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DEWAR
ORTHOPAEDIC CLUB

GLORIA

ORTHOPAEDICA

TENTH ANNIVERSARY

Dr. F. P. Dewar

INTRODUCTION

A great leader in medicine leaves, as part of his legacy, his students and a worthy successor to carry on his work. Thus it was that when, after a long and distinguished career, the late R. I. Harris retired, his place was taken by one of his students, Frederick Plummer Dewar. Now F. P. Dewar's own students, on the Tenth Anniversary of the Dewar Club, wish to dedicate a series of their papers to a second great leader in orthopedic surgery in Toronto.

Following the Second World War, it became apparent that a training course in orthopedic surgery was needed to match the highly successful Gallie Course. R. I. Harris laid the groundwork and F. P. Dewar, in the relatively short time since 1951, has brought the course to a high standard.

The standard of academic endeavour set by "R.I." was high and the work done to establish the training course was added to an already heavy teaching and service load. Furthermore, this task was carried out in an environment that was oriented towards the training of the complete general surgeon. Despite this and some initial misgiving on the part of other specialties, a highly successful training course has been established through the quiet, patient determination which characterizes "Ted" Dewar.

The man honoured in this presentation has many other attributes that have contributed to his achievement; chief among these is his willingness to efface himself, encouraging thereby the co-operation of his colleagues—difficult at the best of times for "A Chief". This outstanding attribute—this gracious self-effacement—has given to all those involved in orthopedic teaching at Toronto the feeling that they belong to the team and share a real measure of responsibility.

At present there are 24 postgraduate students in various stages of training. Seven hospitals are engaged in the teaching program, which includes training in basic surgery, plastic surgery, neurosurgery and orthopedic pathology. A complex rotation ensures that each candidate is exposed to the thinking and practices of all the orthopedic surgeons in this centre—for better or worse. At some stage, most candidates have the opportunity to engage in clinical or basic research and, finally, once the Fellowship is passed, almost all graduates of the program have been able to go as graduate fellows to see what is going on in other parts of the world, chiefly through the support of the McLaughlin Travelling Fellowships. With regard to the Royal College examinations, I am proud to say that, to date, no graduate has failed to pass his Fellowship, although some have required a "second go".

It is to such a leader and such a system that the members of the Dewar Club pay tribute on their Tenth Anniversary.

Few endeavours are more gratifying than that of training one's successors. We of "The Team" feel privileged to play a part in such training under a director whose leadership is superb, whose friendship we cherish, and to whom we wish unbounded continuing success.

G. F. Pennal

THE DEWAR ORTHOPAEDIC CLUB: A SHORT HISTORY

Former residents on the orthopedic service of the Toronto General Hospital, who served under F. P. Dewar, its chief, held organizational meetings in 1959, and founded the "Dewar Orthopaedic Club" when eight orthopedic surgeons met on March 28, 1959.

The purposes of this club were the advancement of orthopedic surgery through the sharing of experiences, ideas and ideals, the stimulation of academic interest in the practice of orthopedic surgery, and the fostering of scientific investigation and presentations.

Membership is open to that resident each year who holds the appointment of Chief Resident on the service of F. P. Dewar. Each in rotation serves as president, vice-president, program chairman and secretary-treasurer. Meetings have been held annually and each member either presents or submits in writing at least one paper on an orthopedic or related subject to each meeting.

The founding meeting of the club was held at the Ottawa Civic Hospital and subsequent annual meetings were held at the Hotel-Dieu Hospital, Quebec City and Manoir St. Castin; the Hotel-Dieu Hospital, Kingston, and Lake Placid Club, New York; St. Joseph's Hospital, Toronto and Muskoka Sands Inn; the Toronto East General and Orthopaedic Hospital and Guild Inn, Toronto; St. Joseph's Hospital, Hamilton and the Royal Connaught Hotel, Niagara Falls; the Hotel-Dieu Hospital, Montreal and Mont Gabriel Lodge, Quebec; the Kingston General Hospital and Seigniory Club, Quebec; the New Mount Sinai Hospital, Toronto and Talisman Ski Resort; the Ontario Crippled Children's Centre, Toronto and Muskoka Sands Inn.

The 10 annual meetings have been well attended and more than 150 scientific papers have been presented. The present membership—18 orthopedic surgeons—includes graduates of seven different Canadian medical schools and, at present, represents the surgical departments of six Canadian medical schools.

The members of the club find their association richly rewarding and look forward in anticipation to each annual, late winter meeting, which provides the opportunity for the sharing of experiences and ideas with each other, Dr. Dewar and the honorary guests of the meeting.

The Tenth Anniversary of the founding of the Dewar Orthopaedic Club is being celebrated by the publication of papers of wide surgical interest contributed by members of the Club. These will appear in this and a following number of the *Canadian Journal of Surgery.* This has been made possible by the energy of the members of the Club's editorial subcommittee (Drs. D. E. Hastings, E. H. Simmons and G. A. McDonald) and the sympathetic co-operation of Dr. F. G. Kergin, Chairman of the Editorial Board of the Journal.

<div align="right">

J. W. Hazlett
Archivist,
Dewar Club.

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F. P. DEWAR: A TRIBUTE FROM HIS COLLEAGUES OVERSEAS

In over 40 years in orthopedic surgery, it has been interesting to watch the growth and development of the specialty in various parts of the world, and to note the part played by individual orthopedic surgeons, e.g. Robert Jones in Great Britain, Putti in Italy, Murk Jansen in Holland, Haglund and Waldenstrom in Sweden, Guildal in Denmark, Kirmisson, Nove-Josscrard and Ombredanne in France, Lorenz in Austria, and so on. But none of these has fascinated me more than the growth of the specialty in Canada and particularly in Toronto. Here the surgical scene was dominated for many years by surgical giants with a vast operative repertoire like Starr, Gallie, D. E. Robertson, Le Mesurier and R. I. Harris. R. I. Harris was probably the first to break away and concentrate on orthopedics alone. After a struggle to confirm the position of the specialty in Toronto and Canada, he was to live to see himself held in the highest regard throughout the English-speaking world as one of the important father-figures in international orthopedic surgery. But always in my pre-war journeys to Toronto, I came away with the feeling that here was a lone figure fighting a lone

battle. Clearly the time was not then ripe for expansion.

