] we have shown a high frequency of early colonization by potentially pathogenic organisms which are known to suppress liver function. It is possible that increased colonization in our over-crowded environment is contributing to the genesis of jaundice in our infants also.

(3) Increased entero-hepatic recirculation

(A) Feeding related jaundice

Breast feeding has long been observed to be associated with neonatal hyperbilirubinemia. Although demonstration of maternal 3∝-20ß-prednanediol which suppresses liver function[4] has attracted most publicity as a cause for the jaundice, the majority of infants with 'breast-milk jaundice' is not due to the steroid. Inadequate fluid intake, reduced foecal bilirubin loss, and abundance of ß-glucuronidase enzyme in the breast milk have been shown to be important factors. These increase the entero-hepatic load of bilirubin. Indeed even bottle-fed infants receiving insufficient fluid develop similar jaundice[18] also.

(B) Intestinal stasis

Conditions resulting in ileus of the intestine tend to enhance entero-hepatic re-absorption of bilirubin causing jaundice. Pyloric obstruction is an example.

TABLE 2. HERB CONSUMPTION IN CHINESE NEWBORN

Herb	1972	1982
Chuen Lin 川蓮	50.9*	28*
Ngau Huang 牛黃	35.9	23.2
Wax Roses 腊梅花	30	14
Others	25	32
None	25	62.4
Infants	220	125

* Percentage mothers surveyed

THE PROBLEM WITH CHINESE HERBS

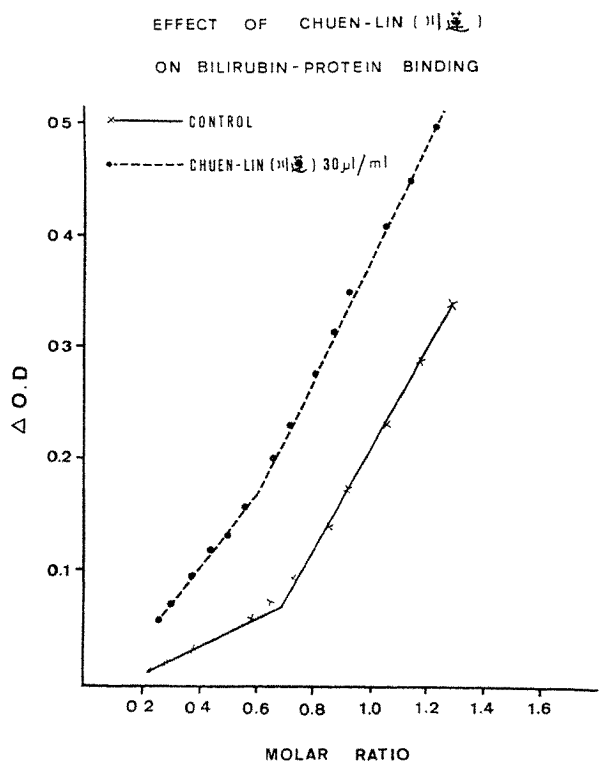
Herbal tea consumption is a popular tradition of Chinese. Pregnant mothers take herbs as tonics and to ensure the well-being of the pregnancy and to promote foetal growth. Herbs are given to the newborn infants to 'detoxify the toxins of pregnancy'. Surveys conducted in Hong Kong have shown a declining trend of herbal usage for the neonates over the past 15 years (table 2) but the rate is still high. During pregnancy, 12 Tai-Bau (十二太保) and 13 Tai-Bau (十三太保) are often taken as tonics, while Chuen Lin (川連), Wax Roses (腊梅花), Ngau Huang (牛黃) and Bau Ying Pills (保嬰丹) are most popular for the infants.[7,8,10]

Previous observations[7,8,10] on the association of herbs with severe neonatal jaundice and kernicterus were re-examined recently. It has been found[10] that apparently healthy infants who are G6PD deficient often develop sudden, severe hyperbilirubinaemia after receiving herbs, notably 'Chuen Lin'. Cases of kernicterus have also occurred.

As kernicterus is due to an excessive amount of unbound bilirubin[19], the effect of herbs on the binding of bilirubin to protein is also studied in our laboratory. The horseradish peroxidase oxidation method to assay 'free bilirubin' has been adopted. It has been found (figure) that a couple of herbs, especially Chuen Lin, are capable of displacing bilirubin from protein binding. An increased amount of free bilirubin is then generated, thus increasing the risk of brain damage and kernicterus (In preparation for publication).

With a more sophisticated animal model we have also obtained preliminary study results to indicate the deposition of bilirubin to brain tissue when an excessive amount of free bilirubin is present (In preparation for publication).

Herbalists and some Chinese doctors have claimed that many of these herbs have shown some clinical effects on neonatal jaundice. However, scientific documentation of these claims are lacking. Indeed a lesson[20] was learned by neonatologists in the late 1950s when gantrisin was used to prevent neonatal sepsis. It was found that



the gantrisin treated infants were less jaundiced during life but had a significantly higher frequency of kernicterus at death compared to the control infants or infants receiving other antibiotics.[20] It was this observation which has led to the discovery of the importance of free bilirubin producing damage to the cells[21] resulting in kernicterus.

The therapeutic application of herbs has continued in China for several thousand years. There must be some clinical effects in them if their use has been continued for so long. We have demonstrated, in our laboratory, that some of the commonly used herbs are potentially risky in jaundiced infants. We do not know which particular ingredients of the herbs are responsible. Many other clinical effects may also be present and even be beneficial. More research is needed to clarify these issues. Meanwhile, with the present available data showing potentially harmful effect, use of herbs in the neonatal period should be strongly discouraged.

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THE MATERNAL AND CHILD HEALTH MODEL COUNTIES IN CHINA

C.E. TAYLOR
Department of International Health,
The Johns Hopkins University
Maryland, U.S.A.

BACKGROUND

The improved system of health care in the People's Republic of China has become a model for other developing countries. The WHO/-UNICEF sponsored World Conference on Primary Health Care at Alma Ata in 1978 encouraged other countries to follow the model of China's services, especially the innovative mass use of barefoot doctors and health cooperatives. In 1979, as part of the new open policy under Deng Xiao Ping, China established the Economic Responsibility system which returned economic decision making to families. This has been very good for economic production with an approximate doubling of agricultural and industrial growth rates. The health cooperatives and bare foot doctor system, however, essentially collapsed. They had been based on communal financing through workpoints. Now that people could work for money and were assigned land for families to farm on contract, they naturally tended to concentrate on production. The tremendous effort required to maintain basic social services was diverted to economic activities to benefit families. It seemed a reasonable assumption that now that families could earn much more money they would be able to pay for services that had previously been provided by the commune.

The barefoot doctors responded to the new chances to make money along with everyone else. They took contracts for land and concentrated more intensively on farming and sideline ecomonic activities. Since they had been part-time farmers anyhow this might have made little difference, but the whole workpoint system had collapsed in most counties so there was no way to pay them for health work. Decisions about how health care would be financed were decentralized by Beijing and delegated to local units of government. In perhaps 90 percent of communes the decision was made that if farmers can have a responsibility system, then health workers should also be given that right. As a result most of the upgraded barefoot doctors opted for fee-for-service private practice. Perhaps ten percent of communes maintained the previous system of health cooperatives and had people pay premiums to maintain services under a health cooperative. In affluent areas.village doctor income has increased so that they frequently get more than party cadres or assistant professors at medical schools. Two adjustments had to be made.

Now that people were paying fees for each visit they began to demand better quality care. For a long time officials had been embarrassed by the term barefoot doctors and used to take time to explain to visitors that they really did wear shoes. With the need for new respectability, the barefoot term was abolished and they were now called village doctors. One requirement was that they had to take upgrading courses and pass credentialling exams. This was especially important for those who went into private practice, often as a group in the old village health post. The upgrading courses of 6 months to two years were held in county and township hospitals. About half of the 2 million barefoot doctors successfully passed the upgrading exams. There are now about 1.2 million village doctors, most of whom spend more time in health care than previously because it has become a good source of income and many are full-time.

The second adjustment was to learn how to accommodate the resulting shift from preventive to curative activities. With fee-for-service payments such a shift seems inevitable because people tend to pay only when they get sick. Even when services are free it may take considerable health education to get compliance with preventive activities. Especially in maternal and child health care it became evident that there was a critical need to improve preventive services. One factor that helped greatly was that following the one child per family policy the demand for total health care of children increased. Parents said if we can have only one child we want a perfect child, and they became very serious about following advice on preventive measures but they still did not want to pay for preventive services.

As the need for new approaches to MCH services were becoming evident the government and UNICEF cooperatively developed a project to develop model counties as a means of working out the necessary innovations in each part of the country.

METHODOLOGY

Over the past several years WHO has been promoting the concept of Health Development Networks as an ongoing methodology for local problem solving. A recent monograph (WHO 1986) outlines the rationale and proposed methodology for national and local linkages of "services, agencies and institutions competent in the areas of service development, training and research, in order to mobilize resources and coordinate activities such as management, planning, implementation, monitoring and manpower development to achieve "health for all". The China MCH Model Counties Project is an excellent example of such a network.

In 1982 the MCH Bureau of the Ministry of Public Health and UNICEF cooperatively started support for ten model counties in ten of the most populous provinces, each linked closely with the local medical school and school of public health. A series of one week visits and workshops were held in each county involving the Ministry of Public Health MCH Bureau, high level provincial health officials, faculty from pediatrics, obstetrics and public health departments, county health officials and UNICEF. From 50 to 100 people participated in a detailed situational analysis and preparation of a plan of action for three types of activity in the model county. The first emphasis was on improving services for children and their mothers using available knowledge, including provision of better facilities and equipment. The second was training for all levels of staff. The third was applied field research to address the remaining highest priority problems relating both to specific diseases and management.

The organizational structure included setting up a special office in the MCH bureau for coordination supported by a central advisory group. In each province there was a Leading Group to support the cooperative activities and ensure rapid extension of findings to other counties, made up of intersectoral representatives including the All-China Women's Federation. Each medical college formed a technical advisory group of faculty who had administrative responsibility, expertise and a committment to work in the county. Each county assigned special administrative staff and organized a county leading group headed by a top county official and including agencies such as the education department and the local women's federation chapter.

Within three years the general enthusiasm led to an extension of activities to include 34 counties and prefectures in 18 provinces and autonomous regions, including Tibet. The situational analyses in each of the original ten counties provided a basis for agreeing on national priorities, even though the solutions clearly had to be developed locally because conditions differed greatly in a country as large and diverse as China. Highest priority was given to immunization against six major childhood diseases (polio, measles, diphtheria, pertussis, tetanus and tuberculosis); control of acute respiratory infections which was far and away the first cause of childhood death in all parts of the country; diarrhea which was still a major cause of morbidity

even though mortality seems to have been largely controlled by the promotion of ORT using salt and sugar solution by barefoot doctors which started some 30 years ago (Taylor and Xu); perinatal mortality which was next to pneumonia as a cause of death in children; anemia found in about half of children in rural areas and in pregnant women; stunting found in over a third of children in the south; and rickets found in about a third of the children in the north.

In the initial workshops the basis of discussion was a matrix. It showed how for each of the areas of services, training and research it was necessary to go through the same five steps of analysis: definition of priority problems, definition of possible solutions, developing a local implementation plan, providing resources to proceed with implementation, and evaluation and feedback.

Uniform guidelines were provided for all units of the project. Annual conferences were held to bring together officials from all parts of the network. At the beginning great efforts were made to establish a uniform information system but this was only partially successful because of reluctance of provincial officials to give up their existing information systems. A more gradual transition is proving necessary.

IMPROVEMENT OF SERVICES

In each county a pilot area was first identified as the site where new ideas would be tried. This was usually one xiangs (township of 20,000 to 50,000 population). After much discussion it was agreed that it was more important to have easy access from the county headquarters than representativeness so these pilot areas tended to be somewhat better off than the rest of the county. The expanded county MCH staff took responsibility for helping develop the pilot activities themselves through visits several times a week. The thought was that by going through the experience of direct involvement themselves they would then be able to supervise expansion to the rest of the county. A strong tendency developed, however, for the county staff to become so involved and possessive of the pilot area they found it difficult to turn over responsibility to township staff after one to two years as planned. This interfered with the rapid extension of services to the rest of the county. Considerable pressure sometimes had to be exerted by central and provincial officials to facilitate this transition.

In each county a major investment was made in improving the county MCH facilities. The province invested large sums of money in building health centers which often ended up as special hospitals. A certain amount of competition developed between counties to see who could build the biggest buildings. In a formal evaluation after three years of field work a definite conclusion was that the counties with the least effective supervision of the total county services by the MCH center were where a significant number of inpatient beds had been added to the MCH center rather than using the existing county hospital. Major investment also went into building some county health schools which was certainly needed. UNICEF was requested to provide the most sophisticated technical equipment available internationally for those county facilities. When this was done there was complaints from some medical colleges that the county now had more elaborate equipment than the teaching hospitals. This tendency stopped only when it became evident that the foreign exchange necessary to provide consumable supplies and reagents was going to be completely impossible for county resources to maintain.

In the improvement of services faculty from medical school departments had an important advisory role. The clinical specialists helped especially in the county hospital. The various public health specialists helped reorganize specific services. The statistics department typically conducted the baseline survey in each of the original ten counties. Some of the data from those surveys will be presented in slides at this meeting. The diversity among counties was great with population size ranging from 200,000 to 1.2 million and IMR from about 25/1,000 to over 70/1,000, with IMR in Tibet estimated to be over 150/1,000.

An outside consultant conducted a

careful evaluation two years after the field work started. She worked intensively in the field in six of the ten original counties. Some key improvements identified included : 1) maternity care had been generally improved with better screening for high risk cases and improved referral. In two counties the proportion of home deliveries increased after village level services were improved because of convenience and lower cost. 2) the expanded program for immunization had been stressed even in counties that were not yet included in the national EPI program. Coverage was high in all counties except one where they were still using the previous pattern of two cycles a year of immunization campaigns rather than providing immunization as a continuing service. There was not, however, adequate attention to completing immunizations in the first year of life. BCG and tetanus toxoid for pregnant women is not yet included in the EPI program which is under the anti-epidemic bureau rather than the MCH bureau. 3) Pneumonia dominates in all counties as the first cause of death. In the first round of workshops detailed explanations were given of recent findings in other countries of the possible impact of better case management. Training in simple methods of diagnosis by village doctors using indicators such as rapid respiration was extended to all staff. In Rudong county in Jiangsu province preliminary reports indicated that following this training, in one year the infant mortality rates had fallen from 24/1,000 to 16/1,000, with the decrease being largely in the pneumonia deaths. Other services had also improved and some preliminary data will be presented in the slides.

IMPROVEMENT IN TRAINING

Permanent improvement in services depends largely on improved training of staff. Large numbers of training courses have been held for county, township and village personnel. Statistics and improvements in the information system have received special attention in all counties. Most of this training has been in short in-service courses run by medical school faculty. The next emphasis will be on longer courses. The main effort will be to improve the county health schools which have many deficiencies. Increasing numbers of doctors are being sent for one to two years training at the local medical school. This is an activity that will require much more emphasis. One aspect of training that is also just beginning to be implemented is the regular use of the model county for the training of medical students. Thus far they have been used mainly to assist in surveys but not with a clearly defined educational purpose.

APPLIED FIELD RESEARCH

As general services improved the project emphasis shifted to the problem areas where more knowledge is needed to make further improvements in health conditions. Project personnel are encouraged to conduct their own research projects on problems that are important locally. For the six priority health problems mentioned above and for the specific management problems of financing of village doctors there have been separate multicenter research projects set up in comparative trials in six to ten counties. Some general comments will first be made about each topic and then more detailed information will be presented with slides.

