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The **January** General Meeting of the Association was held at the W.N.L.A. Hospital, Johannesburg, on Thursday, 20th January, 1955, at 3 p.m., **Dr. C. Berman** (President) in the Chair.

There were present 17 members and the Secretary.

IMPRESSIONS OF A RECENT VISIT TO THE UNITED STATES OF AMERICA WHILE ATTENDING CONFERENCES ON CANCER.

By Charles Berman, M.D., M.R.C.P.

My recent visit to the United States, which was sponsored by the University of Pittsburgh, was undertaken primarily to attend the Fifth International Conference of Geographic Pathology at Washington, where I had been requested by the International Society of Geographic Pathology to read a paper on primary liver cancer. However, more invitations to speak at other conferences followed, so that the greater part of my time in the United States seems to have been occupied with these gatherings.

The journey to America was accomplished in an incredibly short space of time. We left Johannesburg by Pan-American Airways Clipper on a wintry morning one Sunday in August, 1954, and although we stopped over for four days in that old-world city of Lisbon, New York was reached at dawn on Friday of the same week.

THE GORDON CANCER RESEARCH CONFERENCE.

After a preliminary two-day stay in New York, we set out by train for New London, in New Hampshire, where I had been invited to attend the Gordon Cancer Research Conference (of the American Association for the Advancement of Science) as one of the principal speakers. The journey, which lasted almost a day, was through very beautiful country, for New Hampshire, a mountainous state bordering on Canada, is rich in lakes and rivers, and its vast stretches of forest were already assuming the characteristic brilliant orange and purple autumn ("fall") tints so famous for this part of the world.

The Gordon Cancer Research Conference (the last of 24 weekly conferences embracing many branches of science) was held during the summer recess in the elegant red-brick buildings of the well-known Colby College, which stands on a spacious campus of carefully tended vividly green lawns. This relatively small cancer conference (there were only 80 scientists attending) is regarded to-day as one of the most important in the United States, for, by bringing together scientists in related fields, it is designed to stimulate research in universities, research foundations and industrial laboratories. Moreover, much of the work presented was recent and had not yet been published. Among those attending were experimentalists, physicians, surgeons and representatives of the National Cancer Institute and the American Cancer Society (which were sponsoring a great deal of the research projects), as well as research workers from world-famous pharmaceutical laboratories.

All the meetings were informal. They consisted of scheduled lectures followed by free discussions during the mornings and evenings, the afternoons being set aside for recreation or group discussions. Each paper lasted 25 minutes. The principal speakers, however, were allowed 50 minutes.

The five invited speakers, all from different parts of the world, were as follows :—

(1) Dr. V. R. Khanolkar, of the Tata Memorial Hospital, Bombay, spoke on "Cancer in Relation to Usages." He showed that cancer of the mouth, so common in Bombay, occurred in Indians who smoked cigars with the lighted ends *inside* the mouth, as was the local custom. This form of cancer was also prevalent in North Behar, but here mixtures of tobacco, lime and betel nuts are commonly chewed. He also showed that skin cancer of the groin and the lower abdomen, frequently observed in North India, was due to the use of a "kaangri," a smouldering earthenware stove, which is carried under the clothes of these people to keep the body warm.

(2) Professor C. Oberling, of Paris, spoke in faultless English on 50 years' experience in cancer research.

(3) Dr. I. Berenblum, of the Weizmann Institute of Science, Israel, discussed his work on the experimental induction of stomach cancer in mice.

(4) Dr. P. Stocks, of England, spoke on environmental factors in the causation of cancer. He dealt mainly with cancer of the lung and its relationship to tobacco and air pollution in the large British centres.

(5) Dr. C. Berman, South Africa. My own contribution was an illustrated lecture on the general problem of primary liver cancer, with special reference to the Bantu.

Among the general subjects discussed, the following require special mention: "Carcinogenic Hazards in Industry," by Doctors W. C. Hueper, M. J. Shear and P. Shubik; "Changing Concepts in the Surgery of Cancer," by Dr. E. Dunphy, who described heroic surgical measures on metastatic growths; Dr. S. Farber spoke on the results and current trends in the chemotherapy of cancer, especially in children suffering from leukaemia and lymphosarcoma.

Perhaps the most notable event at this memorable conference was the showing of a 16 mm. black-and-white movie film by Doctors R. E. Zirkle and W. Bloom, on the "Effects of Irradiating Parts of Simple Dividing Cells." It was fantastic to behold the dramatic movements and groupings of chromosomes before the division of the cell into two, and the slowing down and gradual disintegration of one chromosome which had been singled out for irradiation by X-rays and by ultra-violet rays, whereas the remaining chromosomes appeared to be unaffected and continued with their grouping for cellular division. The task which these investigators have before them is to determine whether cells irradiated in this manner will ultimately reproduce cell-types showing radical differences in structure and behaviour from normal cells.

The New Jersey and Pennsylvania Turnpikes.

My next and principal assignment was the International Conference of Geographic Pathology at Washington, D.C., which was reached by road via New York and Philadelphia. The greater part of this journey was on the magnificent New Jersey Turnpike. This, like the Pennsylvania Turnpike, over which we were to travel later, is one of the world's greatest highways and stretches for hundreds of miles through park-like country, unsullied by the ugly hoardings that one had expected to find in this country. Constructed of reinforced concrete, it has four wide lanes of incessant traffic and is without a single sharp turn, a pedestrian hazard, a stop-light or a steep hill. The road goes straight through mountains in well-lit, clean, tiled tunnels. It has service stations and excellent restaurants, and numerous efficient "motels" are to be found in the vicinity. The turnpikes are carefully maintained, mainly through the toll-money collected.

Washington.

To-day, Washington is in every sense an imperial city. The wide, well laid-out streets, flanked by massive public buildings of proud classic design, the many varied and picturesque foreign embassies, the noticeable absence of industries, the important museums of art and science and the impressive monuments to her famous statesmen conjure up visions of imperial Rome in her heyday.

The Fifth International Conference of Geographic Pathology.

This important conference, like the International Congress of Clinical Pathology and the International Meeting of the Association of Medical Museums, was sponsored by the World Health Organization of the United Nations Organization. All these three conferences took place simultaneously in the gigantic Shoreham Hotel in Washington.

This combined conference was stupendous. More than 1,400 doctors attended (many accompanied by their wives) from many parts of North America, South America, Europe, Africa, Asia and even Australia. It had taken more than three years to organize and lasted one week.

The Shoreham Hotel has more than 1,200 rooms and many reception rooms and is placed in a beautiful setting. With the exception of the plenary sessions (one at the beginning, the other at the end of the conference) all three sections held their separate meetings simultaneously in the great halls, the largest of which had over 1,200 seats.

The organization was superb, and the conference included excellent scientific, trade and technical exhibits.

For its fifth conference, the International Society of Geographic Pathology considered cancer involving the following five organs: stomach, liver, lung, breast and the uterus.

I was one of four principal speakers in the section on liver cancer. We had been invited to act as reporters and read papers based on the results of a questionnaire on primary liver cancer that had been circulated to various parts of the world. My particular task was to discuss "Nutritional States in the Causation of Primary Liver Cancer." The other three reporters included

Dr. P. F. Denoix, of Paris, who spoke on "The Geographic Distribution of Cancer of the Liver," Professor F. C. Roulet, of Basle, Switzerland, whose paper was "Pathologic Anatomy of Primary Cancer of the Liver," and Dr. J. Higginson, of Johannesburg, whose subject was "The Relation of Carcinoma of the Liver to Cirrhosis, Malaria, Syphilis and Parasitic Diseases."

Other speakers on the subject included contributors from China, Malaya, Uganda and the United States. Professor Pao-Chang Hou, of Hongkong, showed some interesting coloured lantern slides demonstrating the relationship between *Clonorchis sinensis* and primary liver cancer, the most common form of malignant tumour found among in-patients in the Queen Mary Hospital, Hongkong.

The International Conference of Geographic Pathology held all its meetings in the largest hall, where effective amplifiers had been installed. There were four official languages, including English, French, German and Spanish: the speaker's remarks were translated simultaneously into these languages by a group from the UNO headquarters in New York and could be heard through a number of head-phones in a specially cordoned-off portion of the great hall.

The meetings of the three conferences took place during the day, the evenings being devoted to such special functions as visits to the Library of Congress and the National Gallery of Art, receptions, etc. Interesting round-table discussions were also arranged during the lunch hour, when special topics related to pathology were considered.

On the last afternoon a joint scientific session was held, when the Askanazy Lecture, commemorating the founder of the International Society of Geographic Pathology, was delivered by Professor Henschen of Stockholm. His topic was "Hereditary Disease in the Four Nordic Countries." This was followed by four other speakers who had been especially invited to deliver short papers on diverse subjects other than cancer. I had the honour of being one of these. My paper was on "Onyalai." The other speakers included Dr. E. W. Gault, of Madras, who spoke on "Brain Lesions Due to Parasites and Other Infections in India"; Professor M. Straub, of Rotterdam, whose subject was "Pathology Figures from Netherlands during and after the War"; Doctors K. L. Joe and S. Tjokronegoro, of Indonesia, who spoke on "Hepatic Cirrhosis and Fibrosis in Children in Djakarta"; and Dr. J. Davidsohn, of Chicago, who delivered the Ward Burdick Award Lecture on "Immunohematology, a New Branch of Clinical Pathology."