After the war changes began to occur. Young men coming back from visits to the North American continent began to report on an excellent Toronto school of orthopedics now under the guidance of Ted Dewar. On my last visit to that city in 1965 as visiting professor to an orthopedic postgraduate course in the University of Toronto, I was able to see for myself the enormous advances that had been made. I was immensely impressed by the quality of the day-to-day orthopedic work, by the research and training programs, and especially by the close *camaraderie* between Dr. Dewar and his colleagues at all the hospitals I visited in Toronto. It is alas only too common in many, if not most, cities of the world to find orthopedic surgeons in covert, often overt, personal and professional hostility to their colleagues. It is the absence of this, among many other qualities, that has made Toronto unique in the world of orthopedics today. Even taking into consideration the support of the Professor of Surgery, the Dean and other members of the academic board of the University, it is to Ted Dewar with his qualities of intellect, foresight, leadership, ability to choose the right men and inspire them, and almost above all his capacity for friendship, that the greatest praise is due for establishing the Toronto school of orthopedics as one unsurpassed anywhere in the world today.

H. Osmond-Clarke, C.B.E.
Orthopaedic Surgeon to
Her Majesty The Queen.
Past-President, British
Orthopaedic Association.

DEWAR CLUB PAPERS

TALAR AND SUBTALAR TILT: AN EXPERIMENTAL INVESTIGATION*

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and ROBERT ST-JACQUES, M.D.,[†] Montreal, Que.

THE object of the investigation described in this communication was threefold: (1) to study the lateral mobility of the normal talus,[1] (2) to determine the function of the various ligaments on the outer aspect of the ankle[2] and (3) to investigate subtalar motion.[3]

CLINICAL ASPECTS

During the clinical phase of the project, the physiological limits of laxity in a normal ankle joint were determined in 92 subjects from 6 to 60 years of age who had no history of previous injury or systemic ligamentous instability (Table I). The lateral

TABLE I.—SUBJECTS IN THE STUDY.
NORMAL TALAR TILT

Number of subjects............	92
Average age...................	30 years (6 to 60)
Number of films..............	633

mobility of the talus within the ankle joint was determined using a special apparatus which incorporates a tensometer and a goniometer; the ankle joint can then be examined in forced eversion and inversion using a known, standardized force (Fig. 1).[4] The talar-tilt angle was calculated from lines drawn along the articular surfaces of the talus and tibia (Fig. 2).

The normal talus does tilt laterally and clinical inversion can occur through a normal ankle joint. The average talar tilt was 7° with a range extending from 0 to 27° (Table II). The talar tilt was more marked in younger children but a lateral talar tilt is not uncommon in normal young adults between the ages of 20 and 30.

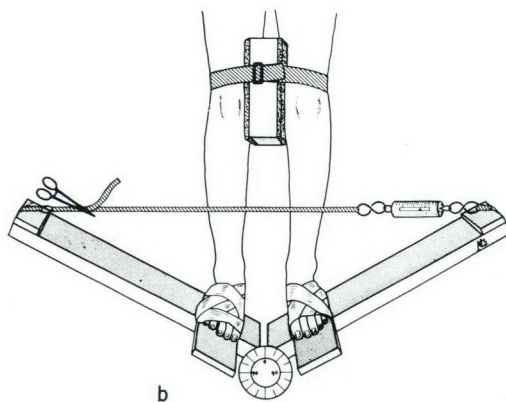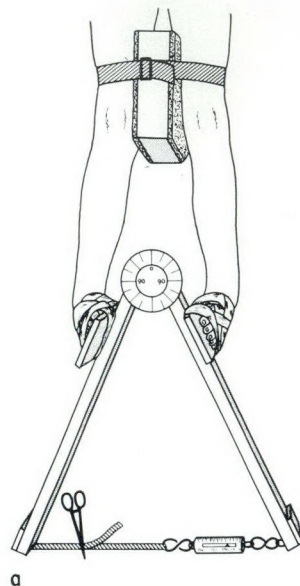The degree of talar tilt depends on the position of the ankle when the stress films

Fig. 1.—Apparatus to standardize the examination of the ankle joint: (a) inversion and (b) eversion.

are made, being greater in a position of equinus. Since the superior articular surface of the talus is narrower posteriorly, it is not surprising that the astragalus is more unstable in the position of equinus; but even in 90° of dorsiflexion (the position of the foot during this investigation), the normal talus does tilt during inversion.

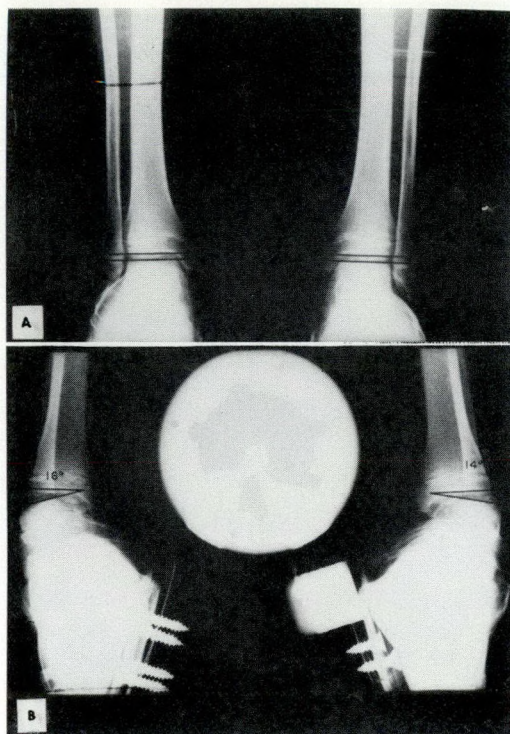Although the average talar tilt of right

Fig. 2.—A normal ankle in (A) eversion and (B) inversion. In eversion, a normal mortise is seen. In inversion, there is a talar tilt and the lateral malleolus is superimposed on the talus.

and left ankles was the same, it need not be identical in the two ankles of any one individual; however, physiological asymmetry never exceeded 15° and was usually less than 10°. It should be pointed out that, although a normal talus tilts in inversion, it never does during eversion (Fig. 2).