(1) ARI and pneumonia

In Ball counties at least a third of childhood deaths are caused by pneumonia. In some counties most of the childhood deaths are from lower respiratory infections. In 6 counties in various parts of the country two hypotheses are being tested in appropriate sample populations. First, is that child mortality can be rapidly lowered by better case management with provision for simplified diagnosis and prompt treatment by village doctors. Second, that a major cause of neonatal pneumonia is the ancient practice of swaddling babies after birth so they never get complete expansion of their lungs.

(2) Diarrhea

As indicated above mortality from diarrhea is low but morbidity high in comparison with other Asian countries. It is important to find out why. Present practices of oral rehydration will be studied to

determine how they can be improved. Local studies of diarrhea prevention will try to determine the influence of environmental and social factors. One thing is clear that a major effort will be needed to stop the overuse of powerful drugs in simple diarrhea.

(3) Perinatal mortality

Considerable improvement has occurred in methods of delivering babies in most areas, although there are some counties where neonatal tetanus still occurs. The greatest need is to improve prenatal monitoring of pregnancy to screen for high risk. The monitoring methods need to be adjusted to the varying competence of the peripheral staff responsible in various areas.

(4) Stunting

In South China, as in most rice eating areas, people are smaller and shorter than in the northern wheat eating areas. This is not genetic as indicated by the stature of elite groups in the south. It is probable that the rice eating physique is determined by the weaning food used in the first two years. Diluted rice porridge is just about the worst weaning food that could be invented. Preliminary calculations indicate that it would take 17 bowls of rice porridge to meet the caloric needs of an 8 months old child and 40 bowls to meet the protein requirements. The research will focus on the use of growth charts and defining appropriate weaning foods based on what is available in homes in each area and season.

(5) Anemia

Between a third and a half of all children and pregnant women in China are anemic. There should be enough iron in their diet according to analyses of the food eaten. One topic being studied is the possibility that blocking agents in the food may interfere with absorption of iron. In a largely vegetarian diet, there are a number of phytates and oxalates that may have this effect. Tea is the most efficient blocking agent in common use, causing a 50 percent reduction in absorption. Vitamin C causes a 50 percent increase.

(6) Rickets

About a third of the children in northern provinces and in the mountain areas have clinical rickets. Little milk is taken and this may lead to calcium deficiency. Supplementation with Vitamin D is either not done appropriately or does not seem to have the effect it should depending on the studies reported. One possible factor is the cultural tradition of totally bundling up a child in many layers of clothing during cold weather. The child gets no sunlight and on the streets they are even carried with a screen over their face.

(7) Financing for village doctors

For any of the plans for improvement in MCH services to work there will have to be some means of reimbursing village doctors for preventive work. In many counties they are expected to provide preventive services in exchange for the privilege of doing private practice for curative services. They cannot be expected to devote the attention required for improved services for the total population, especially in remote and poor areas, if their time is not compensated adequately. It is probable that the same solutions will not suit the varying needs of all parts of the country. Therefore appropriate local measures need to be developed which means that alternative means of financing will have to be tested, including various forms of health insurance.

CONCLUSION

The most encouraging feature of the model county project has been the rapidity with which innovations tested or demonstrated in special projects have spread to other counties. This process of moving from pilot projects to general implementation has been especially difficult in most efforts to improve health services. UNICEF calls this going to scale. The health development network may be an approach which can be tried more generally to get rapid implementation of improved methods in routine services

NEONATAL HYPOCALCEMIA AND RICKETS OF PREMATURITY

F. MIMOUNI R.C. TSANG
The Department of Pediatrics, Division of Neonatology,
University of Cincinnati, College of Medicine,
Cincinnati, Ohio, U.S.A.

NEONATAL HYPOCALCEMIA

(1) Definition

Neonatal hypocalcemia has variously been defined as a serum Ca concentration < 8 mg/100 ml, <7.5 mg/100 ml, or < 7 mg/100 ml. All these definitions are arbitrary, based on the total serum Ca and not on the ionized fraction that is biologically important. The ionized fraction is affected by 1) serum protein concentrations (in hypoproteinemia, decreased total Ca may be associated with relatively normal iCa) and 2) blood pH (acidosis decreases Ca binding to proteins, resulting in a higher ionized fraction). It seems therefore that hypocalcemia is best defined by a direct measurement of iCa. However, there is limited information regarding ionized Ca concentrations in newborn infants, and a distinction between hypocalcemia and normocalcemia based on iCa awaits further studies. In the experience of the authors of this article, a full term infant (with higher protein serum concentration than a preterm infant) should be regarded as hypocalcemic for practical purposes in the presence of a total serum Ca of < 8 mg/dl, and a preterm infant in the presence of a total serum Ca of < 7 mg/dl. In both preterm and full term infants, utilizing older ion selective electrodes, an iCa concentration under 3 to 3.5 mg/dl has been suggested as indicative of hypocalcemia. Using modern ion selective electrodes, these numbers may need to be revised upward, but insufficent data are available to be specific.

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(2) Causes of Neonatal Hypocalcemia

The causes of neonatal hypocalcemis are traditionally classified as "early" and "late" neonatal hypocalcemia. "Early" neonatal hypocalcemia typically occurs during the first 24 to 48 hours of age, while "late" neonatal hypocalcemia occurs towards the end of the first week of life. In some cases the clinical separation between early and late hypocalcemia may not be distinct.

(A) Early Neonatal Hypocalcemia

(a) Hypocalcemia of Prematurity

About 30% of premature infants develop neonatal hypocalcemia. Serum Ca values correlate directly, in the first few days of life, with gestational age [1,2] (Figure 1). The natural decrease of serum Ca after birth seems to be accentuated and prolonged in preterm infants due to decreased parathyroid function [2,3]. In spite of hypocalcemia, the normal surge of CT occurs also in preterm infants [4,5]; this surge may be, for un-clear reasons, apparently blocked with an oral Ca supplement [6], but does not ear to be affected when serum Ca is maintained by IV Ca ion [4,5]. The role of this CT surge in the pathogenesis of ypocalcemia in premature infants is not known. Hypocalcemia in premature infants may be aggravated by the presence of other risk factors such as decreased milk (and Ca) intake, birth asphyxia and correction of acidosis with sodium bicarbonate [7]. Decreased serum 25OHD concentration may

also co-exist with hypocalcemia in some instance [8], although its role in hypocalcemia is dubious. End-organ resistance to $1,25(OH)_2D$ may also exist in extremely preterm infants and theoretically could contribute to their hypocalcemia [9].

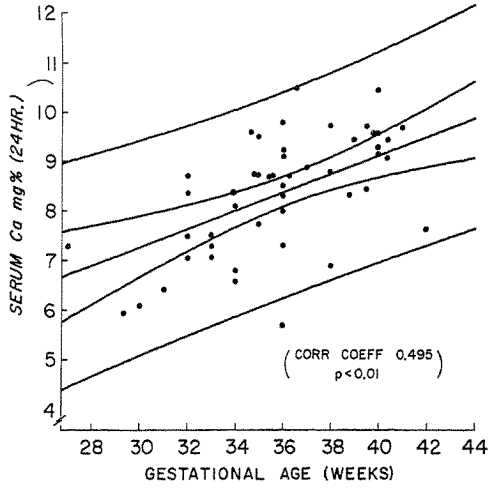

Figure 1.
Twenty-four hour serum Ca in relation to length of gestation. The graph shows the regression line for all infants with the 95 percent confidence limits for the regression line and the 95 percent confidence limits for the sample (from Tsang Rc, Light IJ, Sutherland JM, Kleiman LI, J Pediatr 1973; 82:423, reproduced with permission).

(b) Post-Asphyxiatic Hypocalcemia

Neonatal asphyxia leads to a high rate (30%) of hypocalcemia, even after controlling for the effects of prematurity [2,7,10]. Increased endogenous P loading and decreased Ca intake may play a significant role [7], as well as increased CT serum concentration observed recently in asphyctic infants [11]. Correction of acidosis with alkali may further aggravate hypocalcemia, theoretically by inducing a decrease of Ca flux from bone to extracellular space [7,10].

(c) Infants of Diabetic Mothers (IDM's)

Neonatal hypocalcemia may occur in 50% of IDM's. In a prospective matched controlled study, neonatal hypocalcemia in IDM's was shown to be significant even after controlling for the confounding variables of prematurity and birth asphyxia, both of which occur at a higher incidence in IDM's [12]. Furthermore, the rate and severity of hypocalcemia are directly related to the severity of maternal diabetes [12]. Decreased serum Ca in IDM's is related to decreased serum Mg and decreased PTH secretion [3,13,87]. It has been speculated that these abnormalities may be due to a maternal, and consequent fetal, state of Mg deficiency [14]. Serum CT elevations in theory may aggravate hypocalcemia in IDM's also, since serum CT rises after birth in IDM's to a degree similar to infants of non-diabetic mothers [15].

(d) Infants of Epileptic Mothers

Infants of epileptic mothers theoretically may also be at risk for developing early hypocalcemia [16]. Drugs such as phenobarbital and phenylhydantoin increase the hepatic catabolism of vitamin D and its metabolites, and epileptic patients treated with these drugs are at risk for osteomalacia [17]. Pregnant mothers receiving anti-epileptic drugs who are supplemented with vitamin D (1000 U/d) give birth to infants having no neonatal hypocalcemia [18], but the exact relationship with neonatal Ca homeostasis is unclear.

(B) Late Neonatal Hypocalcemia

High P Intake

Infants receiving high P-containing formulas [19-21] or rice cereals [22] are at particular risk for hypocalcemia. Secondary hyperparathyroidism may occur in some of these infants, presumably

in an attempt to restore Ca homeostasis. It is thought that hyperphosphatemia leads to hypocalcemia by increasing tricalcium phosphate deposition in bone, enhancing the hypocalcemia effect of CT, and/or inhibiting the calcemic response to PTH [23].

Other causes of late neonatal hypocalcemia include Ca intestinal malabsorption [24], hypomagnesemia [1], hypoparathyroidism [25-29]. The latter may be transient, due to maternal hyperparathyroidism, or permanent. If permanent, it may occur sporadically, with or without the other features of the DiGeorge's syndrome: thymic hypoplasia or aplasia, and cardiac malformations, due to abnormal development of the 3rd and 4th embryonic branchial pouches Hypoparathyroidismmay also be inherited, as a sex-linked or autosomal dominant inherited trait.

The hypocalcemia of nutritional vitamin D deficiency rickets is mainly due to decreased Ca and P intestinal absorption [30]. It may be partially compensated by a secondary hyperparathyroidism. Neonatal liver disease may lead to poor vitamin D (and metabolites) intestinal absorption, decreased 25 hydroxylation of vitamin D, rickets and hypocalcemia [30]. A congenital block of the renal l-alpha hydroxylation causes vitamin D dependent rickets and may lead to hypocalcemia [30].

Phototherapy has been described as a possible cause of neonatal hypocalcemia [31]. The mechanism is unclear; it has been suggested that phototherapy could decrease melatonin secretion. Since melatonin decreases bone Ca uptake by enhancing corticosterone action on bone, a decrease in melatonin secretion would increase bone Ca uptake, resulting in hypocalcemia [32].

(C) Decreased iCa

A decrease in iCa however may occur without a decrease in the total serum Ca concentration. This occurs mainly during "exchange" blood transfusions with citrated blood, which results in a chelation of Ca by citrate [33-36]. Alkalosis (metabolic or respiratory) may cause a decrease in iCa by increasing Ca binding to protein and decreasing the Ca flux from bone to the extracellular space [37]. In vitro, free fatty acids may combine with iCa to form Ca soaps [38]. It is possible, although not demonstrated in vivo, that intravenous lipid therapy may cause a similar problem.

(3) Signs, Symptoms and Complications of Neonatal Hypocalcemia

Hypocalcemia may be asymptomatic in the neonate, in particular in premature infants [39]. The presence of clinical signs is not well correlated with the degree of hypocalcemia as defined by a total serum Ca concentration [40]. It is therefore important to "screen" all infants at risk for hypocalcemia, such as preterm and asphyxiated infants, and IDM's. The signs of hypocalcemia are not specific and may be confused with signs provoked by hypoglycemia, sepsis, central nervous system injury, or narcotic withdrawal. The common signs of hypocalcemia are the expression of neuro-muscular irritability: jitteriness, apneic episode or even frank seizures. The classical signs of carpopedal spasm and laryngospasm are often not observed in the neonate, particularly in the preterm infant. Irritability or lethargy with poor feeding may be both observed. Decreased cardiac contractility may be a consequence of severe hypocalcemia [41]. The electrocardiogram (ECG) may show a prolonged QT interval corrected for heart rate (the interval from the beginning of Q to the end of T, Q-Tc interval, < 0.2 sec) [42]. However these ECG measurements correlate only weakly with iCa determinations and are not specifically helpfully on a

clinical basis [43,44]. In clinical practice, therefore, the diagnosis of hypocalcemia is generally made from the determination of serum total Ca. We recommend the measurement of serum Mg at the same time, as the signs of hypomagnesemia are similar to those of hypocalcemia, and since both conditions may coexist. Measurements of PTH, CT, and vitamin D metabolites are not routinely indicated, and are useful mainly in cases of prolonged, refractory, or recurrent hypocalcemia. Chest films (identifying the absence of thymus) and immunologic studies are indicated when DiGeorge syndrome is suspected.

(4) Treatment

(A) Symptomatic Hypocalcemia

It is generally accepted that symptomatic hypocalcemia should be corrected with Ca salts. Ca gluconate 10% (containing 9.4 mg of elemental Ca per ml) is preferred to Ca chloride which may cause acidosis. It may not be administered together with bicarbonate solutions, due to the risk of precipitation within the intravenous solution.

Route

The intravenous (IV) route has potential hazards: skin sloughs (due to extravasation of the Ca solution into soft tissues) and bradycardia or cardiac standstill (when Ca is administered at a high rate). It is therefore mandatory to maintain neonates receiving IV Ca infusion on constant cardiac monitoring, and to check frequently IV sites. Ca-containing IV tubing should be labelled clearly to help prevent mistakes such as "flushing" the line. Intraarterial or intramuscular administration of Ca is prohibited, due to the risk of tissue necrosis. When feeds are well tolerated, oral Ca supplementation is preferable, to avoid the risks of parenteral administration. However, preparations such as Neocalglucon, with their high sugar content are hypertonic and may cause an increased frequency of bowel movements. The potential of high osmolarity solutions for precipitating necrotizing enterocolitis should be considered, and we recommend only the use of Ca gluconate 10% for oral route.

Dosage

Acutely, in cases of seizures, 2 ml/kg body weight of Ca gluconate 10% may be administered intravenously over 10 minutes, while monitoring the heart rate for bradycardia, which would require slowing or stopping the infusion. In less acute cases the authors have found satisfactory a regimen of 75 mg elemental Ca/kg per day IV, until the serum Ca concentration reaches the normal range. Thereafter IV Ca may be reduced to 37 mg/kg per day for a day, 18 mg/kg per day the following day and then discontinued. With this regimen, most cases of "early" neonatal hypocalcemia have required 3 days of therapy. The continuous IV infusion is preferred over intermittent IV administration, being more efficient and less dangerous: the increase in serum Ca following a bolus infusion is of short duration and may cause bradycardia and cardiac standstill in case of too rapid administration. The daily dose for the oral route is similar, and mabe given at 4 to 6 hours intervals.