This was a very impressive session—more than 1,200 doctors crowded the great auditorium.

Medical Centres in and around Washington.

Before leaving Washington I took the opportunity of spending some time at the celebrated Armed Forces Institute of Pathology and its Medical Museum, the National Institutes of Health and the large ultra-modern National Naval Hospital, the famous Walter Reed Army Hospital and the extraordinary newly-designed Armed Forces Institute of Pathology.

The National Institutes of Health and the Clinical Centre.

Seven institutes in close proximity to each other and under one central control constitute the National Institutes of Health. These institutes are, respectively, engaged in research on cancer, microbiology, mental health, dentistry, arthritis and metabolic disorders, and neurological diseases, including blindness.

Serving these institutes is the adjoining newly-opened "Clinical Centre." This unique 500-bed hospital has for its purpose a unified approach to clinical and basic research and provides twice as much space for the laboratories as it does for beds.

When any of the above health institutes has decided upon a research project and the necessary details have been elaborated (including personnel, apparatus, space, etc.), the investigation is conducted on the same floor of the hospital by the research group as a unit.

During the course of the research project the only patients admitted to this unit are those who meet with the diagnostic requirements agreed upon. Thus, if the investigation is on a particular form of thyroid cancer, the patient is admitted only on the basis of a common diagnosis which meets specific criteria such as age, sex, the particular type of cancer, etc. All the patients enter the hospital on a voluntary basis and receive the best and most up-to-date treatment possible, including surgery, nursing and social services.

The New Armed Forces Institute of Pathology.

This remarkable institute, which was almost ready for occupation, is located in the grounds of the Walter Reed Army Medical Centre. It is an eight-storey building, which has been specially designed to resist attacks by atomic bombs: with

the exception of two small wings, it is entirely without windows. Five of the floors are above ground, the other three are underground.

The roof and exterior walls, as well as the beams and floor slabs, are of heavily reinforced concrete. The rooms are artificially lighted and air-conditioned. It is noteworthy that the wall facing the city of Washington is designed to provide twice the strength of the roof and the other walls.

Openings through the blast wall can be closed by motor-driven remote-controlled or manually-operated blast doors. There are standby emergency electric power units with automatic controls and a reserve water supply.

Facilities for television have been devised. By means of a colour television circuit planned between the surgical pathology laboratory and the operating theatres at the Walter Reed Army Hospital, the pathologist will be able to observe and talk with the surgeon during an operation. The surgical staff will be able to observe the gross and histological aspects of a surgical specimen and will be able to discuss it with the pathologist. To supplement this television circuit, there is a pneumatic system that can speedily deliver specimens from the operating room to the pathological laboratory, approximately three blocks away.

Pittsburgh.

The notorious fogs and smoke which for years were the bane of the important industrial city of Pittsburgh are to-day things of the past. Thanks to the wise regulations recently enacted, prohibiting the use of inferior fuel in the giant steel works, other industries and in the homes, the problem of air pollution has to a great extent been mastered. Moreover, many of the older buildings are to-day being replaced by beautiful aluminium-faced sky-scrapers, an excellent example of which is the novel 30-storey Alcoa Building.

Pittsburgh is a great educational centre. Its elegant university, a sky-scraper of more than 40 storeys built in the shape of a beautiful Gothic church, is appropriately called "The Cathedral of Learning" and stands on a spacious green campus surrounded by other impressive buildings. These latter are libraries, art galleries, halls, hospitals and the world-famous Carnegie Museum. The equally famous Mellon Institute is also located here; a great deal of important research work is being carried out at this institute, mainly on industrial problems, including industrial hygiene.

Through the generosity of the University of Pittsburgh I was privileged to spend more than two weeks in this dynamic city—mainly at the Medical School and at the celebrated Presbyterian Hospital. The Medical School is at present being rebuilt at a very great cost and will doubtless become one of the leading medical centres in the United States.

New York.

My final assignments were in New York, which was reached from Pittsburgh via the already mentioned famous Pennsylvania Turnpike. I had been invited to speak on various aspects of primary liver cancer to a number of gatherings, including the New York Cancer Society at the Academy of Medicine, Dr. George Pack's surgical group at the Memorial Cancer Hospital, and the Conference on Experimental Hepatomas at Arden House, Harriman.

New York is awe-inspiring. Its gigantic sky-scrapers glittering in the heat of day, its nights transformed by the prodigal use of multicoloured neon lights, its teeming populace, and its ever-moving traffic were constant sources of delight and wonder. It was quite impossible to resist being caught up by the exaggerated tempo prevailing in this greatest of all cities.

The Conference on Experimental Hepatomas.

This conference was held at Arden House, Harriman, situated some 60 miles from New York.

Arden House was built by the late Mr. E. H. Harriman, the railroad millionaire, as a family residence. Recently, however, it was presented to Columbia University by his son, Mr. Averell Harriman, the newly-elected Governor of New York State, as a meeting place for scientific and educational purposes.

Arden House is a massive structure, in many respects resembling an 18th-century continental castle, and consists of almost 100 rooms. Situated on a hill overlooking a river, it is surrounded by forests and is set amidst beautifully laid out grounds containing lawns, terraces and gardens. There are many conference rooms of varying sizes, the largest seating more than 200 people. The furnishings and service are excellent. Educational and scientific conferences are held here throughout the year.

The Hepatoma Conference, to which I had been invited by the National Cancer Institute (under whose auspices it was held), was attended by 75 scientists. It was a crowded affair, there being morning and afternoon as well as evening sessions,

when papers on various aspects of experimentally-induced hepatomas were read. Among the subjects discussed were spontaneous liver tumours, genetics, histopathology, histophysiology, carcinogenesis by deficient diets and by chemical compounds, biochemical investigations, and hormonal influences in the evolution of the liver tumours. Of particular interest was a graphically illustrated paper demonstrating the fine structure of normal liver tissue as observed under the electron microscope.

My particular task, which I shared with Professor J. N. Davies of Uganda, was to assess the human implications of the experimentally-induced hepatomas as presented at the conference. The views put forward by us at its termination provoked a considerable amount of discussion. The proceedings of this very instructive and interesting conference are in the course of publication.

This address cannot be concluded without mentioning the cordial friendship and hospitality which we received everywhere in the United States—experiences that will never be forgotten.

In conclusion I would like to thank the University of Pittsburgh and Dr. Robert A. Moore, its Vice-Chancellor, for sponsoring my visit to the United States, and Dr. A. J. Orenstein, the Directors of Rand Mines, Ltd., and the National Cancer Association of South Africa for making it possible to prolong my stay in that very great country.

A number of coloured and other lantern slides illustrating some of the localities visited were shown.

Dr. Retief said they had all greatly enjoyed Dr. Berman's talk and illustrations, and asked if he would tell them something of what he himself had told the conferences.

Dr. Berman replied that this would really require to be the subject of a separate talk.

Dr. Sonnenfeld asked what researches were being carried out in connexion with cancer of the lung and tobacco smoking.

Dr. Berman said that this was a very controversial question. Researches were currently being carried out in an endeavour to induce cancers of the lung in animals by allowing them to live in an atmosphere of tobacco smoke, and by injecting them with the products and suspensions of tobacco, etc., but no definite results had as yet been obtained. On the other hand, statistical surveys in America and Britain had shown that there was something in the theory, and that cigarette smoking was more likely to cause lung cancer than smoking cigars or pipes. The tobacco companies were also carrying out researches to disprove the theory connecting tobacco smoking with lung cancer and had engaged a well-known American cancer research worker for the purpose.

Dr. A. Smith congratulated Dr. Berman on his talk and asked if there was any great incidence of primary cancer of the liver amongst people in the United States.

Dr. Berman replied that primary liver cancer was as rare amongst both the whites and negroes in North America as it was amongst the white people in South Africa or in Europe. The disease, apparently, was not due to a genetic factor. Recent evidence was forthcoming that the most probable cause was a combination of dietetic and carcinogenic factors. The dietetic factor was most probably due to lack of first-class proteins, but the carcinogenic factors were as yet unknown. In countries such as Indonesia, Malaya, and other parts of Africa where economically the populations were as backward as the Bantu, the incidence of primary liver cancer was often as high as it was in South Africa.

In reply to a question by **Dr. Sonnenfeld,** Dr. Berman said that the induction of carcinoma of the stomach in mice was effected by means of carcinogens, such as benzpyrene and other aromatic hydrocarbons. The induced stomach cancers in mice, however, could not be compared with those found in humans, since they arose from a portion of the stomach that was lined by squamous epithelium derived from ectoderm (which was absent in humans), and most resembled that of skin cancer.

Dr. Dreosti thanked Dr. Berman for his talk and congratulated him on having been chosen to attend the International Conference in Washington. Many years ago, when they were both working together at the City Deep Hospital, Dr. Berman, who at the time had suffered a great personal loss, confided in him his desire to make a personal contribution to the study of cancer. He was glad that Dr. Berman had succeeded in his ambition and had also achieved international recognition.