Since clinical stress films are usually performed manually, the examination was

TABLE II.—PHYSIOLOGICAL TALAR TILT. AVERAGES. INFLUENCE OF AGE, EQUINUS AND TIBIAL ROTATION

Normal talar tilt		Talar-tilt angle (degrees)
Average (range 0 - 27°)		7
Effect of age (years)	6 - 9	7.5
	9 - 15	5
	20 - 60	3
Effect of degree of equinus	90°	5
	130°	6
Effect of rotation:		
Unrestricted rotation		5
Restricted rotation		2

repeated manually when a talar tilt was demonstrated using the apparatus. The degree of talar tilt was the same with either method; this suggests that the physiological talar tilt is easy to demonstrate and that an excessive force need not be applied. These examinations were done without any anesthesia and the subjects did not complain of discomfort during the stress films. Since the object of this investigation was to demonstrate the limits of normal lateral motion, the examination had to be painless. The ligaments were not under excess strain and the mobility thus demonstrated was physiologic.

A surprising and constant radiological image seen during stress inversion was the superimposition of the lateral malleolus upon the talus and tibia (Fig. 2). This appearance was originally attributed to a posterior displacement of the fibula during inversion; however, lateral radiographs of the ankle, taken in positions of eversion and inversion, revealed no such posterior displacement of the fibula during inversion. Similarly, even after the anterior-inferior tibiofibular ligament is sectioned, there is hardly any movement between the tibia and fibula during forced inversion.

A critical analysis of the stress films reveals that, during inversion, not only is the lateral malleolus superimposed on the tibia and talus, but the contour of the medial malleolus is also different. In other words, in spite of determined efforts to control rotation, external rotation of the whole leg takes place during inversion. This explains the radiological image, but does not dispose of the question: why should the leg go into external rotation when the foot is inverted?

The explanation of this external rotation during inversion is interesting and clinically most significant. The calcaneofibular ligament is oblique and, from above downwards, it travels posteriorly (Fig. 3A). During the movements of inversion and eversion this ligament changes its direction considerably. When the foot is everted, the calcaneofibular ligament is almost horizontal (Fig. 3C) but, in the position of inversion, the ligament is almost vertical (Fig. 3B) as the distal attachment moves forward.
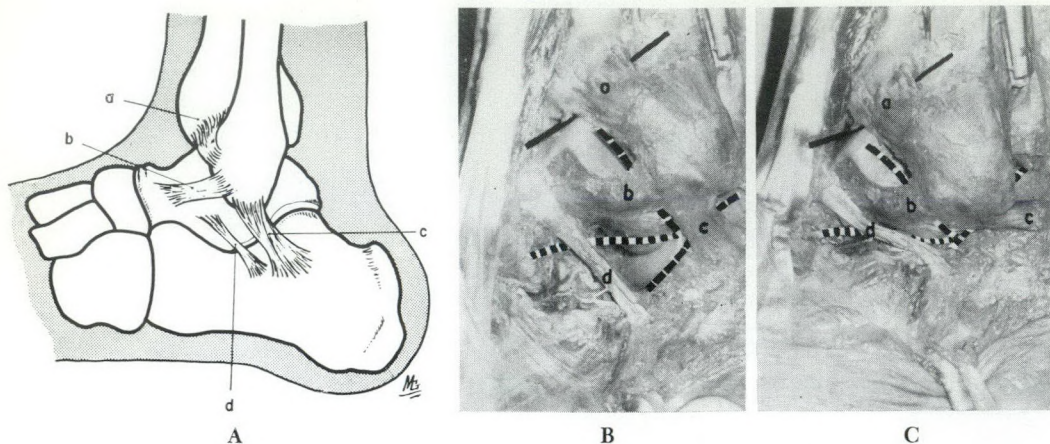
Fig. 3.—(A) Dissection of the lateral ligaments of the ankle and subtalar joints: a—anterior-inferior tibiofibular ligament, b—talofibular ligament, c—calcaneofibular ligament, d—talocalcaneal ligament. (B) Ankle in inversion—the calcaneofibular ligament (c) is almost vertical. (C) Ankle in eversion—the calcaneofibular ligament (c) is almost horizontal.

However, if the distal end of the ligament is fixed, as it was in the apparatus or when the foot is fixed to the ground, the calcaneofibular ligament can only become vertical during inversion by a posterior displacement of its fibular attachment as the calcaneofibular ligament becomes taut. This posterior pull on the fibula is immediately transmitted to the anterior aspect of the lower tibia via the anterior-inferior tibiofibular ligament; hence, the whole leg is drawn into external rotation during the movement of inversion. This mechanism was confirmed repeatedly by cineradiography; as inversion progressed, a normal talus tilted within the ankle mortise and, as it did, the leg was gradually drawn into external rotation.

External rotation of the leg during inversion is an important defence mechanism because it decreases the tension on the calcaneofibular ligament during forced inversion. When such rotation is blocked (Fig. 4), excessive rotation will tear this ligament. In order to confirm this interpretation, external rotation was tentatively restricted using circular adhesive tapes around both calves. Even with such inadequate restriction of rotation, there was an obvious diminution of the talar tilt (Table II) because the calcaneofibular ligament came under tension immediately, that is, as soon as inversion began.

Bearing these observations in mind, a physiological talar tilt can be distinguished

radiologically from a post-traumatic talar tilt. A normal talar tilt is associated with an external rotation of the leg (Fig. 5A) while post-traumatic talar tilt is not (Fig. 5B) because, if the calcaneofibular ligament is torn, the leg is not pulled into external rotation during forced inversion stress films. The difference is not always striking but, when it is correlated with the clinical findings, it becomes most significant. Great care must obviously be taken to keep both knee caps in the sagittal plane, otherwise this distinction cannot be made. Furthermore, the examination should include both ankles for comparison. Any talar tilt that is grossly asymmetrical is suspicious and, if it is not associated with an external rotation of the leg and if the lateral malleolus is not superimposed on the tibia and talus, the tilt is most likely pathological.