(B) Asymptomatic Hypocalcemia

There is no general agreement as whether asymptomatic hypocalcemia should be treated or not. Some investigators question the usefulness of treating asymptomatic infants who have decreased serum total Ca concentration on the basis of: 1) lack of prediction of iCa from total Ca measurements [43-45],

2) self-correction of hypocalcemia with time [44], 3) lack of clear demonstration of long term benefit of therapy in follow-up studies [45,46], and 4) potential theoretic dangers of IV Ca infusion, in particular brain calcification [47]. However 1) the data related to time of self correction and risk of brain calcification are controversial [48,49], 2) long term studies are difficult to perform due to the common occurrence of associated factors such as birth asphyxia or prematurity, and 3) neonatal hypocalcemia appears to have other potential long-term side effects such as tooth enamel hypoplasia [50]. We recommend consideration of Ca supplementation in asymptomatic neonatal hypocalcemia in view of the major potential adverse effects on central nervous system and cardiac function during hypocalcemia, provided "the treatment is not worse than the disease". In practice, we generally treat asymptomatic preterm infants who have a serum total Ca < 6 mg/dl, as iCa concentrations are extremely low in these infants; in term infants wuse 7 mg/dl as the cut point.

(C) Other Approaches

$1,25(OH)_2D$ and l-alpha hydroxy vitamin D (an analog of $1,25(OH)_2D$) have been successfully used in neonatal hypocalcemia in term and large preterm infants [51,52] but their side effects are uncertain and we would still consider these drugs as experimental. Similarly IV PTH administration should also be considered experimental [25,53].

(D) Prevention of neonatal hypocalcemia in asphyxiated or preterm infants has been accomplished by various regimens including oral [54] or IV Ca [55], or vitamin D metabolites [56-60]. The need for such a preventive approach is controversial, and we do not recommend it routinely.

RICKETS OF PREMATURITY

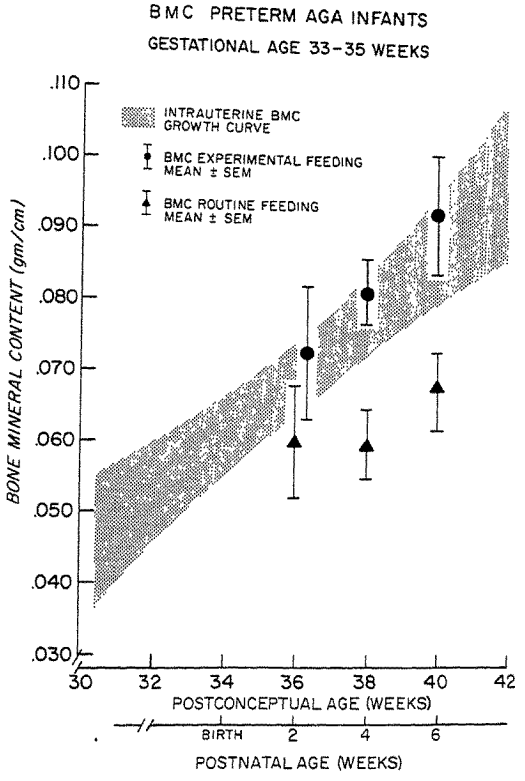

Figure 2.
Postnatal bone mineral content (BMC) of Ca and P fortified formula group of infants born between 33 and 35 weeks' gestation, compared to the intrauterine bone mineralization curve and to the postnaltal BMC of standard formula fed infants born between 33 and 35 weeks' gestational age. BMC in experimental feeding infants is not different from the intrauterine BMC and is significantly higher than BMC in standard feeding infants at 4 and 6 weeks' postnatal age. (From Steichen JJ, Gratton TL, Tsang RC. Osteopenia of prematurity: The cause and possible treatment. J Pediatr 1980; 96:528-534, reproduced with permission).

[61-63] . This frequency is inversely related to the gestational age of the infants and directly related to the severity of infant illness [61,64] . In most cases, vitamin D deficiency does not appear to be the cause, and 25-OH D serum concentrations are in the normal range [65-72] . These infants often have been receiving total parenteral nutrition (TPN) for extended periods with intakes of Ca and P much below fetal intrauterine requirements (which are up to 150 mg Ca/kg/day and 75 mg P/kg/day) [68] . TPN cholestasis [74,75] , and hypersulfatemia [76] may aggravate the picture. It is also possible that parenteral nutrition solutions contaminated with aluminum cause loading and contribute to the bone disease [77] . In infants with bronchopulmonary dysplasia, particularly those with cor pulmonale or patent ductus arteriosus, chronic fluid restriction may lead to inadequate caloric and mineral intake, and chronic furosemide therapy theoretically may further decrease bone demineralization [65] ; secondary hyperparathyroidism described in some of these infants also may be a complicating problem [65,68,72].

(2) Diagnosis

Rickets in premature infants is usually subclinical, and standard radiographic methods are unable to detect early bone demineralization [78] . However fractures occur frequently (up to 30% of a population of very low birth weight infants in a very recent study [79] , involving mainly long bones and ribs. Bone mineral content (BMC) measurements by direct photon absorptiometry, though much more accurate in detection of early osteopenia, are available only in a few institutions. Although this method has proven itself for research purposes [68,80-82] , its clinical usefulness has not yet been validated. Typical clinical and radiological signs of rickets frequently occur during periods of rapid growth, after the period of acute illnesses has been overcome.

(3) Prevention and Treatment:

Measurements of bone mineral content (BMC) by direct photon absorptiometry have been performed to evaluate the efficacy of different dietary regimens in preterm infants for the prevention or treatment of rickets of prematurity [68,80-83] (Fig 2). The studies done using this method had led to the development of "premature infant formulas" that are fortified with Ca, P and vitamin D, and which allow BMC values to remain in the expected intrauterine range. Preterm infants fed their own mother's milk may be at a higher risk of P deficiency in addition to Ca deficiency [82] . Routine Ca and P supplementation has been advocated in these infants [84] . Commercial powders which may be mixed with human milk are now available. These powders contain a lactose and cow milk derived whey protein base with multiple trace elements and minerals other than Ca and P added to the preparation. More studies are needed to validate the efficacy of these supplements to human milk in the prevention of rickets [85] The exact requirements of Ca, P, and vitamin D in preterm infants with rickets are unknown. From our experience, we would recommend a daily intake of up to 200 mg Ca/mg, up to 113 mg P/kg, and 400 IU of vitamin D [86] . In addition, it is important to reduce the exposure of infants to risk factors for bone demineralization such as TPN or furosemide therapy.

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RECENT ADVANCES IN THE PRENATAL DIAGNOSIS OF BIOCHEMICAL DISORDERS

P.W.K. WONG
Department of Pediatrics,
Rush Medical College,
Chicago, U.S.A.

INTRODUCTION

Shortly after the rediscovery of Mendel's Laws [1], Garrod in 1902 first proposed the concept of "inborn errors of metabolism" [2]. In 1941, Beadle and Tatum established the hypothesis of one-gene-one-enzyme [3]. Gibson in 1948 demonstrated, for the first time, a deficiency of the enzyme NADH-dependent methemoglobin reductase in the inherited type of methemoglobinemia [4]. Since the elucidation of DNA structure by Watson and Crick in 1953 [5] and the decipher of the genetic code, numerous investigators contributed to the understanding of biochemical disorders at the molecular level. The first systematic attempt to elucidate a group of inherited biochemical disorders was made by Cori and Cori in glycogen storage disease [6]. Numerous other studies [7] demonstrated that diffent enzyme defects could produce a similar or identical clinical disease. In addition, genetic heterogeneity was observed in most biochemical disorders [7]. Hence, in order to make accurate diagnosis, demonstration of a specific defect in the proband or affected relatives is a prerequisite.

DIAGNOSTIC APPROACHES

Figure 1 illustrates the concept of biochemical disorders. A mutation at the DNA level may result in a lack of transcription of a specific gene or may result in an abnormal mRNA. Hence, deficiency of a specific protein (enzyme) or the synthesis of an abnormal protein with defective function may occur. Since metabolic processes generally occur in series of reactions, a defective enzyme usually produces accumulation of metabolites proximal to the defect such as M2 and results in a deficiency of metabolites distal to the defect such as M3. Hence, a diagnosis may be made by the study of metabolites, specific enzymes, mRNA or DNA. In practice, metabolites and mRNA are less commonly used for prenatal diagnosis.

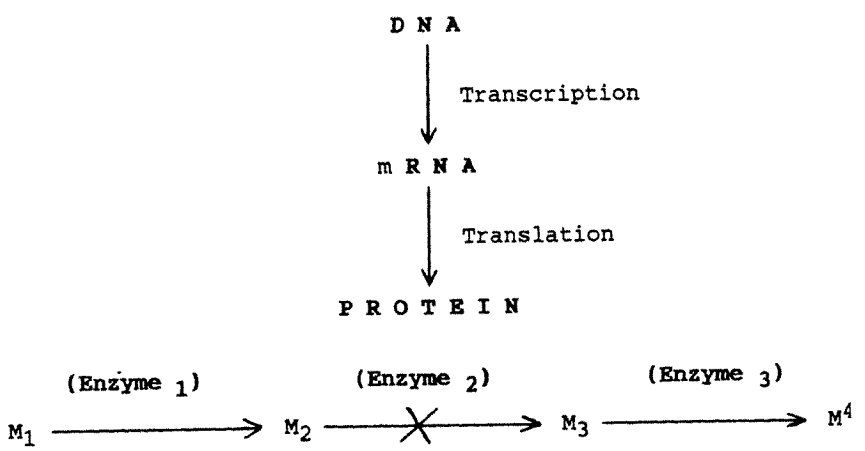

Fig. 1 Schematic representation of metabolic defect.

Amniocentesis

The established approach in the prenatal diagnosis of biochemical disorders has been the use of amniocentesis at 16 weeks of gestation to obtain amniotic fluid and amniocytes for biochemical studies[9]. The advantages of this approach include extensive experience in the performance of amniocentesis, the low risk of the procedure [9-11], the absence of maternal tissue contamination, the availability of a large number of control samples to establish the normal range and the easy transportation of the fluid and cells to specialized laboratories. The disadvantages of this approach include a delay of 3 to 4 weeks required for tissue culture before enzyme studies and the variation of enzyme activities due to differences in cell cycle, rate of growth and cell passages.

Fetoscopy

Direct visualization of the fetus and placenta is possible by the use of fiber optic fetoscope [12]. Fetal blood sampling through fetoscopy at about 18 weeks of gestation has been successfully used for the diagnosis of hemoglobinopathies [13], hemophilia due to factor VIII deficiency, factor IX deficiency [14] and Von Willebrand's disease [15]. However, fetal loss varied from 0 to as high as 16%. More recently, aspiration of fetal blood from the cord close to the placenta through transabdominal insertion of a spinal needle under ultrasound guidance has become more popular. This permits the collection of fetal blood without contamination by maternal blood or amniotic fluid [16] by using a 22-gauge needle with a 3mm long, 26-gauge tip for insertion into a blood vessel. There is so far inadequate data for an accurate estimate of fetal loss associated with this procedure.

Chorionic Villus Sampling

Hahnemann and Mohr used transceical chorionic villus sampling for genetic diagnosis in the late 1960s [17]. The technique required a special but rather large fiber optic hysteroscope without real-time ultrasound guidance for chorionic villus biopsy. This technique was associated with some 42% risk of complications. In about 1970, chorionic villus sampling with a blind technique was used in Anshan, China [18]. Syncytial cells and chorionic villi were obtained using metal cannula and introducer for the examination of Barr bodies with an accuracy of about 90%. The incidence of spontaneous abortion was about 6%. With the availability of real-time ultrasound machines to locate the placenta,

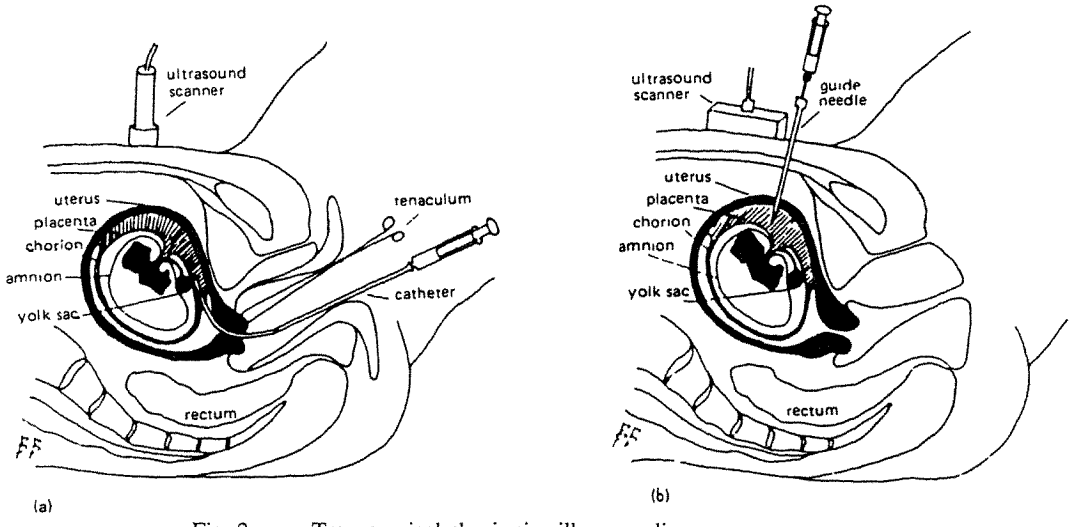

Fig. 2 a. Transcervical chorionic villus sampling.
b. Transabdominal chorionic villus sampling.

(From Brambati and Oldrini 1986)

chorionic villus sampling became a practical technique. For example, in 1982 Kazy et al [19] using biopsy forceps, were successful in obtaining chorionic villi in all 26 pregnancies at high genetic risk. Thirteen pregnancies were terminated. The other 13 ended with the delivery of normal infants. There were no spontaneous abortions.

With the development of the Portex catheter, a soft plastic catheter with a deformable metal introducer to adapt to the shape of the uterus and sonographic guidance (Fig. 2), Old et al successfully obtained chorionic villi for the diagnosis of hemoglobinopathies in three cases in 1982 [20]. Kaplan et al [21] in France and Brambati and Simoni [22] in Italy also demonstrated that chorionic villus sampling was a practical approach for first trimester antenatal diagnosis in 1983. Currently transcervical chorionic villus sampling using a Portex catheter under ultrasound guidance is the most commonly used technique. When microscopy is used immediately to verify the adequacy of the sample, a success rate of over 97% can be achieved. A single aspiration may yield 10-25 mg of wet weight. However, sometimes multiple aspirations may be required. The optimal time for this procedure is at about 10 weeks of gestation.

Due to the risk of infection in the transcervical approach, Smidt-Jensen and Hahnemann [23] developed the transabdominal approach. Using ultrasound guidance, a 15 cm, 18-gauge spinal needle is inserted transabdominally to the edge of the placenta. A second spinal needle (18 cm, gauge 22) is introduced through the first needle for aspiration. About 30 mg of tissue can be obtained by repeated aspirations (Fig.2) [24]

In 1985, the experience of Jackson in Philadelphia, Pergament in Chicago and Brambati in Milan was pooled in a WHO report [25]. Table 1 compares the relative risk of different approaches in chorionic villus sampling. The transabdominal approach may have risks similar to or less than the transcervical approach when more experience is obtained.

Acute infection followed by maternal septicemia and fetal loss in the transcervical approach occurs in approximately 1 in 300 cases and remains a concern [26]. The advantage of chorionic villus sampling for prenatal diagnosis of biochemical disorders is that results may be obtained at 9 to 11 weeks of gestation. Since the tissue obtained can be used directly for biochemical studies, the duration of waiting and the anxiety of the parents are markedly reduced.