The **February** General Meeting of the Association was held at the W.N.L.A. Hospital, Johannesburg, on Thursday, 17th February, 1955, at 3 p.m., **Dr. C. Berman** (President) in the Chair.

There were present 27 members and the Secretary.

A BRIEF OUTLINE OF THE DYNAMICS OF HEAD INJURIES.

By K. Lewer Allen,

Head of the Neuro-Surgical Department, Johannesburg General Hospital and University of the Witwatersrand.

An initial feeling of confusion and hopelessness invariably comes over me on seeing a head injury for the first time. I imagine that this is the experience of others too. Probably the reason for this is our sense of the complexity of all the forces, latent and acting, that are hidden in the head before us. Of course, we cannot hope to recognize all the factors in this complexity immediately. We can, through our signs, assess some of the static mechanics, but it is more important to understand and anticipate the tendency to change —the serial consequences which follow upon a given event. We have to assess which tendency is good, and which bad, and therefore what is the natural course, whether it is heading for resolution or dissolution, and we must find out how we can guide the trend towards recovery. It might be a very small piece of interference, which, if timely, can reverse a sequence. My experience has come to make me realize that it is more often unnecessary to employ a dramatic force, such as a major surgical procedure, to achieve this end, and the timely, thoughtful use of lumbar puncture, ventricular tap, or even a sedative, may be better and more natural than a heroically executed decompressive operation.

By understanding the dynamics of acute head injuries it becomes possible for the surgeon to carry out a method which we might call "preventive surgery" rather than "emergency surgery." I might say humbly that such a concept should ideally apply to all surgery beyond acute work. The recognition of the trends and the time sequences can easily be clouded by the preconception of the stages of illness, or the "syndromes of signs," set up in our minds by prescriptive and empirical teaching, which classifies grades of injury into blocks of time. I confess I never quite knew what was particularly differential between "closed and open head injury," "major and minor contusion," "commotio cerebri," nor the passage of one to the other state, assessed in retrospect by the ticking of a clock of 24, 48, 78 hours. Equally confusing to me was the dogmatic teaching of treatment, which advised such controversial and divergent methods as, "always do a lumbar puncture," "never do a lumbar puncture," "push fluids," "restrict fluids," etc. Let us recognize the trend of things and not the stage. The exception to this, of course, is the first viewing of the problem, where we must make some form of static assessment and know what are the tangible features of the injury, as expressed by open wounds, pulse rate, blood pressure, etc. But immediately we are alerted to put these facts into such perspective as to begin our assessment and anticipation of the sequence that is about to follow. At this moment we must check our understanding of the normal dynamics within a head. What is required most of a brain to ensure its proper function?

(1) It should remain protected by the varying elastic membranes which bound it.
(2) The cerebrospinal fluid must be contained by these membranes, so as to surround and support the brain, and cushion or diffuse velocity changes.
(3) The brain should be free within this environment to act uninhibited as a pump.
(4) By virtue of this pump action of the brain, and the balanced counter-tensions of its environment, an adequate blood circulation and C.S.F. circulation is maintained, with consequent vital oxygenation of the brain tissue.
(5) Electrical and biochemical function is to be efficient by virtue of this circulation.

There is precedence in these requirements, and the initial assessment as to where in the sequence has the dynamic state in the head been rendered by the disruptive forces of the injury. One or other requirement may be satisfied partially, while another priority may be deranged. Such a state may be compatible with life, but not with full efficiency, which in the end depends on the integrity of each prior requirement. For instance, we might pump the blood round the brain by artificial means, but we would be unable to maintain this efficiently unless the brain could regain its activated pump action by the reconstitution of all other mechanical requirements within its environment.

I will concentrate now on the less evident, but perhaps more important requirement, namely the so-called cerebral pump. The normal brain is not inert. It expands and contracts by change in its

total volume, due to the transmission of fluid volumes and pressure fluctuations from extra-cranial sources or systems. We should conceive the cerebrospinal fluid, thoracic, abdominal and vascular fluid systems being within a total surround, subject to barometric pressure, and first of all imagine that these various systems are not separated by an intervening resistance membrane. Changes in the barometric pressure, or in any imaginary component system, would be absorbed equally throughout all systems. However, when we introduce resistance barriers between them, the diffusion of pressure fluctuation is slow and becomes unequal for a given time in each system, but the systems still remain interdependent, provided the membrane resistance of any one enclosed system is not completely rigid. If any one system is enclosed within an almost completely rigid membrane, there will be recoil effects on the transmitted pulses, which may summate with complex effect within that cavity. So we must conceive the cerebrospinal fluid system as being influenced by pressures initiated in all other pressure systems throughout the body, and the barometric surround, and the brain tissue itself can be conceived, for simplicity, as a resistant barrier interposed between the C.S.F. and the other fluid systems. This resistance barrier is able to absorb and to transmit the pulses from extra-cranial sources, and further to absorb or transmit the recoil effect within the partially opened cranial cavity, and further since this resistance barrier enfolds in a complex fashion to enclose the cerebrospinal fluid so as to form ventricular chambers, the C.S.F. partially trapped within these chambers will be displaced or compressed by any expansion or contraction within the brain barrier. We must look upon the brain, therefore, as a passively activated pump with a systolic and diastolic phase, and a consequent directional flow of cerebrospinal fluid, and also of the blood volumes, mainly venous, which likewise are enmeshed. The direction of flow of these two fluids respectively is determined in large measure by the graded resistance found in the resistance brain barrier, and further influenced by the balance, at different stages of its activation, with counter-pressures within the cranial cavity. It is not easy to gain this conception, but when we do, we have a glimpse at the reason for the remarkably standard, though weird, shape of the normal ventricle, and we have some understanding by which we might interpret or anticipate the changes within that shape, following upon pressure or resistance change within that barrier or its surrounding systems and their confining structures. For instance, it becomes the more obvious why a brain would tend to herniate through a skull defect, and the ventricle to wander into this herniation, but further why this herniation should be controlled by regulating the pressures on either side of the skull defect. We also get a glimpse at why the C.S.F. should show the pressure pulses that it does, and why it should be maintained within a certain normal pressure range. We begin to see why a lumbar puncture, carried out quite early in the assessment, can give us a fund of information as to the dynamic state and trend, were we only able to interpret and use this information properly. Now, when this cerebral pump is inert, both the C.S.F. and the blood circulation become stagnant. CO_2 tensions rise, anoxia develops, changes in the capillary wall occur, osmotic tensions are disturbed, and extra and inter-cellular fluid increases, producing cerebral oedema. This is a vital chain of events, which links the mechanical with the final functional sufficiency. As this sequence continues, the intracranial pressure rises, due to the simultaneous rise in the intracerebral tension and the accumulating C.S.F. tension. The C.S.F. itself cannot readily be displaced to its absorption areas, which are obliterated by the expanding brain, and ultimately the brain must herniate through the tentorium or through foramen magnum, unless some compensatory change can occur. The cerebrospinal fluid secretion appears to be reduced under these circumstances, thus tending to reduce the volume of C.S.F., but the balance between secretion and absorption requires a longer time to occur than is required to offset the more acute changes in pressure in these other senses. Therefore, we often have to interfere to tap off the cerebrospinal fluid, and this is beneficial and safe when done early in the sequence, either by lumbar puncture or through a burrhole, because at this stage we may succeed in increasing the pump excursion and thereby improve the directional flow and circulation of the blood and C.S.F., and thus reduce oedema and anoxia, and preserve function. However, if we were to tap off the C.S.F. by either route too late in the sequence, we can aggravate the dominance of the cerebral oedema and aid the rapid encroachment of the swelling brain upon the ventricle. Sometimes it is expedient to introduce a small amount of air into a ventricle at an early stage in the sequence. This can help to improve the counter-tensions to the advancing oedema, air being better than water, because as a gas it is compressible, and its effect is smoother and safer. How much air to introduce becomes a

matter of experience in feeling through the glass-barrelled syringe, and the sense of touch and resistance tells us just how to balance the air tension against that of the surrounding brain, and yet to leave a margin for the gas to expand initially as it warms to the body temperature. The experienced use of air in this way, into the ventricle, may prove a valuable weapon in offsetting the progressive shift tendencies of a sub-dural haematoma, or to reduce that shift or sheer of a brain under the free edge of the falx, after the removal of a sub-dural haematoma.

The cerebral pump may become sluggish due to the following causes :—

(1) Weakness of the pulses reaching it from the other pressure systems. Such weakness may occur in a state of shock or syncope.

(2) An increase in the physical resistance of the intervening brain barrier. Oedema is the major cause of this increased resistance in head injury. Other causes are the increase in the extra-cerebral intracranial spaces, due to haematomas, such as sub-dural collections; or tension cysts, arising in areas of laceration, by the accumulation of intra-cerebral free blood.

These same tension factors may lead to—

(3) Compression and displacement of the brain, which limit its free pulsation.

(4) Loss of physical resistance in the brain barrier, due to softening or dehydration. This state implies loss of tone within the hydraulic system, again with sluggishness of circulation of both C.S.F. and blood, again with the chain effect, leading to anoxia and functional failure.