ANATOMICAL ASPECTS

The investigation was completed in the anatomy department where the ligaments on the outer aspect of the ankle were systematically sectioned in fresh, mobile cadavers. Stress films were then made to determine the precise role of the following: the anterior-inferior tibiofibular ligament, the talofibular ligament, the talocalcaneal ligament and finally the calcaneofibular ligament (Fig. 3A).
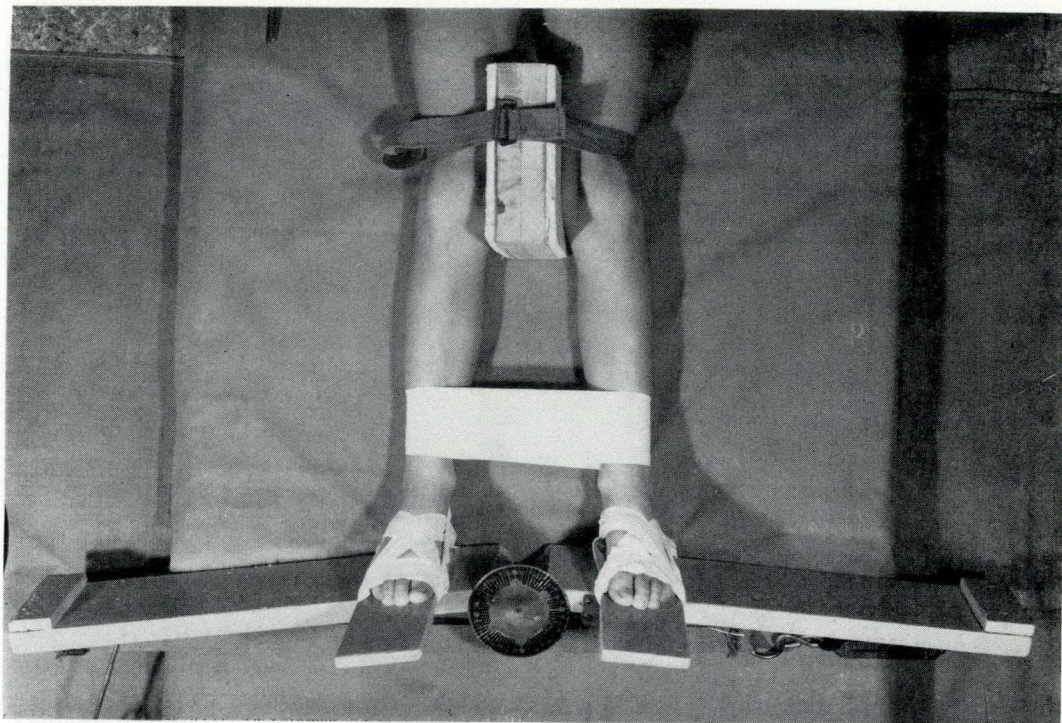
Fig. 4.—The ankles are taped together to control the external rotation of the tibia during forced inversion.
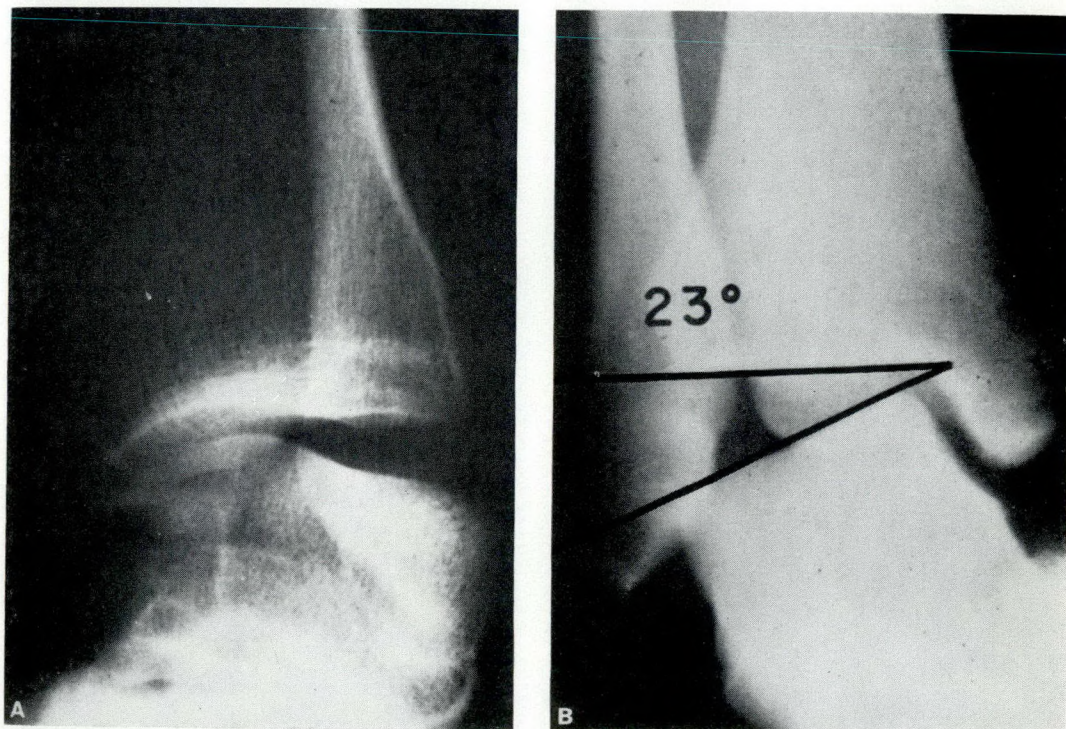

Fig. 5.—Radiological differentiation between (A) physiological and (B) pathological talar tilt. Physiological talar tilt is associated with external rotation and the fibula is superimposed over the tibia and talus. Pathological talar tilt shows the talar tilt without any external rotation.

In one sequence of experiments, the calcaneofibular ligament and then the talocalcaneal ligament were cut successively. After the calcaneofibular ligament was cut, forced inversion produced a tilt of the os calcis underneath the talus, at the level of the subtalar joint (Fig. 6), without any excessive mobility at the level of the ankle joint. Subsequent section of the talocalcaneal ligament increased the subtalar tilt without again affecting the stability of the ankle joint, indicating that these two ligaments are mainly responsible for subtalar stability.