Table 1. Data concerning the four main CVS procedures (WHO registry, 17 April 1985)

CVS Procedure	No.of cases	Failed sample	Continuing pregnancies	Fetal loss Max.	Delivered
Portex catheter (Chicago, Milan, Philadelphia)	1884	24 (1.2 %)	1734	44 (2.4 %)	725
Biopsy forceps (Paris, Rome, Cagliari)	324	4 (1.2 %)	252	12 (4.6 %)	43
Transabdominal Aspiration (Aalborg)	125	1 (0.8 %)	118	4 (3.4 %)	47
Endoscope (Lund)	113	1 (0.8 %)	100	7 (7.0 %)	

One of the potential disadvantages of chorionic villus sampling for biochemical assays is the contamination by maternal tissue. Under the microscope, the villi appear as semitransparent finger-like processes. In contrast, decidual tissues appear structureless and membranous. All atypical tissue should be removed by dissection and discarded. In addition, trophoblast, and extraembryonic tissue, may not represent fetus. Kalousek and Dill [27] observed chromosomal mosaicism confined to the chorion in 2 of 31 cases studied. The abnormal cells were observed only in the chorion but not in cells in the amnion, skin or cord blood. Potential misdiagnosis remains a great concern.

BIOCHEMICAL ANALYSIS

Kazy et al were the first to perform enzyme assays on homogenates of chorionic tissue samples for prenatal diagnosis in 1982 [19]. Simoni et al determined the normal activities of a number of lysosomal enzymes [28]. Normal values for some 20 enzymes associated with biochemical disorders were established by Kleijer et al [29]. Most enzyme activities are lower in chorionic villi than in amniotic fluid cells. However, some are higher in chorionic villi.

Table 2. First Trimester Diagnosis of Metabolic Disorders

Disease	No.	Normal	Affected	Carrier
Tay-Sachs	19	14	5	1
Gm^1	1	—	1	—
Sanfillipo	1	—	1	—
Sickle Cell	1	1	1	3
Gaucher's	1	1	—	—
Alpha-l-antitrypsin	2	1	—	1
Neimann-Pick	1	—	1	—
Beta thalassemia	5	4	—	1
Lesch-Nyhan	1	—	1	—
Farber's	1	1	—	—
Zellweger's	1	—	1	—
I cell	2	2	—	—
Metachromatic Leukodystrophy	1	1	—	—

From Jackson et al 1985.

First trimester diagnosis has been performed in more than 40 different inborn errors of metabolism. Enzyme determinations can be performed by using chorionic villi directly or cultured chorionic cells Table 2 shows examples of some of the disorders studied by Jackson and his colleagues [30] . In an international survey in 1984, a total of 106 pregnancies at risk for metabolic disorders had been monitored by biochemical studies in chorionic tissue. Twenty seven embryos (25%) were diagnosed to be affected. The diagnoses were confirmed subsequently by analysis of fetal cells after abortion. However, of the 79 cases where the embryos were diagnosed to be either normal or heterozygotes, a false negative tests were observed in affected fetuses. Hence strict criteria must be used in performing biochemical studies in chorionic tissues. There should be an adequate number of studies in normal chorionic tissues to establish the normal range and the simultaneous determination of a second enzyme as control. As the enzyme in the chorionic tissue may be different from that in the fetal tissue, more detailed studies such as electrophoresis or other biochemical characterization may be required in addition to specific activity studies. When tissue culture is required before biochemical studies, maternal cell contamination is a real potential for error. Hence in doubtful situations, amniocentesis at a later date for confirmation is advisable.

Determination of Metabolite

Quantitation of "abnormal" metabolite in chorionic tissue for prenatal diagnosis has also been used. For example Smith and associates determined cystine content in chorionic tissue (Table 3) and successfully diagnosed an affected embryo with cystinosis [31] .

Incorporation of Precursor

A third approach is based on the incorporation of radioactive precursors in cultured chorionic cells [32] . For example, xeroderma pigmentosa and Lesch-Nyhan syndrome are characterized by defective utilization of thymidine and hypoxanthine respectively. Hence, when [^{3}H] thymidine or [^{3}H] hypoxanthine is added to the culture medium of the corresponding chorionic cells, there is decreased or absence of incorporation of radioactive precursor in the abnormal cells when compared with that in normal cells. This can be demonstrated by autoradiography as a decrease in or absence of grains.

DNA ANALYSIS

In single gene defects resulting in metabolic disorders, there are alterations of specific DNA sequences such as deletion, addition or point mutation. Since all nucleated cells contain the same genetic material in a given indivi-

Table 3. Prenatal Diagnosis of Cystinosis

	Cystine content of chorionic villi	
Gestational Age (weeks)	Fresh tissue	Cultured Cells
	(nmol 1/2 cystine/mg protein)	
Normal controls		
8	0.11	0.11
9	0.09	0.18
10	0.13	
11	0.13	0.12
Case at risk		
9	34.7	9.7

From Smith ML et al.

dual, recombinant DNA technology has been used to detect mutations specific to various inherited diseases in chorionic tissues. Three approaches have been devised for diagnosis.

Gene Dosage Determination

Kan and his associates were the first to use gene dosage determination to establish the genotype of individuals with alpha thalassemia [33]. The most common alpha thalassemias are due to gene deletion and the phenotypes can be classified according to the number of deleted genes. Using a radioactive alpha globin cDNA probe, the amount of radioactivity hybridized with the chorionic DNA can be quantitated. Hence, the number of copies of the alpha globin gene can be determined. This approach can be used for the diagnosis of hemoglobin H disease and hemoglobin Bart's hydrops fetalis. Recently, using modification of this approach called restriction endonuclease mapping, Wang and associates in China have successfully evaluated chorionic villus samples from 33 pregnancies at risk for alpha thalassemia [34].

Mutation Site Analysis

When the normal and the mutated gene sequences are known, the single nucleotide substitution is apparent. Specific endonuclease may be found that cleaves the normal sequence but fails to cleave the mutated sequence. Figure 3 shows the use of endonuclease Mst II in the study of sickle cell anemia. In the normal gene, Mst II produces a 1.1 kb fragment. In the s gene, Mst II fails to recognize the mutation in codon 6, hence producing a 1.3 kb fragment [35]. Hence, normal cells have only the 1.1 kb band, the cells from the heteroxygote have both 1.1 kb and 1.3 kb bands and cells from the homozygote for sickle cell disease have only a 1.3 kb band.

Linkage Studies

It is known that 1 in 100 to 200 nucleotide bases differ between the chromosome pairs. This high frequency of polymorphism has been fruitfully used to detect genes closedly linked to these polymorphic sites. Kan and Dozy were the first to recognize the usefulness

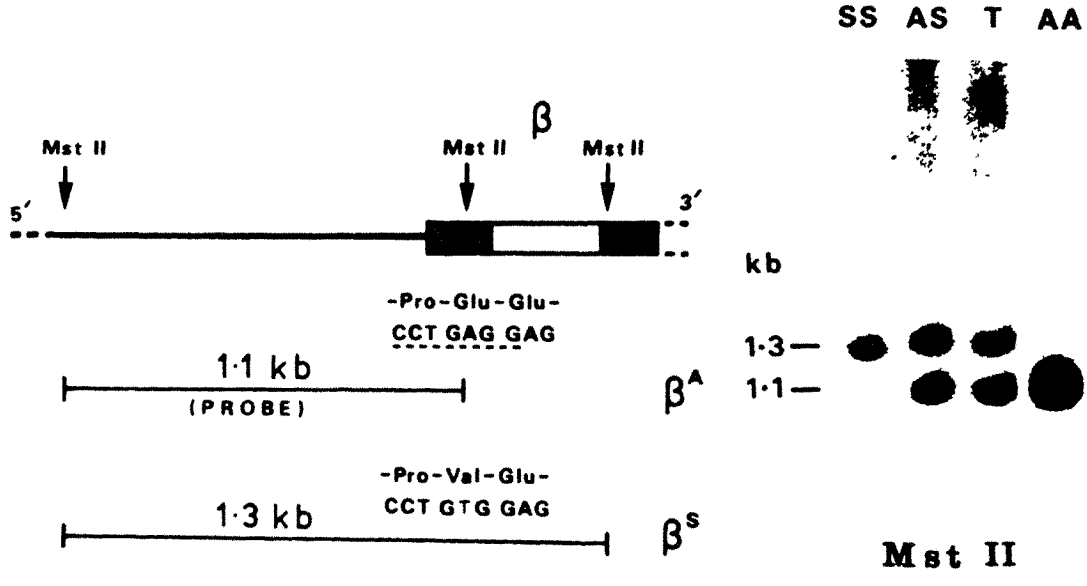

Fig. 3 First trimester diagnosis of sickle cell disease. Autoradiograph shows a normal subject (AA), a sickle cell carrier (AS), a patient with sickle cell disease (SS) and chorionic villus sample (T).

(From Old 1986)

of such linkage in the diagnosis of sickle cell disease [36] . This approach requires family studies including the affected individuals, the parents and the unborn. Currently this technique is employed for prenatal diagnosis of cystic fibrosis, Duchenne muscular dystrophy, haemophilia phenylketonuria and alpha-1- antitrypsin deficiency. Many more probes are in the experimental or developmental stage and will soon be used clinically.

SUMMARY

In the last 5 years or so, we have witnessed a rapid adoption of these newer methods so that the antenatal diagnosis of biochemical disorders can be made earlier. In the next 5 years we can anticipate the use of "gene therapy" to correct the chemical derangement in inborn errors of metabolism.

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OBSERVATION OF THE BLOOD SUGAR PATTERN AND HYPOGLYCAEMIA IN NEONATES

X.J. Guan, G.Z. Li, S.J. Yu.
Sun Yet-sen University of Medical Sciences, Guangzhou,
People's Republic of China.

Neonatal hypoglycemia, with symptoms or not may cause brain damage. So, it must be diagnosed in time. The Dextrostix method was chosen for determination, which may give the blood sugar value by using Glucometer, Dextrostix paper and one drop of blood within 5 minutes.

Three groups totalled up to 195 neonates were studied: (1) 100 normal term neonates and their mothers; (2) 50 neonates of fetal asphyxia and their mothers; (3) High risk group, including 45 neonates of neonatal asphyxia, macrosomia, infant of maternal eclampsia or infant of diabetic mother.

Results : The blood sugar of normal neonates declined sharply to its lowest point at 2 hrs after delivery and it would be the time when hypoglycemia occurred. The difference of maternal-neonates blood sugar was lower in neonates of fetal asphyxia than normal neonates. The incidence of hypoglycemia in normal neonates and high risk group were 14% and 33% respectively. The manifestations of hypoglycemia were non-specific, most of them without any symptom.

Conclusions : A routine test for neonatal blood sugar must be made to detect hypoglycemia in regular hour as early as 1-2 hr after delivery. It is advisable to give mother intravenous glucose solution in case of fetal asphyxia. Neonatal hypoglycemia could be avoided by early feeding.

A2

NEONATAL SEPTICAEMIA - A FIVE YEAR REVIEW

A.Y.C. Tam, E.Y.W. Kwan, R.W.H. Yung[1].
Departments of Paediatrics and Microbiology[1], University of Hong Kong.

A restrospective review was carried out of all culture proven septicaemia occurring in the neonatal intensive care unit from the years 1981-85. A total of 174 blood cultures were reported to be positive during the study period. An additional 3 cases of positive cerebral spinal fluid (CSF) cultures were included in the study. In 128 cases (72.32%) the isolate was considered to be indicative of septicaemia by a clinical-pathological correlation score. Fifty-five cases were due to gram-negative organisms, with Escherichia coli being the commonest (17 cases), followed by klebsiella (7 cases), enterobacter (5 cases) and acinetobacter (5 cases). Gram-positive organisms constituted 63 cases. Staphylococcus (SA) was the commonest (39 cases), followed by group B streptococcus (GBS) (11 cases) and Staphylococcus epidermidis (9 cases). Two cases were due to Bacteroides fragilis while 3 were caused by Candida albicans. Five cases had mixed growth. 20.3% of the septicaemic episodes occurred within the first 48 hours of life, with a predominance of gram-negative organisms. Another 25.8% occurred up to 1 week of age. Twenty-four babies died, giving a mortality of 18.8%. The mortality was highest when septicaemia ocurred within the first 48 hours of life (29.2%). A lumbar puncture was done together with the blood culture in 106 instances (82.8%). The same organism was cultured from both blood and CSF in 9 cases (GBS 6, Listeria monocytogenes 1, SA 2). Two other babies who had GBS septicaemia had biochemical and cytological abnormalities in the CSF although no growth was obtained. They also had neurological involvement clinically and were therefore treated as if they had meningitis. In 3 babies the organism was only isolated from the CSF (GBS 1, Listeria monocytogenes 1, moraxella species 1). Altogether 14 babies had meningitis, giving an associated incidence of 10.9%. The in-vitro antibiotic sensitivity pattern for the organisms was also examined. In early septicaemias, gentamicin is usually effective against the gram-negative organisms. Penicillin is useful against the gram-positive organisms except methicillin-resistant SA, which is only sensitive to vancomycin or co-trimoxazole. In septicaemias occurring after 48 hours of life gentamicin is still effective for the gram-negative organisms although resistance is occasionally seen. From in-vitro tests ampicillin does not offer any advantage over penicillin as most gram-negative isolates are resistant to it. Cefamandole and cefuroxime have better activity against both gram-positive and gram-negative isolates than ampicillin. The presence of MRSA requires the use of strong antibiotics like vancomycin. It should be used in septic babies who are already colonized by the organism.

A3

IMPACT OF A NEONATAL INTENSIVE CARE PROGRAMME ON NEONATAL MORTALITY OF VERY-LOW-BIRTH-WEIGHT BABIES

P.T. Cheung, C.Y. Yeung, A.Y.C. Tam.
Department of Paediatrics, University of Hong Kong.

A retrospective analysis was carried out of the neonatal mortality rate (NNMR) of the VLBW live infants (501-1500 gm) born in Queen Mary Hospital from 1975-86. In late 1980 a neonatal intensive care programme was introduced. Comparison was made of the two 6-year periods before and after this re-organization. The Chi-square test was used for statistical analysis.

NEONATAL MORTALITY RATE

Baby Weight (gm)	1975 - 1980		1981 - 1986		
	Total live births	Death rate/ 1000	Total live births	Death rate/ 1000	P
501 - 750	5	1000	26	846	>0.25
751 - 1000	18	778	35	514	0.1 > p > 0.05
1001 - 1500	64	797	83	108	<0.001
Total	87	805	144	340	<0.001

A significant fall in the NNMR of the VLBW infants was seen in the 6 years after introduction of the neonatal intensive care programme (340/1000 live births vs 805/1000). It is obvious that data from the 1001-1500 gm LBW group was chiefly responsible for such change. Further breakdown of the NNMR in 1981-86 showed continual decrease in the overall rate (500/1000 live births in 1981-2 vs 208/1000 in 1985-6 p <0.01). Again the improved survival rate was mainly in the 1001-1500 gm LBW group, while a similar trend was not obvious in the 751-1000 gm weight group. Our department has adopted a policy of being less aggressive in resuscitating those infants whose birth weights are near or below 750 gm.

This pattern of decrease in the NNMR of VLBW infants is in accordance with the experience of other centres.

A4

DEVELOPMENTAL CHANGES OF THYROID FUNCTION IN INFANTS BORN EXTREMELY PRETERM

V.Y.H. Yu, M. Mercado.
Department of Paediatrics, Monash Medical Centre, Melbourne, Victoria, Australia.

Indices of thyroid function were measured in 108 infants born at 23-31 weeks gestation, after birth, at 24 and 72 hours, and at 1, 3, 4, 5 and 6 weeks of age. This group was characterised by low serum thyroxine (T4), normal thyroid stimulating hormone (TSH), low-normal thyroid binding globulin (TBG), low free thyroxine index (FTI) and low triiodothyronine (T3). The incidence of hypothyroxinaemia defined as a serum T4 value of less than 65 nmol/l was 58% after birth, increasing to 84% at 1 week, after which there was progressive reduction to 36% by 6 weeks of age. Mean T4 values were inversely proportional to gestational age during this study period. Infants of 23-28 weeks gestation had significantly lower T4, TBG, FTI and T3 values compared to those of 29-31 weeks gestation. Infants who had hyaline membrane disease (HMD) had significantly lower T4 and FTI values compared to those without HMD for up to 3 weeks of age. Similar differences were found between deaths and survivors in the first week after birth. This study suggests that there is increasing delay in maturation of the hypothalamic-pituitary-thyroid axis control with increasing prematurity. In addition, the data suggest that infants who were extremely preterm or those with HMD had worse and more persistent abnormalities of thyroid function secondary to their illness and metabolic stress. The significance of our findings, in particular that of prolonged hypothyroxinaemia, is uncertain. The role of thyroid replacement therapy in these very preterm infants therefore need to be assessed with a randomised clinical trial.