The low pressure state which occurs in conjunction with this lowered resistance is of utmost importance in the post-traumatic handling of cases, and I will refer to it later as an entity. Obviously the methods used to energize the pump will depend on the analysis of these different and sometimes opposite circumstances. Unfortunately, the clinical signs and symptoms attendant upon these divergent states are very much the same; for instance, depressed conscious level and focal neurological signs may occur in both high and low intra-cranial pressure states, but there is an essential difference in the absence of oedema in the latter, and therefore the risk to life is not, in fact, as great or as acute in the low pressure state. It is sometimes difficult to realize, under cover of common symptoms and signs, the passage from the high to the low pressure state, which, in fact, often occurs, with a brief improvement in the symptoms as they pass through the normal pressure level. It becomes the more important then to make a definite manometric pressure recording, not once, but repeatedly, throughout the changing condition of a patient. It can be accepted, for practical purposes, that lumbar puncture in an acute head injury is almost invariably safe and seldom the cause, *per se*, of a fatal coning of the brain. As an alternative and often preferable method, ventricular pressures may be measured and regulated by needling through a burrhole. The choice of method depends largely on the additional information which can be gained by using a burrhole to inspect the sub-dural spaces and visualize the brain and its pump action directly, and then the ability to needle into haemorrhagic areas and to evacuate intracerebral clots and tensions cysts, and finally the ability to introduce air gently into the ventricles for therapeutic and diagnostic purposes. Note that all mention so far regarding the use of lumbar puncture and ventricular taps has implied combined treatment and diagnosis, rather than their use exclusively or predominantly as diagnostic techniques.

Now I shall say a bit more specifically about oedema and low C.S.F. pressure states.

Oedema, once again, is a most important single state. Hardly any case of head injury, except the most minor concussion, is not attended by some degree of oedema. A mild degree can be looked upon as an almost physiological event to tone up the tension of the cerebral resistance barrier, and consequently the pump effect; to limit further bleeding; and to help in the early reparative process after the initial injury. This degree may be called "reactive" and comes on within an hour or more of the blow, becoming maximal in about 24 hours, after which it decreases and disappears without residue. But beyond this degree oedema can be harmful and it may even become malignant, that is, uncontrollable, and increase to produce death by rapid and extreme anoxia or mechanical coning. This is a rare extreme form of oedema in head injury, and far more common in cerebral inflammations, where the noxious factors continue their action, but much oedema will retard the normal dynamic recovery, depress cortical function by impairing dynamic and electrical procedure, and by prolonging cerebral compression and producing shifts, a low level of consciousness will endure, with greater risk of intercurrent complication. Also, the longer a severe form of oedema persists, the more is the gliosis which eventually replaces large areas of

brain tissue. Oedema of such degree is often caused and maintained by the presence of free blood within the lacerated area, and the sooner this blood is removed or diluted, the quicker the oedema subsides. Since venous tension in the brain tends to rise with oedema, or because of oedema, and because C.S.F. pressures rise concurrently, it is often possible to regulate the oedema and its ill effect by reducing the C.S.F. pressure, and therefore repeated small C.S.F. taps in the early stages of an injury will be, as said before, of vital importance in keeping the oedema within physiological degrees, and it is very often encouraging to see how immediately consciousness can be improved, as also the general state of the patient, after each cerebrospinal fluid tap. The change can be so rapid that it must surely be due to a fairly immediate improvement in the cerebral oxygenation. The more major surgical decompression, through an osteo-plastic flap, should be reserved for the more uncontrolled forms of cerebral oedema, or where the cause from intracerebral haemorrhage cannot be dealt with by discreet needling. It is rarely necessary, in cases of malignant bilateral oedema, with total obliteration of all C.S.F. pathways, to turn a bilateral, balanced, riding osteo-plastic flap. Needless to say, this is an emergency state carrying a high mortality result. Sub-temporal decompressions have no real planned place in head injury, except in the case of reaching a middle meningeal bleeder.

Now finally the low pressure states are, in my experience, very frequent in head injuries, and I go so far as to say that they are often potential from the moment of the initial blow. It would appear that C.S.F. secretion is possibly dependent upon some neural control, which is paresed by the concussion forces in much the same way as all neuronal cells tend initially to discharge their electrical potential at the moment of the blow, and to be followed then by a state of electrical paralysis. The potential low intracranial pressure state, which would follow upon this secretive failure, is usually offset by the reactive oedema, which I mentioned before, over the first two to three days, but as the oedema disappears, the latent low pressure state becomes manifest and too often we find that the lumbar puncture pressure begins to fall below the normal level, down to 80 or 60 mm. of water or less, and sometimes at these lower levels, conscious levels once again decrease, the patient becomes stuporose and irritable, and experiences severe headache. These symptoms may be relieved by posturing in the head down position, increasing fluid and salt administration, and even by the introduction of air into the ventricles, under slightly positive pressure. It must be recognized that the pseudo low pressure state may reflect upon lumbar puncture pressure measurement, although the intracranial pressure is still high, and this paradox occurs sometimes in bilateral sub-dural haematomas. We must also remember that severe low pressure states may accompany actual C.S.F. loss through a basal fracture, such as in rhinorrhoea.

In all this discussion I have left so much unsaid, for instance, the concurrent significance of blood in the C.S.F. and the need to clear it rigorously, the treatment of sub-dural fluid collections, and the particular dynamics of supratentorial shifting pathologies, with shearing effects on the falx and tentorium. One could mention in passing the disappointing effects of intravenous dehydration by the use of hypertonic solutions, and then the use of new drugs such as Cortef. There is so much to be said about this fascinating and frustrating subject, where the very extent and degree of the injury often beats us before we start, but where in the surviving group, regulation of all these factors mentioned may not only save a few more lives, but will reduce in large measure the long-term complications such as personality change and epilepsy.

Dr. Dreosti thanked Mr. Allen for coming to address the Association so soon after his illness. As regards the substance of the talk, he would like to ask how Mr. Allen controlled cerebrospinal pressure. Did he tap continuously until it went down to normal or frequently in small quantities? Was the treatment of head injuries different in cases in which one found blood in the cerebrospinal fluid from cases where one did not find blood?

Mr. Allen showed a specimen of the lumbar puncture set which he used. It was of an easily handled size and was easily sterilized. There was a glass-barrelled syringe and a set of needles for local anaesthesia (skin only). They used a rather fine needle and a simple ungraduated glass U-tube of fairly small bore connected by means of a length of fine rubber tubing. The fluid flowed through to the U-tube and then they measured the pressure against a centimetre rule or graph paper. There was no inertia such as with an aneroid type ("clock") manometer and no difficulties as with the calibrated glass tube, which could not always be easily replaced when it broke. They insisted on the patient being unfolded in a neutral position and a relaxed state, with no abdominal pressure, after inserting the needle, before reading the pressure.

When they had made the measurement he had described, if the pressure was high—say 200—they let it drip very slowly and measured the volume. They took off 2 c.c. of fluid and again measured the pressure. If a quick drop had occurred, one could be sure that one was dealing with an explosive force in the oedema. With a slow fall in pressure one had lots of reserve and therefore the lumbar puncture was done merely to regulate the cerebrospinal fluid pressure. If, however, there was a very quick drop, they used the puncture to find out what they were dealing with. In this "spitting" stage of oedema, they reduced the pressure by frequent punctures.

With reference to Dr. Dreosti's second question, if one found blood in the cerebrospinal fluid, even to a minor degree, there was probably some degree of cerebral irritation. The significance of this had to be worked out with other additional signs. From the point of view of treatment, that blood had to be cleared and they advocated frequent punctures — say 6-hourly — and the giving of sedatives, mainly Luminal, in between punctures. As they cleared the fluid, they put the patient into a low-pressure state.

Dr. A. Miller said that on the mines they saw many compound fractures of the skull. One could often establish immediately that there was clinical depression of the bone and yet there were no neurological symptoms. Should one go in or not in such cases?

Mr. Allen said that one should ignore a depressed fracture as such, unless it was of such massive degree that it was obviously necessary to elevate it. The average non-European case which they saw did not require surgery. If the dura was damaged, however, surgery was necessary. From the point of view of epilepsy it might be necessary to go in, but even if it was over the Rolando, epilepsy was not necessarily inevitable. If there was no cortical damage, he would leave the depressed fracture alone. Apart from the above considerations, general principles still applied, viz., that a contaminated or badly comminuted compound depressed fracture should be dealt with by removing debris, foreign matter, dirt, etc. Otherwise, one could usually place reliance on the antibiotics to control infection. Even a severe closed depressed fracture could, however, be left alone unless there were other indications for operation.

One member asked if when tapping off the cerebrospinal fluid for the oedema, the oedema was not going to take more space owing to the lower pressure.

Mr. Allen said he had advised tapping early in oedema. If one tapped late, then one was simply providing more space for the brain to swell into. If there was a blood clot, which was a common cause of oedema, then one should remove that if possible.

Dr. Sonnenfeld asked if the systolic blood pressure was any reflection of the cerebrospinal fluid pressure.

Mr. Allen said that earlier workers had stated that the arterial pressure set the pace for the cerebrospinal fluid and that the venous pressure was lower, but this did not hold in practice. They often found high arterial pressure with a normal C.S.F. pressure. There was more connexion between the venous pressure and the C.S.F. pressure.