In another group of cadavers, the ligaments were cut in a different sequence, stress examination being performed on each occasion: first, the anterior-inferior tibiofibular ligament was sectioned; then the talofibular ligament, and finally the calcaneofibular ligament was transected. When the anterior-inferior tibiofibular ligament was cut, forced inversion of the specimen produced no lateral instability of the talus (Fig. 7A) and stress films showed no talar tilt. It would appear that the anterior-inferior tibiofibular ligament plays a relatively unimportant role in the stability of the ankle joint.[5] However, when the talofibular ligament was sectioned, the talus was
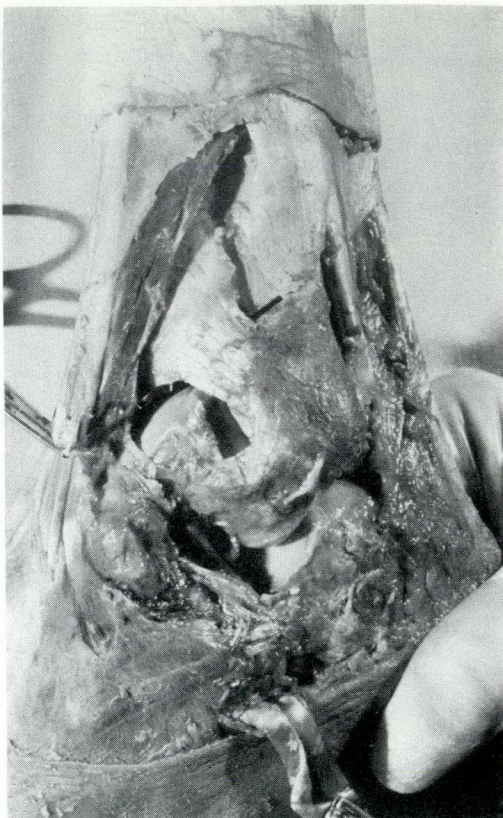

Fig. 6.—Section of calcaneofibular ligament showing that there is subtalar tilt on forced inversion but no talar tilt.
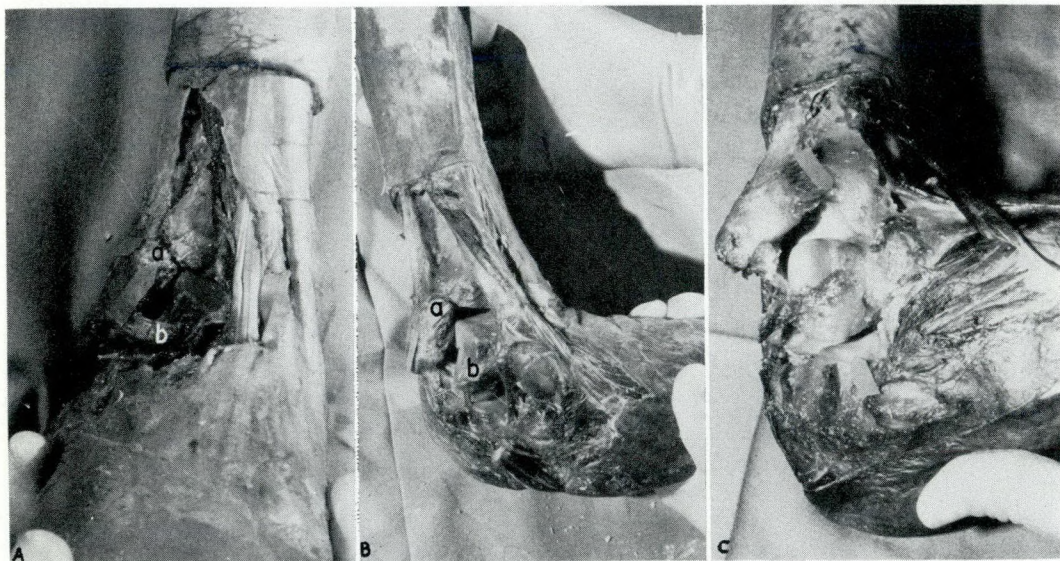

Fig. 7.—Subsequent section of ligaments on the outer aspect of the ankle. (A) Section of the anterior-inferior tibiofibular ligament: forced inversion produces no talar tilt. Note that the talofibular ligament (b) is intact. (B) Following section of the talofibular ligament (b), a talar tilt develops. (C) Following subsequent section of the calcaneofibular ligament, a subtalar tilt also develops.
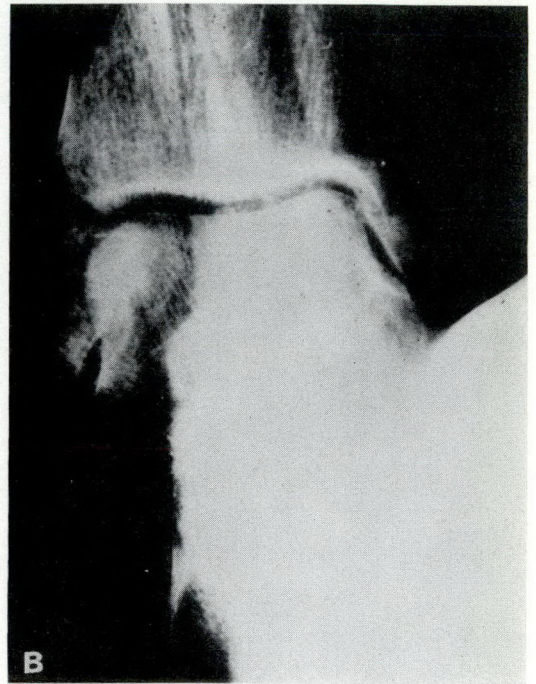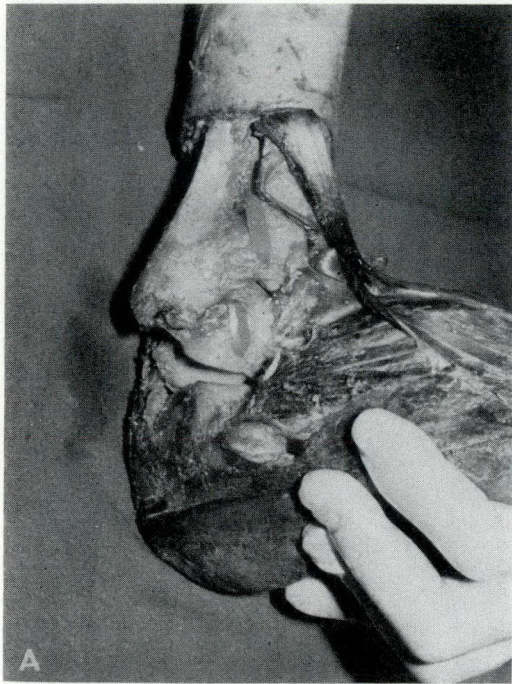
Fig. 8.—Subtalar tilt. (A) Cadaver dissection with an experimentally reproduced subtalar tilt following section of the calcaneofibular ligament. (B) Same specimen showing a subtalar tilt which was overlooked when standard anteroposterior stress films were used. (C) Subtalar tilt visualized using the special technique described in Fig. 10B.

unstable, and stress films of the ankle revealed an obvious talar tilt (Fig. 7B).