A5

NECROTIZING ENTEROCOLITIS IN NEONATES WITH SYMPTOMATIC CONGENITAL HEART DISEASE

M.P. Leung, K.T. Chau[2], P.W. Hui, A.Y.C. Tam, F.L. Chan[1], C.Y. Yeung.
Departments of Paediatrics & Radiology[1], University of Hong Kong.
Paediatric A Unit, Queen Elizabeth Hospital[2], Hong Kong.

The present study examines the incidence, clinical features and predisposing risk factors of necrotizing enterocolitis (NEC) in neonates suffering from major congenital heart disease. Between January 1985 and December 1986, among the 133 symptomatic neonates with proven congenital heart disease (CHD) admitted into our cardiology unit, 9 (7%) developed NEC. The clinical features of these neonates were compared to those of premature babies who developed NEC in our neonatal intensive care unit during the same study period. Factors that may contribute to the development of NEC for the two groups of babies were also noted. Infants with symptomatic CHD and NEC were mostly full term babies weighing more than 2.5 kg. They had more severe gastrointestinal disturbances (frank bleeding per rectum), systemic upset (metabolic acidosis, abnormal clotting profiles) and a higher mortality than their premature counterparts. Commonly quoted predisposing factors for NEC such as birth asphyxia, umbilical catheterisation and other maternal risk factors were conspicuously absent in the group with CHD. On the other hand, when compared with the 124 infants with major CHD who did not develop NEC, three significant risk factors were identified: prostaglandin infusion, ($p < 0.05$), prostaglandin-induced apnoea ($p < 0.005$) and a hypotensive episode ($p < 0.001$). The association of these risk factors with NEC further supports the hypothesis that selective circulatory ischaemia is a major cause of the intestinal pathology.

A6

CONGENITAL HEART DISEASE IN THE NEONATE

R.N.S. Lo, K.C. Lau, M.P. Leung, C.Y. Yeung.
Department of Paediatrics, University of Hong Kong.

Between 1980 and 1984, 308 neonates under one month were admitted because of congenital heart disease. 166 (54%) were under one week old. The reasons for admission were cyanosis (70%), heart failure (24%), and cyanosis and heart failure (6%). Diagnosis was by echocardiography alone in 52 cases (16%). Complications occurred in 49 of 247 cardiac catheterisations (20%), death being attributable to the procedure in 13 cases (5%). The common conditions were univentricular connections (16.5%), severe right ventricular outflow tract obstructions (16.5%), transposition of great arteries (15%), coarctation of aorta syndrome (11%), Fallot's tetralogy (6%), and hypoplastic left heart syndrome (5%). Early palliative or corrective surgery was performed in 125 cases with a surgical mortality of 28%, and 32 survivors underwent a second corrective operation with a mortality of 28%. Overall mortality to date among the 308 cases is 48%.

A7

SEIZURE SUSCEPTIBILITY AND THE NEUROCHEMICAL CHANGES IN THE GENETIC SEIZURE-PRONE VERSUS SEIZURE-RESISTANT RAT'S BRAIN

X.R. Wu, P.S. Lin, Y. Hua, Z.H. Zhang, Q.H. Zuo.
Pediatrics, First Teaching Hospital, Beijing Medical University, Beijing, People's Republic of China.

Our previous work showed that the susceptibility of hyperthermia-induced seizures was significantly higher in P77PMC rats (a genetic audiogenic seizure-prone rat strain) than the Wistar rat (seizure-resistant) on 20-30 days after birth. In order to search the mechanisms of this phenomenon, we established fetal brain (15-16 days gestation age) cerebral cortex primary dissociated neuronal cell cultures in both P77PMC and Wistar rats, and observed the differences of the morphology, biochemical changes during cell development between the two groups. There was no difference in morphology development, but high affinity GABA uptake was significantly lower in P77PMC (480± 87pmole/min/mg protein) than Wistar (1520±152pmol/min/mg protein) on the 20th day in vitro. GABA release evoked by high potassium media had a similar tendency with GABA uptake. Cholecystokinin-Octapeptide 10-7M (CCK-8) significantly enhanced GABA release rate in P77PMC group. These preliminary results suggest that the impairment of GABAergic system and the defect of CCK-8 system during development in the brain of P77PMC rat may be a factor to cause their higher seizure susceptibility? Further work are studying.

A8

EARLY PROGNOSTICATION OF LOCOMOTION IN CEREBRAL PALSY

M.J. Watt and C.M.T. Robertson.
Department of Pediatrics, University of Alberta; Glenrose Rehabilitation Hospital; Northern and Central Alberta Perinatal Program (NCAPP), Edmonton, Alberta, Canada.

The present study investigates the value of selected clinical signs in the prognosis of eventual ambulatory function of 74 children with cerebral palsy. The Neonatal Follow-up Clinic of the NCAPP examined 1083 high-risk children discharged from two tertiary NICUs from 1975-1979 inclusive. The children's diagnoses, milestones and primitive reflexes were recorded at intervals according to a prospective protocol from birth to age 8 by the second author. 74 children were diagnosed to have cerebral palsy. Their ambulatory function at age 8 was determined. Results indicated that 63.5% attained community ambulation (defined as ability to walk independently for 15 metres with or without orthoses and/or upper extremity walking aids), 6.8% were household ambulators and 29.7% were confined to a wheelchair. 100% of the children diagnosed to have spastic haemiplegia before the age of 2 became community ambulators, while 91.3% of spastic quadriplegic children were non-ambulators. 100% of the community ambulators could maintain handfree sitting prior to age 3 while none of the children who learned to sit after the age of 3 became ambulatory. Persistence of obligatory Moro reflex, symmetrical tonic neck reflex and assymmetrical tonic neck reflex beyond age 1 were statistically significant negative signs. Neck righting reflex and extensor thrust beyond age 1 were, contrary to previous reports, not statistically significant negative signs. Presence of placing and parachute reactions at age 1 were positively related to ambulatory outcome. Uncontrolled seizure and severe mental retardation also influenced ambulatory outcome. The combined use of these selected clinical signs in the prognosis of eventual ambulatory function resulted in improved predictive accuracy.

A9

NEONATAL BEHAVIOURAL ASSESSMENT OF CHINESE BABIES IN HONG KONG

L.Y. Ko-Yang, P.K.B. Miu-Lee.
Arran Street Child Assessment Clinic, Mongkok, Hong Kong.

The Brazelton Neonatal Behavioural Assessment Scale has been widely used in North America and elsewhere. For both research studies and clinical intervention. However, its application in the Asian setting is still limited.

The present study was a pilot attempt to examine the behaviour pattern of Chinese newborn babies in Hong Kong, using the Brazelton Scale. 39 infants were assessed on their 2nd and 4th days of life. The local profile was discussed and compared with a group of 54 infants in U.S.A. 9 infants with atypical behaviour pattern were noted.

A conjoint study on the experience of mothers during the prenatal period was also reported.

It is hoped that the present exploratory investigation will stimulate more local research in the field of human infancy.

EXPERIENCE OF CLINICAL GENETIC SERVICE IN HONG KONG

A.S. Chau.
Paediatric A Unit, Queen Elizabeth Hospital, Hong Kong.

The Clinical Genetic Service in Hong Kong was formally established since 1981. It has been working in close liaison with the prenatal diagnostic unit and other units concerning genetic disorders or mental retardation.

The service is composed of 2 sections:-

(A) The Genetic Counselling Clinics with back-up service of Cytogenetic Laboratory for chromosomal analysis. It offers advice and diagnosis on genetic problems. Totally 671 families attended the clinics in 1982, and 1,630 families in 1986; that is the workload increased by 2.4 folds. The cytogenetic section also increased by 2.2 folds in the diagnostic aspect.

(B) The Central Genetic Neonatal Screening Unit started to screen cord-blood specimens for congenital hypothyroidism and glucose-6-phosphate dehydrogenase deficiency (G6PD) in male babies in April 1984. During the period April 1984 to March 1987, there were 123,619 babies screened, 38 confirmed cases of congenital hypothyroid and treatment commenced before 3-week old. At the same time, 2,782 male babies were detected to be G6PD deficient among 63,988 male livebirths, making the incidence of 4.35%. In August 1986 to March 1987, when all female babies were screened for G6PD, 60 out of 14,000 females were found to be deficient, making the incidence among female being 0.43%.

B2

APPROACHES IN GENETIC METABOLIC DISEASES - LOCAL EXPERIENCE

S.T.S. Lam.
Clinical Genetic Service, Queen Elizabeth Hospital, Hong Kong.

The group of diseases known as inborn errors of metabolism is usually considered to be rare, and worthy of only small print in textbooks. This contention certainly does not apply to the more common of these disorders, such as deficiency of glucose-6- dehydrogenase. For the less common disorders, although individual diseases may be of low incidence, collectively they constitute a significant proportion of our genetic load. This paper is intended to summarise the approaches to early recognition and management of these diseases. For the purpose of diagnosis, the most important element is the clinical awareness of its existence, which on occasions would depend greatly on a high index of suspicion. Definitive diagnosis will rely on studies of the metabolites, protein products (including enzymes) and, or, the defective genes. Subsequent management includes the provision of genetic counselling, carrier detection and prenatal diagnosis to the family, and specific treatment to the affected individuals, which at times may prove to be difficult. These approaches will be exemplified by cases referred to the Clinical Genetic Service which includes defects in lysosomal enzymes, and disorders in the metabolism of carbohydrate, steroid, trace metals and others. Finally, the preventive approach to genetic metabolic diseases will be further elaborated in results from our efforts in selective and universal screening.

B3

TREATMENT OF HBsAg ASSOCIATED MEMBRANOUS NEPHROPATHY

E.C.L. Yu, C.B. Chow[1]
Department of Paediatrics, University of Hong Kong.
Paediatric A Unit, Princess Margaret Hospital[1], Hong Kong.

27 cases of membranous nephropathy presenting before 12 years of age were studied retrospectively. All were associated with HBsAg. 16 patients were given steroid therapy. 11 patients entered into remission of their nephrotic state after a course of therapy. The remainder were partly or completely resistant to treatment. Steroids may be useful to bring about an early remission in selected cases. The use of steroids in HBsAg individuals may however cause liver damage. Two patients not on steroid therapy were given a course of interferon. One patient went into remission. The proteinuria in the other case decreased. The prognosis of HBsAg membranous nephropathy is discussed.

B4

ANAEMIA IN HONG KONG ADOLESCENTS

A.M.C. Li, M.Y. Cheng, A.M.C. Yu.
Department of Paediatrics, University of Hong Kong.

From October 1986 to April 1987, we screened 484 healthy Chinese adolescents for anaemia. These students were from two local high schools and anaemia was defined as haemoglobin ‹13 gm/dl in boys, and ‹12 gm/dl in girls. Two millilitres of venous blood was taken from participants for the determination of haemoglobin, red cell count, haematocrit, red cell indices, serum ferritin levels and haemoglobin pattern when indicated. Participants were asked to fill in a simple dietary questionnaire, and relevant personal data were also obtained. Anaemia was found in fifteen students, giving an overall incidence of 3%. Of these fifteen students, six had iron deficiency anaemia, eight were diagnosed to have thalassaemia traits, while one had Haemoglobin H disease. Forty-one additional students showed no anaemia, but of these ten had depleted iron stores and thirty-one were found to carry either the α or ß-thalassaemia traits. The findings in the study suggested that thalassaemia traits are as common a cause of anaemia as iron deficiency in local high school students.

B5

BONE MARROW TRANSPLANTATION (BMT) IN PEDIATRIC PATIENTS

K.W. Chan, P.C.J. Rogers, S.L. Pritchard , C.J.H. Fryer.
Department of Paediatrics, The University of British Columbia and B.C.'S Children's Hospital, Vancouver, Canada.

Between December 1981 and June 1987, 30 children underwent allogenic BMT at B.C.'s Children's Hospital (22 histocompatible and 8 mismatched). Their ages ranged from 0 to 23 years (median 8 years). Indications for BMT were leukemia (17), neuroblastoma (4), severe aplastic anemia (6) and immunodeficiency syndromes (4). Thirteen patients (43%) are alive and free of disease at a median follow-up of 38 months. The figures for malignant and non malignant conditions are 39% and 67% respectively. There is no difference in survival between the histocompatible and the mismatched BMT. Major reasons for failure were recurrence of malignant diseases (6) and severe graft-vs-host disease (6). Standard procedures for BMT include isolation in positive-pressure rooms and low bacterial diet during the neutropenic phase, use of CMV-negative blood products and HLA-matched platelets, and intravenous gammaglobulin for prophylaxis. In the past 2 years there have been notable changes: 1) decrease in the number of BMTs performed for relapsed leukemics; 2) broader indications for the procedure in non-malignant disorders and pediatric solid tumours; 3) variations in preparative regimes; 4) increased usage of alternate sources of bone marrow, i.e., mismatched or unrelated marrow donors. There were also 9 BMTs performed with autologous bone marrow.

B6

A FOLLOW-UP STUDY ON 140 CASES OF HISTIOCYTOSIS X

Y.M. Hu, S.Y. Yang.
Beijing Children's Hospital, Beijing, People's Republic of China.

From 1956-1982, there were 171 patients with histiocytosis X admitted to Beijing Children's Hospital. According to their clinical, roentgenological and pathological findings, they were divided into 5 groups: (1) Letterer-Siwe syndrome 85 cases (about 50%); (2) Hand-Schuller-Christian syndrome 36 cases; (3) eosinophilic granuloma of bone 23 cases; (4) intermediate between the first two types 9 cases; (5) single organ involvement, all were conformed by biopsy or autopsy 5 cases. Besides those, there were 6 cases which were first diagnosed as eosinophilic granuloma but later developed into H.S.C. and 7 cases with very atypical symptoms could hardly be assigned to anyone of the 5 categories.

Among the 171 patients 140 cases were followed-up successfully.

The prognosis of histiocytosis X has changed greatly, since the introduction of chemotherapy. There were 57 cases whom didn't receive any treatment or were only treated with antibiotics, they presented as natural outcome of the disease, most of them were camed before 1973. After 1973 most of the patients were treated with short-term corticosteroids and after 1979 most of patients were treated with long-term combined chemotherapy for more than 3 months, the death rate declined to 27.1%.

Since 1982, 41 cases (not include in the 171 cases) have been treated with both chemotherapy and thymic extract, the death rate declined to 12.2%.

Beside the method of treatment, there were other prognostic factors, namely the number of involved organs, the severity of functional disorder of the organs and the age of the patient. When the disease occurred during infancy with multisystem involvement and marked functional derangement the outcome was very serious.

B7

COUMADIN THERAPY IN CHILDREN

M.Y. Cheng, V. Blanchette[1]
Department of Paediatrics, University of Hong Kong.
Hospital for Sick Children, Toronto[1]

The literature on the use of coumadin in children is lacking. It is everyone's impression that the prothrombin time can sometimes be unpredictable after what appears to be a reasonable dose of coumadin.

Twenty-nine children treated in a one year period were studied retrospectively to find out the loading dose, the maintenance dose, and the effect of drug interaction on the prothrombin time.