Dr. Dangerfield thanked Mr. Allen for his address and said that on the mines they had many cases of fractured skulls. Their biggest difficulty in operating was uncontrolled haemorrhage and he asked what methods Mr. Allen used for controlling haemorrhage in such cases. Did he use an absorbent sponge or cautery? Intraventricular tapping was apparently an important procedure. Was it one for the specialist only?

Mr. Allen said that the control of haemorrhage could only be acquired by practice. The venous tensions were high when the C.S.F. tension was high. Most cases they had had to deal with were venous bleedings and not arterial. Most of the progressive haemorrhages which had not already killed were due to venous bleedings. One could reduce venous bleeding by reducing the C.S.F. tension. If they were operating, they tapped the ventricles if this was indicated. It was very difficult to control bleeding in the brain while tensions were high. Small, thin muscle grafts were very useful. He preferred to use as little foreign material as possible to control bleeding in cavities. Peroxide was very useful when used in small concentrations and did no harm. It was a good haemostatic and showed the pin-point where one could put the piece of muscle. Electrocautery was, of course, used routinely in brain surgery.

Dr. Pearson asked if needling ever gave rise to trauma and if there was any possibility of setting up haemorrhage.

Mr. Allen said it could be dangerous. A burrhole should be put in under ideal circumstances and he thought they should be done only by an

experienced person. One should avoid blood-vessels which might cause haemorrhage and should keep 3 or 4 cm. from the midline. When putting in a burrhole one should have good vision and the patient should be sufficiently controlled. Even if the vein encountered was not important or was one which could be coagulated, it was better to avoid it by moving a bit to the side. Once one had chosen an avascular surface, it was fairly safe to insert the needle. The important thing was not to go on needling in all directions. One should assess where the ventricle was likely to have shifted to and try to get it at the first attempt. If one had not found it at the third attempt, one should give it up and put in another burrhole or try the ventricle on the other side. Which ventricle one tapped was also important. If one tapped the ventricle to the side to which the brain had shifted, one might make the patient worse, but, if possible, one should first remove the cause of the shift.

The Chairman said Mr. Allen had shattered quite a number of their ideas on the treatment of head injuries. With regard to depressed fractures, many of them had felt it was their duty to relieve the depression. Another idea which Mr. Allen had altered was the question of the use of dehydration in cerebral oedema. In cases where bone had been removed and a defect was left in the skull did he subsequently replace the bone with an artificial covering?

Mr. Allen said it depended on the dynamics after acute head injury. If the skull defect was not too big and the pressures settled to normal and there was no indrawing of brain and the patient was not worrying, he would leave it. If it was large or in a position which worried the patient, he would close it, but if there was any danger of infection, one should not close it with a foreign plate for at least a year. They used perforated tantalum plates and sank them so that there were no rough edges. Split rib was very useful for filling certain defects.

The Chairman, on behalf of members, thanked Mr. Allen for giving the talk, particularly just after his illness. It had been most instructive and interesting and had given them a new concept of the subject.

CLINICAL MEETING, 3rd February, 1955.

The **February** meeting of the Clinical Club was held at the W.N.L.A. Hospital and was convened by Dr. N. R. A. MacColl.

Three Cases of Fractured Femur. **Dr. E. Kruger.** —Anatomically, the aim of treatment of a fracture of the femur is to restore the bone to its former length, alignment and shape; physiologically, the aim is to restore joints and muscles to their former function.

In manipulative reduction (under anaesthesia) it is a common mistake to apply perfunctory traction for only a few seconds. Most fractured surfaces are irregular, and projecting spikes prevent the fragments from sliding into position. The limb must be slightly over-lengthened before the serrations disengage, and slow steady traction must often be continued for several minutes. In the thigh where the muscles are powerful and long-bellied and when the fracture is mechanically unstable (because the fragments are not in apposition or because the fracture line is oblique) the tendency is to produce overriding of the fragments. Therefore most fractures of the shaft require traction throughout the period of immobilization. Subsequent check radiographs are essential.

Patient A sustained a simple fracture of the shaft of the left femur at the junction of the middle and lower thirds as a result of being struck by a piece of broken cage engine during winding operations. Using the principle of *balanced traction*, 25 lb. weight was hung over a pulley at the foot of the bed by a Kirschner wire driven through the tibial tubercle and a stirrup fixed to the pin on a Braun splint. The counter-traction was the patient's body weight sliding down the bed, which was raised at the foot.

The final reduction of displacement was completed in three days and was then maintained with a 10 lb. weight; the reduced position was continuously and without interruption immobilized for eight weeks, when a plaster spica was applied. Movements were encouraged while using skeletal traction.

Discussion. —Slow union of fractures had occurred with increasing frequency in recent years. There could be no doubt that this was associated with over-correction by excessive pull, which

separated the fractured surfaces and distracted the fragments, and especially with over-correction continued for several days or weeks by the suspension of heavy weights from a skeletal traction pin. There was even greater danger when traction was used to control alignment as well as to prevent over-riding (i.e., inadequate immobilization). Fractures which would otherwise unite in 8 or 10 weeks united after distraction only in 8–10 months. Such delay was entirely unjustifiable; it would be much better to control instability and prevent redisplacement by internal fixation.

Patient B sustained a compound oblique fracture of his left femur about the middle third (as well as a simple fracture right femur, fractured pelvis, simple fracture left ulna, several lacerated wounds and shock) as a result of fall of rock on the 13th May, 1953. (Fig. 1.)

FIG. 1

Shock was treated and both femurs treated by *combined fixed and balanced traction* with double Thomas's splints, traction being exerted from the fixed point of the patient's pelvis. The traction tapes were tightened. The ends of the splints were tied to the foot of the bed, which was raised about 18 in. The right femur united in eight weeks.

In the case of the left femur union was so slow that some form of internal fixation was obviously preferable. The wound was skin-grafted five weeks after admission and when completely "taken" (a month later) under rigid aseptic technique, using a no-touch technique, a cut was made down to the fracture, the bone ends freshened, the fragments were "angulated" into position and internal fixation with a No. 3 vitallium plate