The subsequent section of the calcaneofibular ligament produced a tilt of the os calcis below the talus; in other words, there was then a talar as well as a subtalar tilt (Fig. 7C).

These two experiments suggest that the talofibular ligament is mainly responsible for the stability of the talus within the ankle joint, while the calcaneofibular ligament stabilizes primarily the subtalar joint.

By straddling the ankle and subtalar joints, the calcaneofibular ligament automatically gives some support to both joints, but its most important role is to maintain subtalar stability. If the calcaneofibular ligament alone is sectioned, there is a subtalar tilt but no talar tilt; conversely, if the talofibular ligament is cut or torn, there is talar tilt but subtalar mobility is unaltered. If both ligaments are sectioned, the talar tilt is greater and there is also subtalar tilt. It is most likely that severe inversion strains cause tears of both ligaments and that an obvious post-traumatic talar tilt is also associated with a subtalar tilt. In other words, isolated tears of the talofibular ligament are probably rare because, once the latter
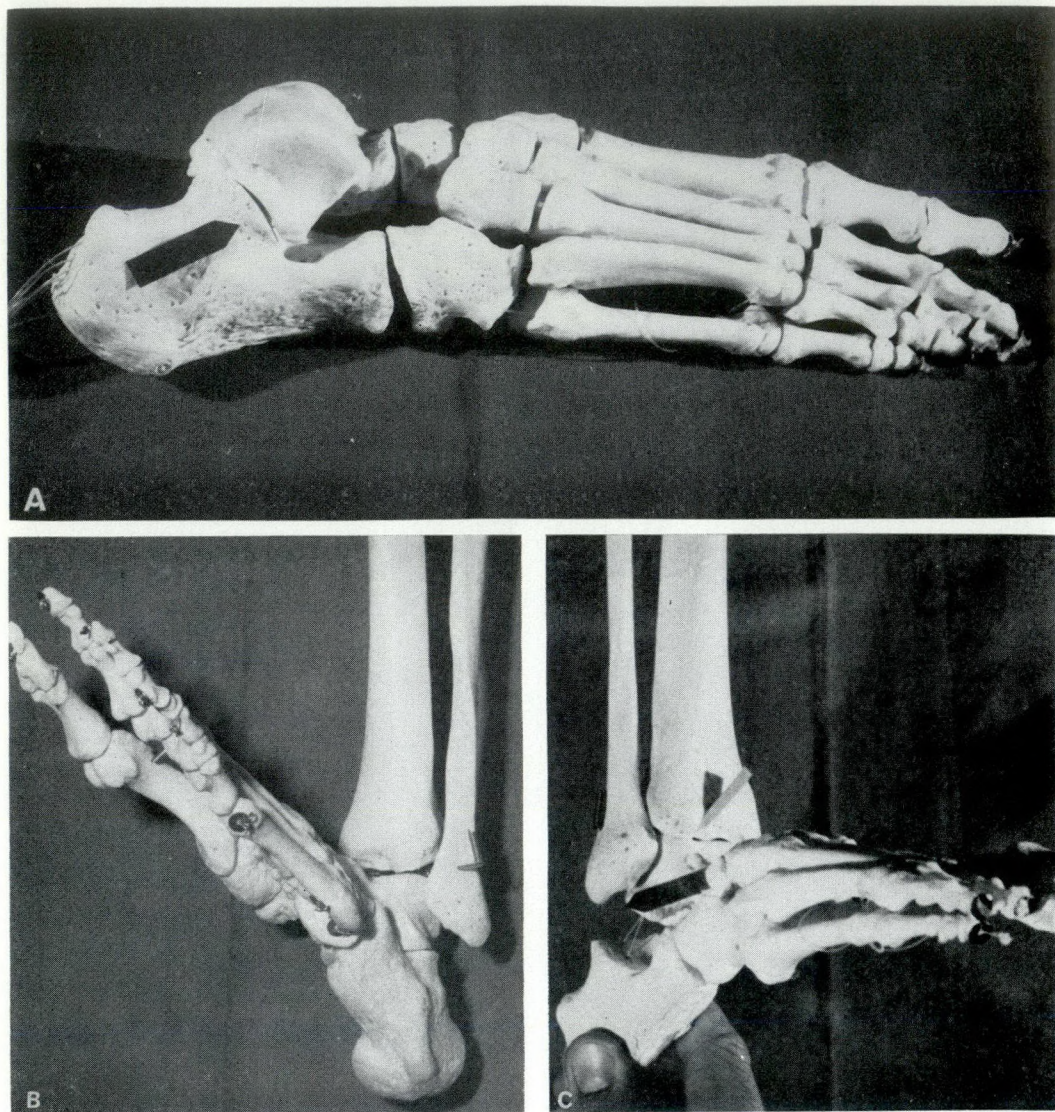
Fig. 9.—Foot skeleton. (A) The posterior subtalar joint is oblique and is at a 45° angle with the plane of the ankle joint. (B) Following dorsiflexion and internal rotation, the subtalar joint is visible and parallel to the ankle joint line. (C) A stress film in dorsiflexion and internal rotation allows the subtalar joint to be seen and any subtalar tilt recognized.

is torn, subsequent stress is then applied to the calcaneofibular ligament which is in turn torn as the talus is pushed downwards. Most appropriately, the Watson-Jones tenodesis repairs both lesions.[6, 7]

However, if the calcaneofibular ligament is torn first (this will be more likely to occur when an inversion strain is applied to a dorsiflexed ankle, since the talus is then stable within the ankle mortise), further strain is not necessarily transmitted to the talofibular ligament and an

isolated subtalar tilt then exists—a lesion that is consistently missed because routine stress films do not demonstrate it (Fig. 8B).