The majority of the children received a starting dose of 0.2 ± 0.05 mg/kg/day for the first day. No patient required more than 10 mg/day for more than 2 days. The total coumadin requirement in the first 3 days was 0.2 to 0.7 mg/kg.

A rough guide for the maintenance dose was 0.22 mg/kg for children <2 years old, 0.15 mg/kg for those 2-12 years old and 0.05 mg/kg for those >12 years old.

The effects of some of the drugs on the Prothrombin time of coumadinized children were also studied.

B8

PHYSICAL CONDITIONING PROGRAMME FOR ASTHMATIC CHILDREN - ITS EFFECT ON CARDIORESPIRATORY FUNCTION

O.K.W. Chow, R.N.S. Lo, M.H.H. Ip.
Department of Paediatrics, University of Hong Kong.

Exercise induces acute airway obstruction in the majority of asthmatic children and limits their physical exertion. This leads to poor participation in games and low self-esteem, hindering normal development of the individual. To promote physical fitness and interest in sports, a physical conditioning programme was organized, with 12 days intensive wet and dry land exercise at Jubilee Sports Centre, followed by 3 months of weekly swimming sessions at the MacLehose Rehabilitation Centre.

Activities were preceded by warm-up exercises and interspersed with rest. 12 asthmatic children, aged 9 - 14 years, with demonstrable exercise induced broncho-constriction and spontaneous variation in FEV_1 of > 15% were enrolled. They continued their usual medication throughout the programme. Premedication with bronchodilators or sodium cromoglycate was allowed before the training. Prior to and after the programme, their cardiorespiratory status was assessed by the standard exercise test with Medtronic IMC compustress treadmill, Gould CPI 5000 IV computerised pulmonary function laboratory, 24 hours Holter ECG recording with Oxford Medilog 4500, and echocardiography MK 600 mechanical section scanner. Throughout the programme, the respiratory function was monitored by MiniWright peak flow meter. All tolerated the programme well without development of bronchospasm. 4 showed slower basic heart rate after training, while 5 showed improved lung volumes/flow rates. There was no change in left ventricular function, and no amelioration of the exercise induced bronchospasm.

The programme had beneficial effects on the asthmatic children, and was well received by both children and parents. With precautions, asthmatic patients should be able to participate in different kinds of sports and should be encouraged to do so.

LOCAL EXPERIENCE OF HEPATITIS B VACCINATION IN NEWBORNS OF HBsAg CARRIER MOTHERS - THE OPTIMAL DOSE SCHEDULE AND TIMING

B. W. Y. Young
Paediatric A Unit, Queen Elizabeth Hospital, Hong Kong.

Aim: To evaluate the immunogenicity and efficacy of different combinations of HBIG-HBV vaccine schedules in preventing the HBsAg carrier state in infants born to HBsAg positive mothers.

Materials and Methods: Sera from 13,594 pregnant women were screened for the presence of HBsAg. 1,409 (10.4%) were positive for HBsAg, of which 42.1% were positive for HBeAg. 556 healthy babies born to these HBsAg positive women were randomised to 3 HGIB-HBV vaccine schedules.

	GPI	GP II	GP III
HBIG (0.5 ml)	0,2 mon	0,2,4 mo	0,2 mon
HBV vaccine (10 ug)	0,1,6 mon	2,3,8 mon	0,1,2 mon

Results: During the 3 years of follow-up, 21 infants became HBsAg positive and all were born to HBeAg positive mothers. The cumulative incidence rate ranged from 8.5 to 9.9% and the protective efficacy rate from 86.4 to 88.4% among the subgroup of HBeAg positive mothers. Anti-HBs seroconversion after 3 doses of vaccine was found in more than 92.4% of infants. Vaccination commenced at 2 months achieved significantly higher anti-HBs titre after 2 doses of vaccine and at 1,2 and 3 years. The number of infants who were still anti-HBs positive at 3 years were also significantly higher in this group of infants.

Conclusion: The three HBIG-HBV vaccine regimens are equally efficacious in preventing HBV infections and its carrier state. HBV vaccine commenced at 2 months after birth, when compared with vaccine commenced at birth, produces a higher antibody response and a significantly higher proportion of these infants remain anti-HBs positive at 3 years. Vaccination given in a schedule which is completed by 2 months, when compared to 6 months of age, produces similar anti-HBs conversion and geometric mean titre.

C2

THE EFFECT OF RECOMBINANT α_2 INTERFERON IN CHINESE HBsAg CARRIER CHILDREN

C.L. Lai, A.S.F. Lok, H.J. Lin[1], E.K. Yeoh[2], and C.Y. Yeung[3].
Departments of Medicine, Pathology[1] and Paediatrics[3], University of Hong Kong.
Queen Elizabeth Hospital[2], Hong Kong.

Since most Chinese acquire their chronic hepatitis B carriage at birth or in early childhood, interferon (IFN) may be more effective in suppressing viral replication when given to HBsAg carrier children than to carrier adults.

This study includes 24 Chinese children between the ages of 1.5-5 years. They were positive for HBsAg, HBeAg, HBV DNAp and HBV DNA for over 6 months on at least 3 occasions. Twelve children each were randomised to receive either placebo (vitamin B complex) or IFN (Roche) $10x10^6$ IU/m^2 IMI thrice weekly for 12 weeks. Sera were checked for HSsAg/anti-HBs, HBeAg/anti-HBe, HBV DNAp, HBV DNA, liver function tests and blood counts weekly for 4 weeks, then monthly for 4 months and then 2-3 monthly for one more year.

Results All 24 subjects had been followed up for 18 months

	IFN	Placebo
Median age in years	3.5	3.5
Male : female	5:7	7:5
Side-effects		
– transient 'flu'	12	0
– fatigue	1	0
Decreased HBV DNAp and HBV DNA		
– transient	10	0
– sustained	2	0
Loss of HBeAg	1	1

Conclusions IFN is well tolerated in children. It induces significant transient decrease in HBV DNAp and HBV DNA, but up to 18 months after the start of the trial, there is no evidence of a long-term beneficial effect with a 12-week course.

C3

ENDEMIC GIARDIASIS IN A HONG KONG NURSERY

G.M. Samuda, C.Y. Yeung, F.T. Lee.
Department of Paediatrics, University of Hong Kong.

Day care centres (DCCs) have been recognised as a unique ecological environment for the young child. Previous reports have shown that children who attend DCCs experience an increased incidence of both bacterial and viral respiratory tract infections, diarrhoeal disease, hepatitis A, cytomegalovirus disease, tuberculosis, and other vaccine-preventable disease.

The identification of several infants with failure to thrive, as a probable consequence of giardiasis, prompted further study of a local DCC in which they were enrolled. Two prevalence studies for Giardia lamblia infection were conducted, separated by a fifteen month interval. During each study, serial stool specimens for each child were collected and studied using both direct wet mounts and a formaldehyde-ether preparation. Results were later correlated with clinical and anthropological data.

Giardia cysts were identified in stool specimens from 29% of the children in the first study and 23% in the 2nd study. Two staff members were also positive.

In this nursery, it appeared that both staff and long term resident children provided a reservoir to infect new entrants to the nursery. Relative crowding, preparation of food, and nappy changing by the same staff members were contributing factors.

Guidelines exist in Hong Kong for the management of children attending DCCs. Staff and inspectors of DCCs as well as parents and physicians must have a clear understanding of the inherent special problems at DCCs and take steps to develop an effective liaison.

C4

NOSOCOMIAL GASTROENTERITIS IN PAEDIATRIC PATIENTS

B. Lam, J. Tam[1], M.H. Ng[1], C.Y. Yeung.
Departments of Paediatrics and Microbiology[1], University of Hong Kong.

Between November 1982 and April 1985, 2228 children under the age of 5 with acute gastroenteritis were admitted to the Paediatric ward of Queen Mary Hospital, Department of Paediatrics, University of Hong Kong. Pathogens investigated include vibrio species, salmonella species, Campylobacter fetus jejuni, shigella species, yersinia, enterotoxigenic and enteropathogenic E Coli. Rotavirus was detected by enzyme-linked immunosorbent assays (ELISA) provided by WHO. 56.2% were identified as infection by a single pathogen or a combination of pathogens. Patients were diagnosed to have acquired nosocomial infection if a pathogen was identified in stool specimens more than 3 days after admission but was not identified before this time. A total number of 163 cases (13.4% of all positive cases) were identified as nosocomial infections. Rotavirus was the predominent agent for nosocomial infection, accounting for 128 cases (20% of all rotavirus infection), while bacteria account for 35 cases (7% of all bacteria positive cases). Nosocomial rotavirus infection occurred primarily during the winter months and corresponded with the seasonal distribution of rotavirus cases. Electropherotyping of the rotavirus by RNA polyacrylamide gel electrophoresis demonstrated that the electropherotypes involved were those prevalent in the hospital at the time. Among these, 44 patients had mixed or sequential infection by 2 or more pathogens (they had positive identification of enteric pathogen from initial stool specimen on admission and subsequently developed secondary enteric infection during the course of hospitalization).

C5

FACTORS AFFECTING FATTY ACID ABSORPTION FROM INFANT FORMULAE

G.M. Kneebone.
Department of Paediatrics, Flinders Medical School, Flinders Medical Centre, Bedford Park, South Australia.

Infant formulae contain similar amounts of calcium and phosphorus and have a fatty acid composition that meets the stated nutritional requirements for infants. It is assumed that the bioavailability of these fatty acids is essentially similar and adequate.

The calcium/phosphorus (Ca/P) ratio of commonly used infant formulae in Australia varies from 1:43:1 to 1:29:1 and the individual fatty acids make up variable percentages of the total fatty acid contents of the formulae.

Fat balance studies in infants show that there are differences in individual fatty acid absorption that are related to the Ca/P ratio of the formula and additionally that essentially "identical" formulae Nan, S26 have differences in individual fatty acid absorption that are independent of the Ca/P ratio.

The importance of these differences in bioavailability of saturated and unsaturated fatty acids has nutritional and commercial significance in infant nutrition.

C6

ACUTE VIRAL HEPATITIS IN CHILDREN

T.T.Y. Lau, C.B. Chow, N.K. Leung.
Paediatric A unit, Princess Margaret Hospital, Hong Kong.

Over a period of three and a half years, 348 consecutive children (205 males, 143 females, aged 3 months to 12 years) with acute viral hepatitis, were studied. Hepatitis A (HA) was the most frequent aetiologic type. It occurred in 81% of the cases. Non-A non-B hepatitis (NANB) occurred in 10% of the patients. Hepatitis B (HB) occurred in 8% of the cases. All the HA and HB cases recovered completely within 1 year. Chronicity was found in one NANB patient. Three patients (1 HB, 2 NANB) died of fulminant hepatitis. HBsAg clearance was fast. By 6 months, 93% of patients had cleared the antigen and 79% had seroconverted. Chronic HBsAg carrier state occurred in 1 patient (3%) by one year.

C7

CHILDHOOD TUBERCULOSIS IN HONG KONG IN THE 1980s

M.K. Sham, M.J. Humphries & M. Gabriel.
Ruttonjee Sanatorium, Hong Kong.

In the 7-year period from 1980 to 1986 inclusive, 301 children (aged less than 15 years) have been treated at the Ruttonjee Sanatorium for respiratory tuberculosis. The clinical presentations and characteristics of disease will be presented, and a comparison made with the features of childhood tuberculosis in other areas of the world.

C8

PARALLEL DEFECT IN TYPE I IGF AND INSULIN RECEPTORS IN TWO PATIENTS WITH ACANTHOSIS NIGRICANS (AN) AND INSULIN RESISTANCE TYPE A

L.C.K Low, M.A. Sperling.
Department of Paediatrics, University of Hong Kong.
Endocrine Division, Children Hospital Medical Center, Cincinnati.

A subset of patients with AN and insulin resistance type A is characterized by hyperinsulinaemia (HI), hyperandrogenaemia, oligomenorrhoea and acral hypertrophy. We investigated the hypothesis that HI cross-reacting with an intact type I IGF receptor being responsible for the acral hypertrophy in 2 patients, by studying the receptor binding, insulin and IGF-I mediated biological responses and autophosphorylation of the B subunit of their receptors. Insulin binding to dermal fibroblasts in culture was reduced by 50% as a result of decreased receptor numbers in one patient and decreased receptor affinity in the other. IGF-I binding was reduced by 50% because of decreased receptor numbers in both patients. Insulin stimulated ^{14}C-glucose uptake, insulin and IGF-I stimulated ^{3}H-thymidine uptake into fibroblasts were proportionately decreased in both patients. In parallel to the defects observed in binding and hormone-stimulated biological responses, IGF-I and insulin mediated autophosphorylation of the B subunit of solubilized WGA purified receptors prepared from cultured fibroblasts was also reduced by 40% to 60%.

C9

PREVALENCE AND SIGNIFICANCE OF MILD BLEEDING DISORDERS IN CHILDREN WITH RECURRENT EPISTAXIS

K.H. Luke
Children Hospital Eastern Ontario University of Ottawa, Canada

Recurrent nose bleeding is a common clinical problem in children. Previous studies in these children using standard coagulation tests did not reveal any significant correlation to bleeding disorders. With the recent advances in the pathophysiology of coagulation and the availability of new tests to document these pathways (particularly the platelet/endothelial functional defects and Von-Willenbrandt Disease Variants), the question reopens as to the prevalence and significance of bleeding defects in these children. We have studied on a perspective basis 36 children with recurrent nose bleeding (more than five episodes per year). A detail "Bleeding/Family history"; Coagulation screening tests (PT; PTT; Platelets and B.T.) and specific tests for Factor VIII Coagulation Activity (VIIIc); Von Willebrandt Factor Antigen (VMF. Ag), Ristocetin Co-Factor (VIII RiCoF) and V.M.F. Multimers were done in each child. 35 days care surgery children without any bleeding history serve as control. Basing on the history, a scoring system (0 to 2) for clinical severity of nose bleeding was assigned to each of five criteria — frequency; site; duration; amount and number of years of bleeding. Children with a score below 6 were designated group A (mild) and above 6, group B (severe).

Results: Among 36 children, 24 belong to group A and 12 group B. Significantly the group B children showed higher incident of Anemia (33%), Iron deficiency (50%) and bleeding time abnormalities (42%). 2 cases of V.M. disease were diagnosed from the group B children with abnormalities in the V.M.F. antigen level and platelet-restocetin co-factor. One of them showed decrease of V.M. Multimers. No case of structural defects of V.M. Factor multimers were seen in the group A or B children. No coagulation defects were detected in group A.

Our studies suggest:—

(1) The Clinical Scoring System is helpful to document severity of bleeding and select cases for specific testings.
(2) B.T. is the most useful screening tests but specific tests are required to document V.M. defect.
(3) Prevalence of V.M. Defect is 17% in group B giving a relative high yield in diagnosis.

CONGENITAL MESOBLASTIC NEPHROMA - A BENIGN TUMOUR THAT USUALLY DOES NOT REQUIRE CHEMOTHERAPY

M.Y. Cheng, H.S.L. Chan[1].
Department of Paediatrics, University of Hong Kong.
Division of Haematology & Oncology, Hospital for Sick Children, Toronto[1].

Congenital mesoblastic nephroma (CMN) is a benign renal tumour, distinctly different from Wilm's tumour.

This report describes the presentation of a 2-month old girl with CMN, referred to Queen Mary Hospital, Hong Kong for chemotherapy.