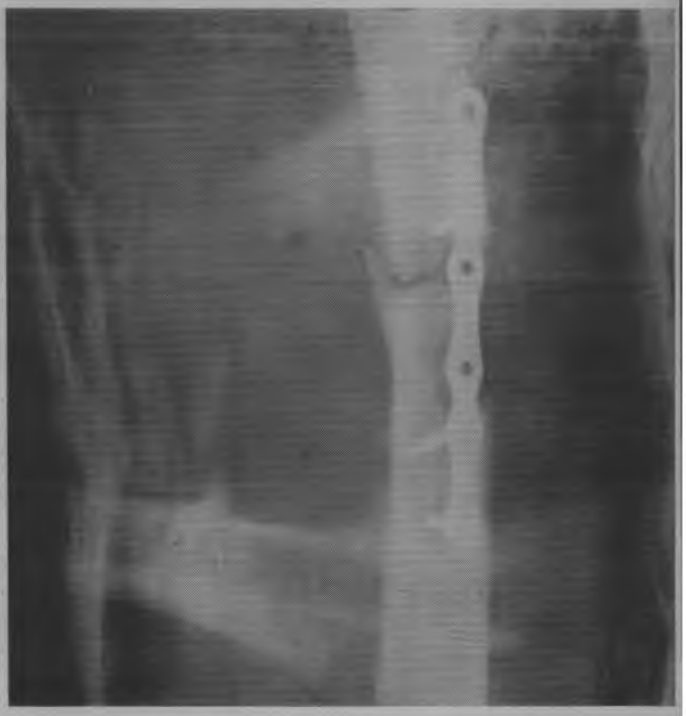

FIG. 2

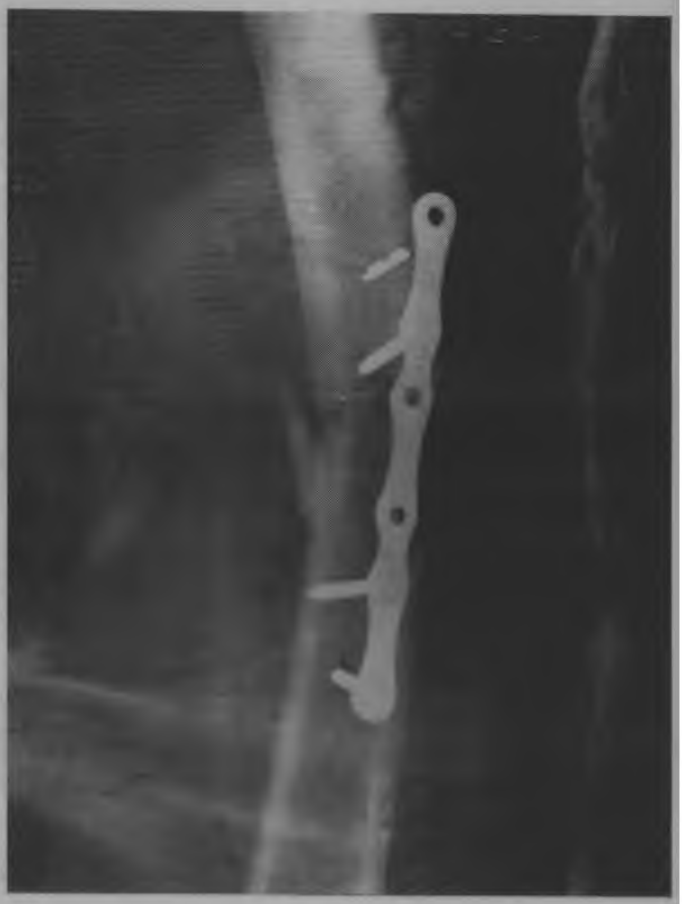

FIG. 3

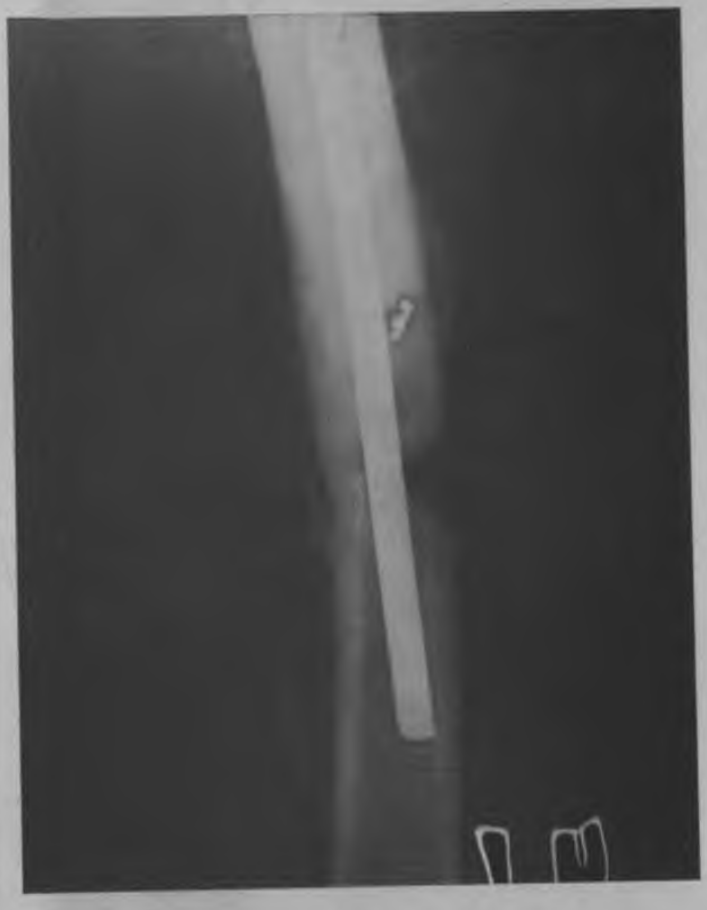

FIG. 4

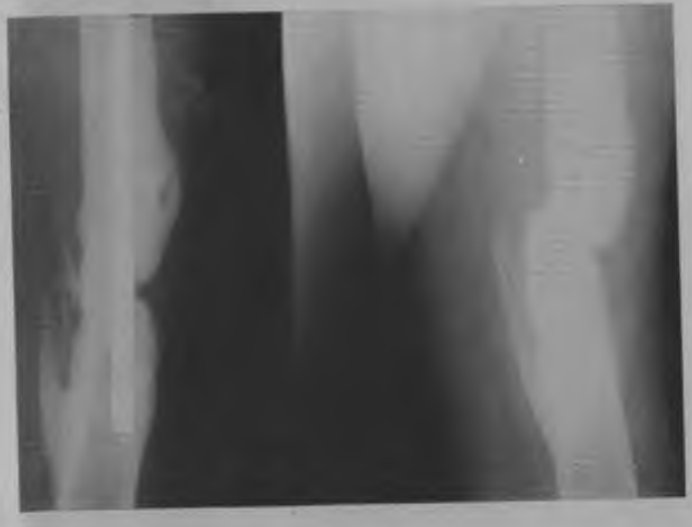

FIG. 5

and four screws effected. The plate and screws were protected from every strain by complete immobilization by a full-length plaster spica cast (Fig. 2). Vigorous chemotherapeutic control (Abbomycillin, Erythrocin and sulphatriad) ensured a smooth post-operative period.

Unfortunately, the plaster spica was cut above the knee after two months, and the screws pulled out (Fig. 3). It was therefore decided to use an intramedullary nail. A Küntscher nail was inserted after freshening bone ends under a spinal anaesthetic on 22nd April, 1954 (Fig. 4). A Thomas's extension was used for about six weeks. Good callus had now formed (nine months after operation) (Fig. 5).

Discussion.—The metal plate and screws immobilized the fragments perfectly. One had therefore been tempted to discard the external splint (i.e., plaster spica) too soon. Complete and prolonged external splinting was indispensable because the fixation secured by the plate was not sustained. Bone reacted to abnormal pressure by resorption, and the more tightly the screws were driven in, the more likely was it that they would loosen. Within a few weeks the fracture was dependent on external splinting for its immobilization, and rotation and shearing strains led to the usual, indeed inevitable, sequel of non-union. Complete immobilization by full-length plaster casts had to be continued until union was sound. A technique that permitted rapid ambulation of patients led to the invention of intramedullary nailing of shaft fractures.

Patient C sustained a badly impacted comminuted fracture of his left femoral shaft during a fight in the compound on 31st October, 1954 (Fig. 6). Fourteen days later, the optimal time, using the most scrupulous aseptic technique, operative reduction was done. The shaft was exposed, using Henry's incision extending from anterior superior iliac spine to the outer border of the patella. The deep fascia was divided and the vastus lateralis separated from the rectus femoris. Under these lay vastus intermedius. The nerve to the vastus lateralis and the descending branch of the lateral circumflex artery formed a neurovascular bundle crossing the upper border of the vastus intermedius. They were retracted upwards and the muscle divided down to the bone. A Hansen-Street nail was introduced, using the retrograde method

FIG. 6

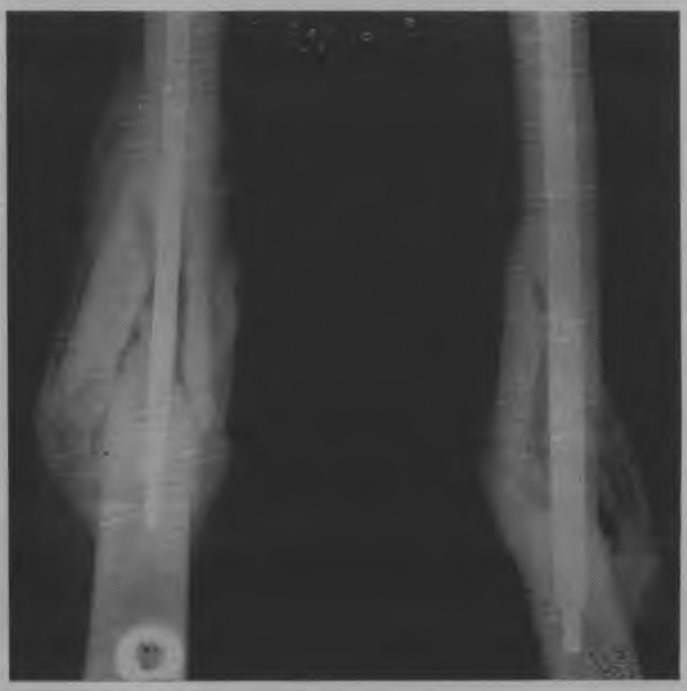

FIG. 7

The patient started walking 10 days later; good callus had already formed after two months (Fig. 7). Every joint of his lower limb had normal mobility. The muscles were well developed, wasting had been minimized, and disuse porosis of the bones had been prevented.

Perhaps the most important point was that the lower limb had never been disconnected from the brain; the patient had not forgotten how to use it; he was not terrified at the prospect of using it.

Discussion.—The range of movement of the knee joint was much more limited when immobilized in plaster than when an intramedullary nail was used. The cause of adhesions and joint stiffness was usually functional inactivity and disuse.

In the discussion, members pointed out that the intramedullary nails used in two cases were too short and, in any case, should perhaps be reserved for fractures of the upper third of the femur. Various reasons were advanced to explain the cause of stiffness of the knee, such as muscle adherence, capsular shortening, etc. The view was also expressed that the conservative treatment of femoral fractures did not necessarily result in stiff joints, provided there was good physiotherapy from an early stage. It was also stated that balanced traction on a Thomas's splint was to be preferred after a plating operation, as a plaster cast resulted in stiffness; the Braun frame was not suitable.

Three Cases of Swellings of the Neck. **Dr. E. Kruger.**

Case A, an adult male Shangaan aged 44 years, was admitted complaining of swellings in his neck, left axilla and both groins for six weeks. They were not painful. The right side of the neck enlarged first, then the left side followed one week later. The groins and left axillary glands started later. All the swellings enlarged gradually. He noticed no interference with swallowing, breathing, speech or sight.

Examination.—The patient did not look ill. Temperature 99·6°F. P.R. 78. Blood count normal. There were large multiple oval swellings with normal overlying skin, no pulsation or translucency on both sides of the neck. They did not feel warm or tender; all were multiple, superficial and discrete. The swellings were soft with a smooth surface with a very well-defined edge and situated in the submaxillary, carotid and occipital triangles of both sides of the neck.