A post-traumatic subtalar tilt is usually overlooked for the obvious reason that it is very difficult to visualize, radiologically, the subtalar joint, let alone to demonstrate a post-traumatic subtalar tilt. Such visualization is difficult because the posterior half of the subtalar joint is oblique, forming a 45° angle with the ankle joint (Fig. 9A). Because of this obliquity, this joint cannot
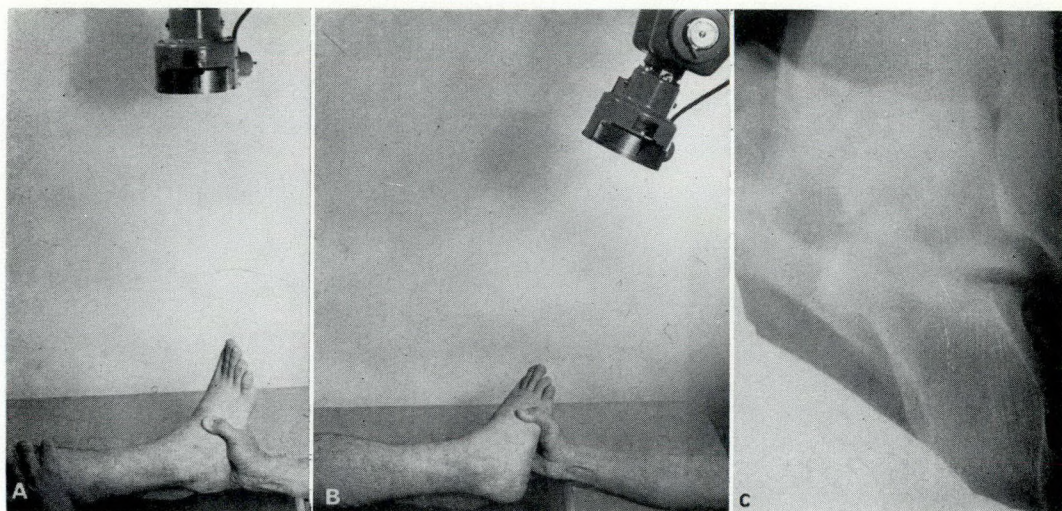
Fig. 10.—Radiographic technique for talar and subtalar tilt. (A) Anteroposterior stress film to demonstrate a talar tilt. (B) Special technique to show subtalar tilt: the x-ray tube is tilted at 45°, the leg is internally rotated 45° and a forced inversion stress is applied. (C) Normal subtalar motion using the special technique described in 10B.

be seen on the anterior-posterior film of the ankle. If the ankle is dorsiflexed beyond 90°, the posterior subtalar joint is then parallel with the ankle joint but it is still impossible to visualize this joint because of the superimposition of the foot over the ankle. The leg must be rotated internally 45° to visualize the outer posterior aspect of the subtalar joint (Fig. 9B). If a stress film is made in this position, the subtalar tilt may be visualized radiologically (Figs. 8C and 9C). Ideally, anteroposterior films should be taken with the leg in internal rotation and the foot in dorsiflexion. However, since it is difficult to dorsiflex a normal foot beyond 90°, let alone an injured foot, the x-ray tube can simply be tilted cephalad by 45°. A regular anteroposterior stress film is done to investigate the ankle joint (Fig. 10A), then the tube is tilted at 45° cephalad and a forced-inversion stress is applied while the foot is held at 90° and the leg internally rotated by 45° (Fig. 10B). Thus, a subtalar tilt, which has been experimentally reproduced in a cadaver (Fig. 8A) by sectioning the calcaneofibular ligament, can be demonstrated radiologically using this technique (Fig. 8C). On the other hand, when this same cadaver specimen was radiographed using standard anteroposterior stress films, the subtalar tilt could not be visualized (Fig. 8B).

Using the above-mentioned technique to visualize the subtalar joint, the normal mobility of this joint was studied radiologically to determine its physiological limits during the forced inversion of a normal ankle. Since inversion of the foot is due mostly to a gliding and sliding movement between the talus and calcaneus, there does not appear to be any physiological subtalar tilt during forced inversion. This was confirmed in the cadaver where, even in extreme inversion, the subtalar joint lines are still parallel to one another (Fig. 10C). For these reasons, we believe that any obvious subtalar tilt demonstrated radiologically using this technique is pathological.

Repeatedly during this investigation, tomograms were used to visualize the subtalar joint. However, even after a subtalar tilt had been produced in the cadaver, tomograms failed to demonstrate this lesion. For this reason, tomography is not recommended in the investigation of a subtalar tilt.

The technique described in this paper for the visualization of the subtalar joint may be most useful in fractures of the os calcis to visualize any subtalar extension of the fracture line. Since there is no need, nor any indication, to invert the painful foot when a fracture of the os calcis is suspected, the foot is radiographed in the lateral position
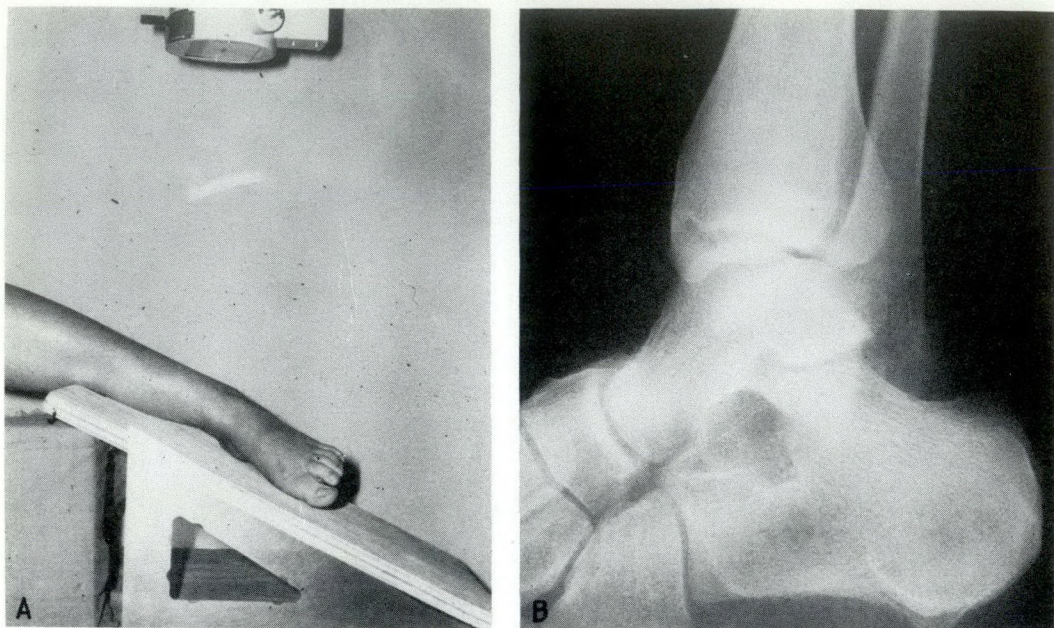
Fig. 11.—Special technique to visualize the subtalar joint without forced inversion—for use in patients with fractures of the os calcis. (A) Slanted plane to compensate for the absence of inversion and a lateral radiograph to visualize the posterior subtalar joint. (B) Radiographic visualization of the subtalar joint using the above technique (11A).