A retrospective review was made of 17 cases of CMN seen at the Hospital for Sick Children in Toronto. The majority of the children were younger than 3 months of age. The most consistent clinical feature was a unilateral hard to firm renal mass. Hypertension was very common. Other features included : material polyhydramnios (2 patients), major genitourinary anomalies (2 patients), transient azotaemia (2 patients), proteinuria (3 patients), haematuria (1 patient), anaemia (1 patient), thrombocytopenia (2 patients), hypocalcaemia (1 patient), hyperkalaemia (1 patient), hypokalaemia (1 patient). A distinctive sonographic 'ring-sign' was recorded in 4 patients. The 17 patients had a tumour histology that fitted into the pathologic spectrum of CMN proposed by Beckwith. All 17 patients were alive with no tumour recurrence at a mean follow up duration of 10.3 years. Nephrectomy was adequate therapy for most patients. Chemotherapy and/or radiation was indicated after nephrectomy for: (a) patients older than 3 months with grossly unresected tumours, (b) tumours with uneqvivocally malignant histology, or (c) tumours with aggressive biologic behaviour such as gross extension, local recurrence or metastases.

P2

A REVIEW OF THE CLINICAL PRESENTATION OF 111 CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKAEMIA IN HONG KONG

M.Y. Cheng, A.M.C. Li, G.M. Samuda.
Department of Paediatrics, University of Hong Kong.

One hundred and eleven children below 12 years of age with acute lymphoblastic leukaemia (ALL) were diagnosed at the University Paediatric Unit, Queen Mary Hospital, Hong Kong over a period of 10 years from 1976 to 1985. A retrospective analysis was made with respect to the pattern of this disease at presentation. The children were further divided into standard risk and high risk groups, based on their age at presentation, initial total white cell count, 'lymphoma' syndrome, sanctuary disease and B cell disease. An attempt was made to identify a subgroup with very high risk ALL.

The most common first presenting complaint included fever, pallor, bone pain, respiratory tract infection symptoms and bleeding. Two unusual initial presenting features were noted in this group of children, namely puffy eyes or face (5%) and abdominal distension (3%). Three other children were wrongly diagnosed and treated for tuberculosis before referral. The mean time from onset of the first symptom to the diagnosis of ALL was 6 weeks (range: 1 day to 6 months). There was a striking preponderance of males in this group, with 85% more males than females. Forty-six percent of the children belonged to the standard risk group and 54% to the high risk group.

The overall induction success rate was 86%. The mortality approached 100%in children with (a) B cell ALL, (b) infants with ALL, (c) high risk ALL and children older than 10 years of age.

P3

A 3 1/2 YEAR OLD WITH WILSON'S DISEASE - THE YOUNGEST RECORDED IN THE ENGLISH LITERATURE

M.Y. Cheng, J.M. Seidman[1]
Department of Paediatrics, University of Hong Kong.
Hospital for Sick Children, Toronto[1]

A 3 1/2 year old girl of Pakistani origin presented to the Hospital for Sick Children in December 1985 with a 7 day history of pallor and jaundice. The parents were first cousins and a younger brother in the family was in good general health. Clinical findings confirmed the presence of acute haemolysis and hepatocellular damage. Ophthalmological examination showed Kaiser-Fleisher rings. The serum copper was 44.1 umol/l (normal 10.5 - 23 umol/l), ceruloplasmin 307 mg/l (normal 180 - 450 mg/l), urinary copper 9.4 umol/l (normal less than 0.6 umol/l).

This patient is the youngest child ever reported in which an acute haemolysis was the presenting feature of Wilson's Disease. Failure to make the diagnosis especially when it presents with haemolysis in young children is all too common. Normal serum ceruloplasmin in such cases is a common source of false reassurance. It is imperative that physicians assessing a child with unexplained haemolytic anaemia should consider Wilson's Disease. The serum ceruloplasmin may be normal or elevated particularly in children presenting with haemolysis.

P4

TYPHOID FEVER IN HONG KONG CHILDREN

C.B. Chow, P.S. Wang, N.K. Leung.
Paediatric A Unit, Princess Margaret Hospital, Hong Kong.

The experience with typhoid fever in 111 children over a 5 year period was reviewed. There were 66 boys and 45 girls. Their ages ranged from 1 to 11.5 years. The symptoms of typhoid fever were quite non-specific. Fever was present in 98.3%. Other presenting features were diarrhoea (25.7%), constipation (22%) vomiting (21.1%), cough (25%), abdominal pain (27.5%), headache (9.2%), epistaxis, meningism and convulsion. Rose spot was detected in 20% of cases. The white cell counts were not found to be of great diagnostic value. Significant Widal reaction was present in 84.7% of cases. Blood and stool cultures were positive in 57% and 44% of cases respectively. Chloramphenicol remained the drug of choice in the treatment of typhoid fever. It was more effective compared with ampicillin or co-trimoxazole. 80% of patients were treated within the first 2 weeks of the illness. Complications were uncommon and were present in 2 patients. There were 2 deaths; both were admitted late in a moribund state. Early diagnosis and treatment is vital in typhoid fever and, as the presenting features are non-specific, a high index of suspicion is required.

P5

THE PREVALENCE OF SMALL-FOR-GESTATIONAL-AGE(SGA) INFANT IN SOUTH CHINA. PART I: THE INCIDENCE

Z.K. Feng.
South China SGA Study Group, Medical College, Jinan University, Guangzhou, People's Republic of China.

With the objective of investigating for the first time the situation of the SGA problem in South China, the study was conducted as one project in 24 hospitals of 10 cities in five provinces and one autonomous region in South China during the period between March 1, 1986 and Feb. 28, 1987. SGA is defined as "birth weight(BW) under the 10th percentile(P10)". The standards used are the data collected in the same area in 1985 and published in 1986, instead of those of Luchenco's which, as Paper and Fitzhardinge have warned, "should be used with caution as they are based on a mixed population of Mexican, Indian and Caucasian origins living at high altitudes and potentially subject to chronic hypoxemia". The incidence of SGA in Guangdong, Sichuan, Gueizhou and Hunan provinces is 8.13 - 8.69% with a male to female ratio 1 : 1.56 - 1.74 (data from Fujian and Guangxi are not completed yet). The incidence of low-birth-weight (LBW, i.e., BW < 2500 gm) in South China is 6.44% and the ratio of SGA to LBW is 61.48%. This high ratio, together with the high incidence of SGA witnesses that the SGA problem (at least in this part of the country) is deserved to be studied further.

P6

THE RELATIONSHIP BETWEEN PLATELET FUNCTION, VITAMIN E, VASCULAR REACTIVITY IN DIABETIC CHILDREN

G.M. Kneebone.
Department of Paediatrics, Flinders Medical School, Flinders Medical Centre, Bedford Park, South Australia.

Experimental diabetes shows a relation between lowered Vitamin E levels and platelet dysfunction. Vascular endothelial disturbance and platelet dysfunction appear to be a major cause of diabetic vascular disorders.

A study of diabetic children has shown significantly higher levels of plasma Vitamin E in these children plus differences in platelet and plasma phospholipid fatty acid levels in linoleic (18:2), arachidonic (20:4) and other plasma polyunsaturated fatty acid levels in diabetic children.

Platelet aggregation studies show significant differences between diabetic and control children.

Transcutaneous oximetry measurements also show impaired responses to vascular occlusion in diabetic children before classical evidence of vascular complications is present.

The abnormalities of platelet function, vascular reactivity and Vitamin E status in diabetic children are present very early in the course of the disease and before recognisable complications become evident.

These findings suggest that these abnormalities may be evident at the onset of the clinical disease of diabetes in childhood.

P7

MONGOLIAN SPOTS IN CHINESE CHILDREN

A.K.C. Leung.
Department of Pediatrics, Alberta Children's Hospital, University of Calgary, Calgary, Alberta.

Ninety two Chinese Canadian newborn infants (49 males and 43 females) and 1,633 Chinese Canadian children (819 males and 814 females) in Calgary, Alberta, Canada were examined for the presence of Mongolian spots. Mongolian spots were present in all newborns and disappeared slowly until 6 years of age when the rate of disappearance increased. At 10 years of age, none were found. The overall incidence regardless of age was 58% in boys and 53.3% in girls. The most frequent site of involvement was the sacrococcygeal area, followed by the gluteal and lumbar area. Both sides were equally affected. In only 7.8% of boys and 3.3% of girls was the involved area greater than 15% of their body surface area. Most (63.8% of boys and 67.4% of girls) had less than 5% involved. The colour of the Mongolian spots varied from gray, grayish blue to grayish black. In general, younger children had darker Mongolian spots.

P8

DOUBLE-BLIND, RANDOMIZED, MULTI-CENTRE TRIAL ON THE EFFICACY AND SAFETY OF TWO ORAL SOLUTIONS AS MAINTENANCE THERAPY OF ACUTE DIARRHEA

A.K.C. Leung, L. Geoffroy, P.G. Taylor, P. Darling.
Department of Pediatrics, Alberta Children's Hospital, University of Calgary, Alberta.
Department of Pediatrics, Hospital Ste-Justine, University of Montreal, Quebec.
Department of Pediatrics, Misericordia Hospital, University of Alberta, Alberta.
Abbott/Ross Laboratories, Montreal, Canada.

Current recommendations for oral fluid therapy in a developed country include the use of two oral solutions, one intended for rehydration (ORS), the other intended for maintenance of hydration (OMS). There is concern that the inappropriate use of these solutions may lead to fluid and electrolyte imbalance. Hypernatraemia may for instance result from the use of a higher sodium containing ORS as maintenance fluid therapy in infants with acute diarrhea and $< 5\%$ dehydration. In order to assess the safety and efficacy of available ORS and OMS, 52 infants 3-24 months of age, with acute diarrhea and $< 5\%$ dehydration were randomized in a double-blind fashion to receive either ORS or OMS which differed only in sodium concentration (75 mEq/L vs 45 mEq/L respectively). There was no significant difference in mean $\pm$ SD volume of fluid ingested ad libitum between the ORS (171.7 $\pm$ 13.3 ml/kg/d) and OMS (160.6 $\pm$ 11.8 ml/kg/d) groups. The mean serum sodium levels of the two groups did not differ significantly ($p > 0.05$) from each other at admission or at the end of 24 hours of oral fluid therapy. There was, however, a significant rise in serum sodium concentration ($+ 2.1 \pm 0.5$ mEq/L, $p < 0.001$) within the ORS group which was not significantly apparent in the OMS group ($+ 0.83 \pm 0.6$ mEq/L, $p=0.2$) and which may not be of clinical relevance. There was no incidence of hypernatremia. All patients remained well hydrated. These data suggest that an oral solution containing 75 mEq/L of sodium recommended for rehydration (ORS) is as safe and effective as a maintenance solution (OMS) when used in infants and children over 3 months of age with acute diarrhea.

P9

ECHOCARDIOGRAPHIC ASSESSMENT OF NEONATES WITH PULMONARY ATRESIA AND AN INTACT VENTRICULAR SEPTUM (PA + IVS)

M.P. Leung, P.W. Hui, C.K. Mok[1], E. Tam, K.C. Lau, R.N.S. Lo.
Departments of Paediatrics & Surgery[1], University of Hong Kong.

The present study compared the efficacy of cross-sectional echocardiography (CSE) and angiography in defining the right ventricular (RV) morphology and associated RV anomalies in neonates with PA + IVS. 24 neonates were studied. A combination of echo- cardiographic views were used to visualize the RV morphology. The findings were compared with the anteroposterior and lateral right ventriculograms. A correct diagnosis was established by CSE and angiography in all 24 neonates. Based on the tripartite approach, the RV morphology was equally well defined by CSE and angiography. 12 babies had a tripartite RV, 9 had a 2-component RV (8 with absent trabecular and 1 with absent infundibular portion), and 3 had a diminutive ventricle with an inlet only. Ebstein's malformation of the tricuspid valve was identified by both methods of investigation in 2 babies. Among the 5 babies with angiographic evidence of coronary arterial-sinusoidal communications, the fistulae could be visualized by CSE in 3 babies only. The echocardiographic dimensions of the tricuspid annulus (4-chamber view), infundibulum and main pulmonary artery (parasternal short axis view) by CSE correlated well with angiographic measurements ($r > 0.8$). Thus CSE allows satisfactory assessment of the RV morphology and associated RV anomalies in PA + IVS.

P10

PATTERNS OF CHROMOSOMAL ABNORMALITIES IN HONG KONG

A.M.C. Li, F.T. Lee.
Department of Paediatrics, University of Hong Kong.

From August 1978 to May 1987, the cytogenetic service in this department had carried out chromosomal analysis on 1830 individuals. Abnormal karyotypes were found in 404; of these, 255 (63.1%) were trisomy 21, including 15 who had translocations. 45 other patients had either 18-trisomy syndrome (Edward's syndrome), 13-trisomy syndrome (Patau's syndrome), or Cri-du-chat syndrome. Another 26 patients had sex chromosome abnormalities with Klinefelter's syndrome and Turner's syndrome as the major groups. The rest had other abnormalities in their karyotypes. The general pattern of chromosomal abnormalities in Hong Kong is similar to that reported from other communities.

P11

CHILDHOOD CANCER: ITS IMPACT ON THE FAMILIES (LOCAL REPORT)

A.M.C. Li, T.L. Que, G.S.C. Chui, G.M. Samuda.
Department of Paediatrics, University of Hong Kong.

Family members of thirteen children receiving treatment for cancer in the Paediatric Department, Hong Kong University were interviewed with the view to define the problems they encounter. Data collection included background information on the family, parents' knowledge of the disease, their sources of information, their reaction to the diagnosis, and other specific problems that arose because of the child's illness. Information obtained confirmed the extensive psychosocial impact of the illness on the patient and his entire family, which is similar to findings reported by overseas workers. Suggestions on ways to tackle these problems are briefly discussed.

P12

THE PATTERN OF PAEDIATRIC BACTERIAL MENINGITIS IN QUEEN MARY HOSPITAL

C.H. Li.
Department of Paediatrics, University of Hong Kong.

A retrospective study of 57 patients with bacterial meningitis was carried out over a five year period (1981-1985). Their data were compared with those in the Report of the Task Force on Diagnosis and Management of Meningitis. Similarities were noted in the age specific incidences, predisposing factors, major presenting features and most of the laboratory findings.

However, there were some notable dissimilarities. Gram negative organisms were rare in the neonatal group and Neisseria meningitidis was also rare in both age groups. The variety of organisms responsible was of interest, notably salmonella species, pseudomonas, tuberculosis and cryptococcus.

2 patients had blood and CSF positive for group B streptococcus, but in these patients the CSF counterimmunoelectrophoresis (CIE) test failed to detect their corresponding antigens.

This study supports the belief that the latex agglutination test is superior to the CIE method in screening for bacterial antigens in the CSF. The present system of following up patients also needs urgent review as currently some patients defaulting follow up may be missed. More formal hearing tests and developmental assessments should be requested when patients are followed up.

P13

THE TREND OF POSTINFECTIOUS NEPHRITIS

C.H. Li, E.C.L. Yu.
Department of Paediatrics, University of Hong Kong.

228 children with acute nephritis admitted into Queen Mary Hospital between 1981 and 1986 were studied. Secondary nephritis, including Henoch-Schonlein nephritis and lupus nephritis, was excluded. 155 patients had poststreptococcal nephritis (PSAGN) confirmed by an increased streptococcal titre, while in 73 patients no cause was found and they were diagnosed as idiopathic (iAGN).

In both PSAGN and iAGN, boys were more commonly affected. PSAGN was particulary common between 7 to 10 years of age while iAGN patients did not show any age clustering. A falling incidence of admission was seen in PSAGN but not in iAGN. PSAGN showed a seasonal incidence, with most cases admitted between December and February throughout the 6 years. In 43% of PSAGN and 45% of iAGN upper respiratory symptoms were present. Sore throat, however, was noted in 50% of PSAGN but in only 30% of iAGN.

20% of all patients presented with nephritic-nephrotic features. In PSAGN patients with purely nephritic features, serum complement C3 was depressed in 82%, while all those with nephritic-nephrotic features had depressed C3 levels. Despite the falling incidence of PSAGN, renal failure is still frequent.