The glands in the left axilla and both groins had the same characteristics.

Biopsy.—Hypertrophic tuberculous adenitis.

Case B was a Hlubi aged 22 years, with a swelling of his neck since childhood (he came from Mount Fletcher). As far as he knew, it had not fluctuated in size nor had it grown bigger. It was not painful and was not associated with pressure effects or endocrine disturbances.

Examination.—There was no hyperfunction. There were two irregular, localized, asymmetrical swellings in the region of the thyroid gland, which did not pulsate but moved with swallowing. The consistency varied but was as a rule fluctuant. While the general surface was irregular, the surface of the lobules was smooth. The edges were well defined and presented no evidence of extension. The lymph glands of drainage were not enlarged, nor was there any evidence of dissemination.

Diagnosis.—Multiple non-toxic cystadenomata developing at puberty. One member thought the condition was foetal adenoma of the thyroid; another thought it was a colloid goitre.

Case C was admitted with influenza and on inspection a swelling was noticed on the right side of his neck, which had been there for many (?) years. It was not painful and had not grown in size or fluctuated, he said. There was no history of any possible cause.

Examination.—The swelling was the size and shape of an egg and did not pulsate. It did not move with deglutition or protruding the tongue and was not translucent. On palpation the swelling was fairly soft and smooth, with a well-defined edge and appeared to be in the sterno-mastoid muscle. There was a gland palpable behind the muscle.

Special Investigation.—W.R. negative.

Differential Diagnosis.—(1) Fibro-myxo-sarcoma commencing in the muscle sheath with very slow infiltration. (2) Desmoid, which was sometimes reported as chronic infiltration. (3) Lymph-gland enlargement behind the muscle traversing it to reach the surface.

It was suggested that aspiration should be done, followed by anti-tuberculosis therapy in case the swelling was tuberculous in origin.

Complicated Dislocation of the Elbow Joint. **Dr. J. H. Marks.**—A Xosa male was admitted to hospital, having fallen from the bolster bar during the Christmas festivities. He had a posterior dislocation of the right elbow joint with no demonstrable nerve or vascular injury. X-ray showed in addition to the dislocation that the medial epicondyle had been avulsed (Fig. 8). Under pentothal anaesthesia the dislocation slipped back easily and on casual inspection of the post-reduction X-rays the bones appeared to be in alignment, but more careful scrutiny disclosed that the bones were not in apposition and that the epicondyle was included in the joint space (Fig. 9). The padded cast was removed and further views taken (Fig. 10).

FIG. 8

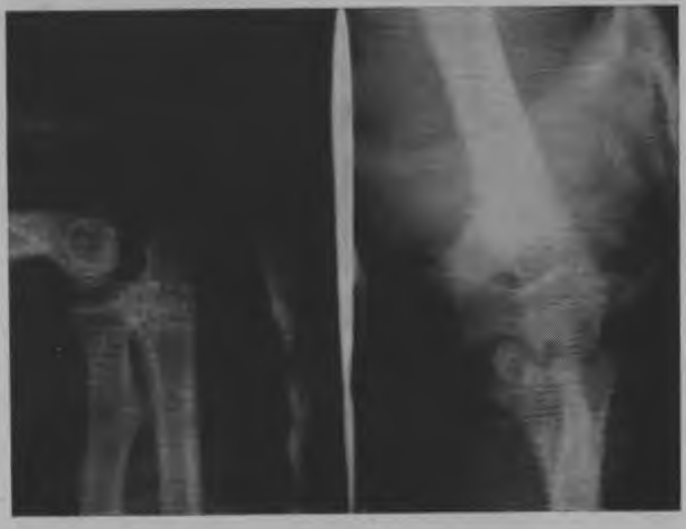

FIG. 9

Open operation was undertaken on the sixth day when the skin was in a fit state for incision. Pre-operative examination showed no evidence of nerve compression. Under brachial plexus block, a medial curved incision was centred over the site of the epicondyle and extended two inches either way. This exposed a triangular rent in the capsule of the joint, from which emerged the flexor origin of the muscles. The ulnar nerve and accompanying arteries were seen to enter the rent, the nerve emerging distally behind the muscles. By forcing the elbow into valgus, the epicondyle and the

nerve were easily extracted. After freeing the nerve proximally for about two inches, and distally as far as the origin of the muscular

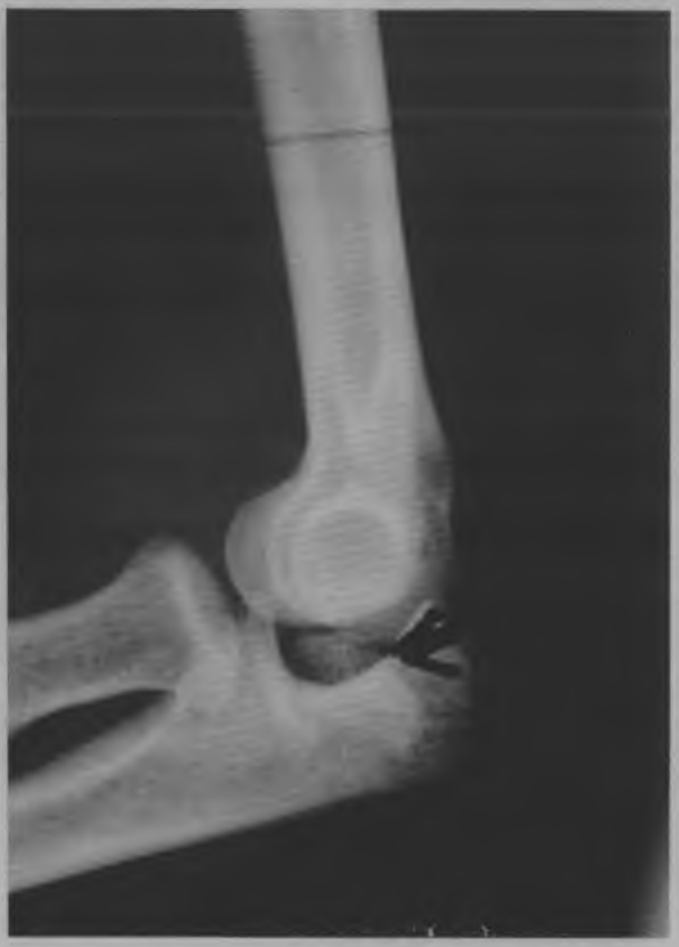

Fig. 10

branches, the nerve was transposed anteriorly and embedded in the flexor muscles for a short distance. The epicondyle and the attached muscles were sutured to the humerus distal to the original insertion to minimize drag. Catgut sutures were employed and passed through the periosteum of the humerus and the muscle origins. The joint capsule was not sutured. After skin suture a padded cast was applied (Fig. 11). Three weeks later the cast and sutures were removed, and an unpadded cast applied for a further 10 days. When the cast was finally discarded (four days before presentation), the elbow was found to have 5 degrees of movement either way from the mid-position. This was now improving and *active movements* only were being used. An almost complete return to normal was anticipated.

Obstruction of the Pylorus by Tuberculous Glands.

Dr. J. H. Marks.—A Xosa male was admitted to hospital on 15th November, 1954, complaining of