P14

ORAL REHYDRATION THERAPY USING RICE POWDER-ELECTROLYTE SOLUTION

G.M. Samuda, B. Lam, C.Y. Yeung.
Department of Paediatrics, University of Hong Kong.

In the management of gastroenteritis 'resting the gut' is now known to be non physiological and may in fact delay recovery. Many recent studies have shown Oral Rehydration Therapy and the introduction of early feeding to be more effective than traditional forms of therapy for most children. Oral therapy has now been widely accepted in such different settings as North America and Bangladesh.

A study was conducted at Queen Mary Hospital to study both the optimal form of Oral Rehydration Solution (ORS) and early feeding regime for our children.

At admission children were randomized to receive one of two ORSs (shown in the table). Six hours after admission, when rehydration was complete, the children were further randomized to receive either a lactose free milk or a lactose containing milk. The volumes of fluid offered and the method of introduction of milk was according to the WHO recommendations. The children were discharged when stools were no longer watery and the randomized milk was tolerated at full strength.

Eighty-seven consecutive children, admitted with gastroenteritis, entered the study. The age range was one month to two years (mean age 10 months). Eighty-five percent had mild dehydration and 15 percent had moderate dehydration.

Statistical analysis of clinical and laboratory data showed both ORSs to be equally effective. There was a significant difference ($P < 0.05$) however between the length of illness and duration of hospital stay when the two milk groups were compared. The children receiving the lactose free milk stayed in hospital 1.5 days shorter than those who received lactose containing milk.

No children who entered the study required intravenous therapy. ORS and the early feeding regime was effective in all cases.

In view of the advantages of Oral Rehydration Therapy we have now adopted a rice-electrolyte solution for use at Queen Mary Hospital.

Table: Constituents of Oral Rehydration Solutions

	Solution A	Solution B
Na (mmol/1)	50	50
K (mmol/1)	20	20
Cl (mmol/1)	40	40
Citrate (mmol/1)	10	10
	Rice powder 50 gm/l	Glucose 20 gm/l

P15

ARTERIAL BLOOD PRESSURE MEASUREMENT IN CRITICALLY ILL PRETERM NEONATES

A.Y.C. Tam.
Department of Paediatrics, University of Hong Kong.

Arterial blood pressure measured by an automatic oscillometric blood pressure monitor (DINAMAP 847 XT, Critikon, U.S.A.) in a lower limb was compared simultaneously with that measured via an indwelling umbilical catheter with its tip placed at L3 to L4 level in 8 critically ill premature neonates. The mean birth weight was 1.15 + 0.34 kg (+ S.D.) with a range of 0.66 - 1.62 kg. The mean gestational age was 29.23 + 3.14 weeks (+ S.D.) with a range of 26.57 - 35.86 weeks. All babies were on artificial ventilation. Four had respiratory distress syndrome, 2 had congenital pneumonia, 1 had persistent foetal circulation and 1 cyanotic congenital heart disease. The measurements were made between day 1 and day 11 of age. A total of 151 pairs of readings were made. The systolic pressure (SAP) measured by the oscillometric method (y) was found to be related to that measured via umbilical catheterization (x) by the equation $y = 0.67x + 19.82$, as analysed by linear regression. The correlation co-efficient (r) was 0.77, $p < 0.001$. However, only 48.0% of oscillometric measurements of the SAP had an error of 0-5 mmHg, while 27% of the readings had an error of more than 10 mmHg. Automatic oscillometric blood pressure measurements in critically ill preterm neonates must be interpreted with caution.

P16

NEONATAL LEUKOCYTE FUNCTION STUDIES

A.Y.C. Tam, S.K. Wan, C.Y. Yeung.
Department of Paediatrics, University of Hong Kong.

The phagocytic and bacterial killing functions of neonatal leukocytes were studied. The phagocytosis index and candida killing activities in term and preterm infants were delineated.

We have found that the Chinese preterm infants possess similar phagocytosis and candida killing activities compared to their term counterparts.

Several severe G6PD deficient infant boys were also studied. They did not show any impaired function.

P17

LEAD POISONING IN INFANCY

K.H. Tam, E.C.L. YU.
Department of Paediatrics, University of Hong Kong.

Between 1985 and 1987 two infants each 2-months old were admitted with lead encephalopathy. Both their parents were fishermen but the families lived on the land. Both children presented with status epilepticus and pallor. In one child, the convulsion persisted and it was necessary to use intravenous diazepam and phenobarbitone. The increased protein in the cerebrospinal fluid, with no cells and a normal CSF sugar in an afebrile child, raised the suspicion of lead poisoning. This was confirmed by a markedly elevated blood lead level and the dense metaphyseal lines on the radiographs. The second child was treated with intravenous phenobarbitone which stopped the convulsion. The cerebrospinal fluid revealed a high protein level, with 22/ul white cells (53% lymphocytes and 47% mononuclear cells). X-rays showed dense metaphyseal lines. The blood smear showed typical basophilic stippling.

The source of lead poisoning in the first child was a herbal powder mixture applied to the buccal mucosa as treatment for frequent regurgitation. The assayed lead content was 23.3% by weight and the leachable lead content was 15.6%. The child was estimated to be taking 62.4 mg of lead per week. The source of lead for the second child was believed to be related to the fishing environment. After treatment, the first child had delayed development. The second child had a high pitched cry and spasticity immediately after treatment. Lead poisoning still occurs in Hong Kong. A high index of suspicion is needed.

P18

FALLOT'S TETRALOGY: 10 YEARS' EXPERIENCE IN HONG KONG

P.S. Tang, K.C. Lau, C.K. Mok[1], R.N.S. Lo, M.P. Leung.
Departments of Paediatrics & Surgery[1], University of Hong Kong.

Between 1977-1986, 283 consecutive cases of Fallot's tetralogy were operated on in the Grantham Hospital. 10% had no cyanosis. Mild cyanosis (arterial saturation more than 80%), moderate cyanosis and severe cyanosis were present in 46%, 38% and 6% respectively. The mean age of onset of cyanosis was 2.7 years. 34.6% already had cyanosis soon after birth, and by 2 years, 88% had developed cyanosis. 56 had cyanotic spells and 4 sustained significant neurological sequelae. More than 60% of the spells started before 2 years of age and $>$ 90% before 4 years. 4 had cerebral abscess and 2 had infectious endocarditis before total surgical repair started. The overall surgical mortality was 8.7%. It was highest when correction was done below the age of 6 months(40%) and it fell to around 7% when correction was done after 3 years of age. 14(5%) had a prior palliative procedure and one of them subsequently died at total correction. Transannular patching of the right ventricular outflow tract was required in 106(40%) while the total mortality in this group was 11.9%. The mortality dropped with increasing age to about 8% after the age of 2 years. Right ventricular patching was required in 71 and the mortality in this group was 7%. Postoperation ECG showed complete right bundle branch block (RBBB) in 93%, left axis deviation plus RBBB in 16%, prolonged PR interval plus RBBB in 6.5% and trifusicular block in 1%. Complete heart block occurred in 4 and 2 of them required pacemaker implantation. 2 had episodic SVT and one had frequent ventricular ectopics on surface ECG. Recatheterisation after surgical repair in 108 showed intact ventricular septum in 70%. In only 6% a significant residue VSD (QP:QS $>$ 1.5:1) was present. No or mild pulmonary stenosis (gradient $<$ 20 mmHg) was present in 68% and pressure gradient between 20-50 mmHg was present in 23.8%. The anomaly could be corrected with acceptable early & late result.

P19

HAEMORRHAGIC SHOCK ENCEPHALOPATHY IN CHINESE CHILDREN

T.S. Tang, A.Y.C. Tam.
Department of Paediatrics, University of Hong Kong.

Three patients are described with a fulminant disorder characterised by rapid onset of generalised convulsion and unremitting coma, profound circulatory collapse and diffuse bleeding due to disseminated intravascular coagulation. All of them died despite vigorous supportive measures. No causative agents could be isolated. Prominent findings in the post-mortem examination included cerebral oedema, haemorrhage in various internal organs and generalised lymphocytic depletion in the thymus and other lymphoid tissues.

We believe that our patients suffered from the haemorrhagic shock and encephalopathy syndrome which is a new clinical entity that is now being recognised with increasing frequency. Common differential diagnoses are discussed and possible aetiological factors reviewed. A speculation concerning the pathophysiology of HSE is highlighted.

P20

RETROSPECTIVE SURVEY OF NEONATAL INFECTIONS IN TSAN YUK HOSPITAL

N.S. Tsoi, C.Y. Yeung.
Department of Paediatrics, University of Hong Kong.

We carried out a retrospective analysis of all positive blood and CSF culture results, sent from Tsan Yuk Hospital to the microbiology unit of Sai Ying Poon Hospital, in the past 3 years. There were a total of 46 positive blood cultures which were associated with clinical features of sepsis in neonates. The annual incidence of neonatal septicaemia was 2.44 per 1000 deliveries and the overall mortality of patient with septicaemia was 8.6%. Onset was early in most cases with 67% presenting with symptoms within 72 hours.

Group B streptococcus (17%) was one of the common organisms and this had a high mortality (33%) and morbidity.

The incidence of meningitis was 8.7%. All these cases were due to group B streptococcus and they all had associated septicaemia. E coli infection was not common in the study (6.7% compared with 20-30% in other series).

In maternal septicaemia, 23% was due to group B streptococcus. 15 mothers had septicaemia during the intrapartum period. 3 of these neonates had clinical features of infection, and one died due to Salmonella pullorum. The blood culture of the baby was negative because the mother was given antibiotic prior to delivery.

P21

MYASTHENIA GRAVIS IN CHINESE CHILDREN

V. Wong., B.R. Hawkins[1], Y.L. Yu[2], C.Y. Yeung.
Department of Paediatrics, Pathology [1] and Medicine[2],
University of Hong Kong.

Eighty-one Chinese children (41 boys and 40 girls) with myasthenia gravis (MG) are reviewed in a territory wide survey in Hong Kong. There are 2 transient neonatal MGs, 18 early onset juvenile (EOJ) MGs and 61 late onset juvenile (LOJ) MGs. Sixty-two children (76.5%) presented with pure ocular manifestations, the majority of which had bilateral ptosis and/or ophthalmoplegia. Fifteen (18.5%) had mild generalized involvement whereas 3 had moderately severe generalized and 1 had fulminating manifestations at initial presentation. The mean age of onset was 4.5 years. Complete remission occurred in 33 children (40.7%), with 2, 7 and 24 each in the transient neonatal, EOJ and LOJ myasthenics respectively. The clinical courses remained static or were marked by fluctuations with partial remissions and relapses. Myasthenic crises occurred in 2 children and one (F/9 years) died soon after presentation. Similarly, cholinergic crises occurred in 2 others and one (M/3 months) died. Pyridostigmine (100-600 mg daily) was the mainstay of treatment. Steroid was given to 16 resistant ocular/generalized MGs. Thymectomy was performed for 6 children with refractory MGs. Three had normal thymic histology, 2 had thymic hyperplasia and 1 had thymoma. The response to thymectomy was good in two, fair in one and nil in three. Associated diseases in the myasthenic children were Grave's disease in 4, autoimmune thyroiditis in 1 and systemic lupus erythematosis in one. Apart from the 2 transient neonatal MGs only one child had a positive family history of myasthenia. Anti-acetylcholine receptor antibody assay showed absent to low titres in the majority, including the two neonatal MGs. HLA typing showed a strong and significant association with HLA BW46.

P22

ACUTE VIRAL ENCEPHALITIS IN CHILDREN

V. Wong, C.Y. Yeung.
Department of Paediatrics, University of Hong Kong.

Fifty seven cases satisfying our criteria of the diagnosis of acute viral encephalitis were studied. They are divided into 2 groups - Group I (Presumed) - 48 cases; Group II (post-infectious) - 9 cases. A possible association of viral aetiology was found in 26%. Viruses isolated were influenza in 3, Coxsackie virus in 2, and adenovirus in 2 cases; mixed CMV and adenovirus and Herpes Simplex viruses in one case each. The mortality rate was 28%. Among the 41 survivors, 76% are completely normal and 24% have neurological sequelae with focal neurological deficit in 29%; personality changes in 6%; moderate mental retardation in 2%; severe mental retardation in 4%; hyperactivity in 4% and epilepsy in 4%. The best predictors to unfavourable outcome are the rapid rate of deterioration in conscious level after admission and the age of the patient.

P23

ATAXIA TELANGIECTASIA IN CHINESE CHILDREN - A CLINICAL AND ELECTROPHYSIOLOGICAL STUDY

V. Wong, Y.L Yu[1], W.Y. Chan-Lui, E. Woo[1], C.Y. Yeung.
Departments of Paediatrics and Medicine[1], University of Hong Kong.

The clinical manifestations, immunological, chromosomal and multimodal electrophysiological studies of five Chinese patients with ataxia telangiectasia are described. One died of hepatocellular carcinoma not associated with Hepatitis B-antigenaemia. Another died of respiratory failure. Two siblings are free of sinopulmonary infections although they are wheelchair bound. Computed tomography of the brain showed cerebellar atrophy in four cases. Nerve conduction studies showed evidence of axonal neuropathy in all cases with the earliest detection at six years. Electromyography showed mild denervation changes in two cases. Two patients had abnormal somatosensory evoked potentials and one had abnormal visual and brainstem auditory evoked potentials. The level of alpha fetoprotein was elevated whereas the serum carcino-embryonic antigen was normal in all patients.

P24

EFFECT OF SERUM pH ON BILIRUBIN-PROTEIN BINDING

C.Y. Yeung, F.T. Lee, H.N. Wong.
Department of Paediatrics, University of Hong Kong.

Progressive increase of the serum pH upon storage, in different conditions, during a period from 2 hours to 2 weeks is documented. Bilirubin albumin titration curves were studied by Sephadex gel filtration and horseradish peroxidase oxidation methods at different pH. Results from the peroxidase method showed that pH correction by carbon dioxide produced more reproducible and consistent results than by hydrochloric acid. For bilirubin albumin binding research studies, we suggest adopting a serum pH of 8.4 to 9.4 to produce consistent results and for clinical studies pH 7.4 is recommended to obtain clinically meaningful data.

P25

INTRA-UTERINE GROWTH PROMOTING EFFECT OF A CHINESE HERB

C.Y. Yeung, C.S. Leung, F.T. Lee.
Department of Paediatrics, University of Hong Kong.

A cigarette smoking model was developed to produce foetal growth retardation in rats. A Chinese herb-mixture (12 Tai-Bau 十二太保) which has been claimed to promote growth and well-being of the foetus was tested.

Pregnant rats were randomised into smoking and non-smoking regimes. They were further randomly divided into control and herb-treated sub-groups.

Smoking in pregnant rats consistently resulted in reduction of foetal weight. However, the herb-treated smoking mothers produced significantly heavier foetuses than the non-herb treated counterparts. There was no such effect of the herb in the non-smoking rats.

Our findings suggest some growth promoting effect of certain herbs in pregnancies which are compromised by adverse factors such as cigarette smoking.

P26

RENAL SIZE MEASURED BY ULTRASONOGRAPHY IN CHILDREN

E.C.L. Yu, E.M.T. Chau[1], L.L.Y. Leong[1], K.P. Fung.
Department of Paediatrics, University of Hong Kong.
Department of Radiology[1], Queen Mary Hospital.

72 Chinese children with no history of renal diseases, normal blood pressure, urinalysis and normal growth parameters were studied. Renal length, width and thickness were measured by ultrasonography. The renal parameters were correlated with body height using a univariate linear model. Kidney length (L) was shown to vary with body height (H) according to a regression equation L (cm) = 0.045 H (cm) + 2.132 cm with a correlation coefficient of 0.88. Percentile charts for renal length, width and thickness were made for use in the diagnosis of nephromegaly and in monitoring of patients with urological problems.