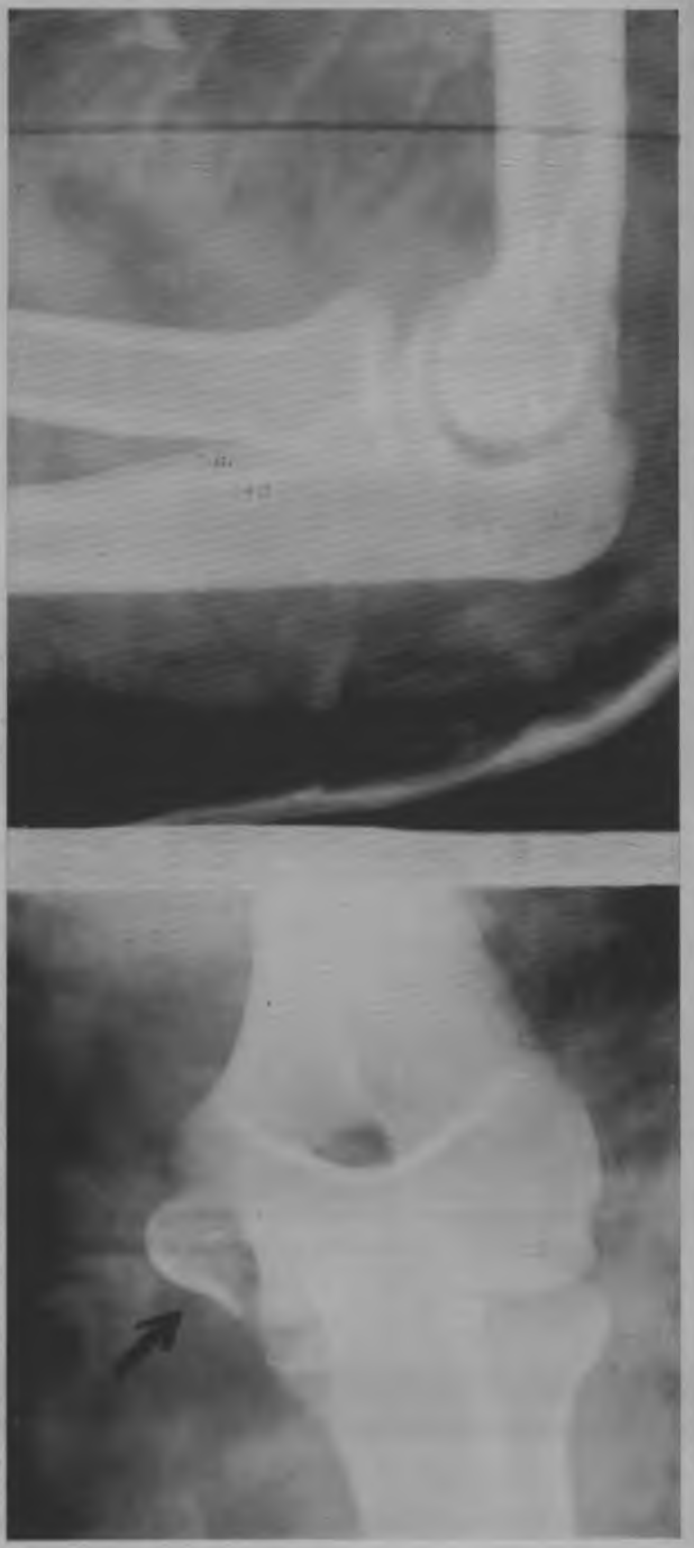

Fig. 11

abdominal pains and vomiting of five months' duration. He had lost a lot of weight, but had not suffered from sweats. He complained of nausea two hours after meals, which was often followed by vomiting (no blood). There was sometimes several days' interval between vomits and he noticed food which had been ingested several days before. The pain also occurred two to three hours after meals and was griping in character for a few hours, or was relieved by vomiting or eating a little more food, but overeating aggravated the pain. A week prior to admission the stools had been tarry for several days. In the past five months (since engagement) his weight had dropped from 103 to 89 lb.

Examination.—Dehydrated, marked loss of skin elasticity. Fullness of the epigastric region. Visible peristalsis. Vomited 72 ounces of mucus (no bile present).

Differential Diagnosis.—(1) Pyloric obstruction due either to a ball-valve tumour or to a peptic ulcer of the duodenum or pylorus. (2) Biochemical disturbance, particularly hypochloraemia and hypopotassaemia following repeated copious vomiting. (3) Tubercular hilar glands.

During the next few days he vomited frequently, as much as 60 to 125 ounces, and was treated with belladonna, alkaline powder and phenobarbitone, and a Meulengracht diet. A barium meal was done on 6th December, 1954. Marked pylorospasm was present and very little barium was seen to pass through into the duodenum. There was no evidence of infiltration into the gastric wall. The conclusion was "duodenal ulcer with marked spasm.' On 29th December a barium meal revealed no evidence of ulcer or filling defect. Peristalsis was marked and pylorospasm persisted for an hour. Two days later he vomited 138 ounces, and two weeks later had a haematemesis. On 17th January, 1955, his abdomen was opened by means of a right paramedian incision. There were numerous fine miliary tubercles on the serous surface of the colon. The stomach was dilated and the pylorus was obstructed by a mass of glands which almost encircled the duodenum from behind. A posterior gastroenterostomy was done (three-finger stoma). Before final closure a finger was inserted into the stomach and the obstructing glands could be felt bulging into the lumen of the pylorus. 7th February, 1955—weight 115 lb. Asymptomatic. Ambulant.

Bile Peritonitis following Ruptured Liver. **Dr. E. W. Crews.**—A Fingo aged about 24 years was admitted to hospital on 23rd December, 1954, having been injured by a fall of hanging. His injuries were abrasions of the right chest wall over ribs 5, 6 and 7 and abrasions of the right elbow. On examination he was found to be severely shocked. Pulse rate 112, B.P. 86/54 mm. Hg. He was tender over ribs 5, 6 and 7 and also in the right hypochondrium. The abdomen was soft. He was put on a half-hourly pulse chart and treated expectantly. On the following day his pulse rate had increased to 130, he was very anaemic and his abdomen was slightly distended and shifting dullness was elicited. Hb. 52%. He was given transfusions of blood and laparotomy was decided upon. At operation an upper midline incision revealed the peritoneal cavity to be filled with blood. The spleen was found to be intact and as the blood appeared to be coming from the liver, a transverse incision was carried round the right costal margin. A large 3 inch tear on the superolateral surface of the liver could now be felt, which, due to its position, it was impossible to suture. The tear was packed with gelatin sponge (Spongiostan) and the incision closed without drainage. The temperature and pulse rate gradually fell until the sixth day when it rose again and a small swelling could be felt in the right hypochondrium. This swelling gradually increased in size and on 18th January, 1955, it was aspirated and 600 c.c. of bile removed which showed coliform bacilli on culture. Since then the patient had made steady progress and the swelling had not reappeared.

Comment.—Treatment of Liver Injuries.—Ian Aird stated that treatment should be obstinately conservative and that operation should be undertaken only if circulatory collapse was dangerously progressive in spite of transfusion. Rodney Maingot stated that as soon as the diagnosis of ruptured liver was made or suspected, the patient should be prepared for operation. This preparation could take one or two hours or longer but should never be so prolonged as to jeopardize the patient. Most authors agreed that suture of the liver was preferable when possible.

Bile Peritonitis.—Claremont and Von Haberer (1910) described a case of bile peritonitis due to transudation of bile through the gall bladder. Leriche described a case in which at laparotomy bile was found to be oozing through a distended gall-bladder. Burkitt wrote on biliary peritonitis without demonstrable perforation as follows:—"The clinical picture has shown wide variations and cases have been described simulating perforated peptic ulcer or with less sudden onset

resembling peritonitis from perforation of the appendix or still more gradual in nature, with increasing abdominal distension, vomiting and rising pulse rate (Rolleston 1938)."

In cases with a relatively acute onset, which constituted by far the greatest number, remission of symptoms after the first acute attack had been the one almost constant feature (Cope). Furthermore, cases were on record where over 30 pints of bile, presumably diluted by ascitic fluid, had been aspirated over a period of six weeks.

Pathogenesis.—The source of the bile which leaked into the peritoneal cavity in spontaneous bile peritonitis had occasioned much conjecture. Sometimes stones were found to be present or cholecystitis but equally often no pathology had been present. Perforation was seldom found. The following causes of leakage had been suggested :—

(1) Escape through a tiny perforation which healed leaving no trace.

(2) Rupture of the fundus of a deep mucous gland from increased biliary pressure with healing as soon as pressure was relieved.

(3) Transudation from subcapsular bile channels on under surface of liver.

(4) Leakage through cholangitic ulcer.

(5) Erosion of bile duct wall by regurgitation of pancreatic juice.

Members commented that is was not the bile but the superadded infection which was harmful to the patient.

The following report has been received from the Ernest Oppenheimer Hospital, Welkom, O.F.S.

Heat Stroke. **Dr. E. M. McLean.**—Two cases of heat stroke were treated recently. The successful management of the patient depended on teamwork, the same method being adopted in both cases.

On admission the patient was placed in bed naked on a waterproof sheet, with an electric fan at the foot of the bed and a large bowl of ice cubes and iced water near at hand. A lumbar puncture having been performed to exclude meningitis, treatment was started by a team consisting of the medical officer, a nursing sister and three native orderlies.

The medical officer supervised the treatment and kept note of the condition of the patient. The sole duty of one native orderly was to record the rectal temperature, the thermometer being kept *in situ* and removed every 60 seconds to record the reading. The other two native orderlies sponged the patient continuously with iced water. A.C.T.H. 25 units and cortisone 50 mg. were given intramuscularly at the earliest possible moment. When the temperature fell below 102° the cold sponging was discontinued, the patient was dried and covered with a sheet only.

Case 1.—A Xosa was admitted shortly after collapsing underground. He had been working underground for a period of three weeks. He was restless and semi-conscious. Rectal temperature 108°. Pulse 150. Skin hot and dry.

Lumbar puncture—normal cerebrospinal fluid not under pressure.

A.C.T.H. and cortisone were given and sponging commenced. After 45 minutes the temperature was 102°. One hour and five minutes after treatment was commenced his temperature was normal.

This patient developed a severe haematuria on the third day. The centrifugalized deposit showed numerous bilharzial ova. He was treated with Nilodin and Anthiomaline.

On the second day he had a herpes simplex.

After thirteen days in hospital he was transferred to surface work.

Case 2.—A young Basuto with only two weeks of recent underground experience was admitted after collapsing underground.

He was semi-conscious and very restless. Rectal temperature 107°. Pulse 130. Skin dry and hot. C.S.F.—normal.

A.C.T.H. and cortisone were administered intramuscularly.

After 60 minutes the temperature had dropped to 102° and cold sponging was discontinued. One hour and fifteen minutes after the commencement of treatment the temperature was 98°. During the next half hour the temperature continued to fall and reached 95°. At this stage the patient suffered a severe collapse, which responded to coramine.

He was discharged to surface work five days after admission.