on an inclined plane (Fig. 11A); the posterior subtalar joint line is then well visualized (Fig. 11B).

The importance of making stress films in all ankle injuries is well established. In clinical practice, however, such films are frequently not made because local anesthesia is inadequate and the surgeon is reluctant to recommend general anesthesia. Local intravenous anesthesia is excellent in this situation. The stress films should include a regular anteroposterior view and an anteroposterior internal rotation view to visualize the ankle and subtalar joint (Figs. 10A and B); occasionally an arthrogram is done to complete the investigation.

SUMMARY AND CONCLUSIONS

The physiological limits of talar stability were investigated. The average talar tilt is 7°. It is present during inversion only and is not necessarily the same in both ankles of any one individual.

Physiological and pathological talar tilts can be distinguished radiologically because the normal talar tilt is associated with external rotation of the tibia and fibula, but a post-traumatic tilt is not.

Post-traumatic tilt can occur at the talar as well as the subtalar levels.

In cadaver dissections, it was demonstrated that the calcaneofibular ligament stabilizes primarily the subtalar joint, while the talofibular ligament stabilizes the talus within the ankle mortise.

Finally, a special radiographic technique is described to visualize the subtalar joint during forced inversion.

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Résumé

Cette étude porte sur les limites physiologiques
de la stabilité astragalienne. La bascule moyenne
est de 7°; elle n'est présente que durant l'inversion
et n'est pas nécessairement identique dans les deux
chevilles d'un même individu.

Les formes physiologiques et pathologiques de
la bascule de l'astragale peuvent être distinguées
par la radiologie: en effet la bascule normale
accompagne une rotation externe du tibia et du
péroné ce qui n'est pas le cas de la bascule
post-traumatique.

Cette bascule post-traumatique peut survenir
au niveau de la cheville aussi bien qu'au niveau
sous-astragalien.

On a montré, dans des dissections nécropsiques,
que le ligament calcanéo-péronier stabilise princi-
palement l'articulation sous-astragalienne, tandis
que le ligament astragalo-péronier stabilise l'astra-
gale au sein de la mortaise articulaire.

Enfin, l'article décrit une technique radiographi-
que spéciale permettant de visualiser l'articulation
sous-astragalienne durant une inversion forcée.

CHRONIC SURGICAL INFECTIONS

Basic principles and new ideas related to
the complex problem of chronic surgical infec-
tion are discussed in this paper. Adequate
drainage remains the first, and often the only,
necessary step. The conditions requiring addi-
tional antibacterial chemotherapy are general-
ized sepsis; danger of complications by spread
to certain anatomic locations, for example, the
face and the brain; danger of complications
because of coexistent systemic disease, such
as leukemia and diabetes, threat of recurrence
posed by incomplete drainage or removal of
the infectious focus, for example, burns, com-
pound fractures, and abscess of the gastro-
intestinal or genitourinary tract; and particular
susceptibility of grafts or implanted foreign
materials to circulating microbes.

To be effective, antibacterial therapy must
be sufficiently high, long and frequent, and
applied at the right moment to carry an ade-
quate concentration of the chemotherapeutic
agent to the site of infection. Factors to be
considered are differences in diffusion of a
given agent into different tissues, differences
in diffusion of various chemotherapeutic agents,
offending micro-organisms, route of administra-
tion and toxicity. The so-called problem micro-
organisms are staphylococci, *Escherichia coli,*
Klebsiella, Proteus, and Pseudomonas. Exact
bacteriologic diagnosis is often difficult or even
impossible, especially in chronic surgical infec-
tions. The choice of a chemotherapeutic agent
must be based on the clinical picture and
educated guess. The selected agent must not
be changed for at least four to eight days, and
then only for sufficient reason.

The treatment should be organized in
periods of curative therapy alternating with
periods of interval therapy. In either of these
a single agent or a combination may be used

provided that bactericidal drugs are not com-
bined with bacteriostatic drugs, so as to avoid
antagonistic effects. Penicillin, streptomycin,
kanamycin, polymyxin, and vancomycin are
bactericidal. Sulfonamides, the erythromycin
group, tetracyclines, and chloramphenicol are
bacteriostatic.

Penicillin and its semisynthetic derivatives
remain the first choice for treatment of most
chronic infections because the virtual absence
of toxicity permits high dosage and high tissue
concentrations. In patients known to be sensi-
tive to penicillin, cephalothin may be sub-
stituted.

Periods of curative therapy should last no
less than three weeks and should be separated
by periods of interval therapy lasting three
months and the use of tetracyclines in low
dosages. In patients with impaired renal func-
tion, the dosage should be lower to prevent
accumulation and toxicity. The leukocyte count
and urine should be examined periodically. In
situations involving the central nervous system,
electroencephalograms should be obtained peri-
odically to prevent epileptiform attacks caused
by too high a concentration of penicillin. In
certain situations, the amount of sodium or
potassium contained in high doses of penicil-
lin, e.g. 1 g. of sodium chloride in 10 million
U. of penicillin, may be clinically significant.
The appearance of eosinophilia is followed in
three weeks by a scarlatiniform rash. Strepto-
mycin, kanamycin, and colistin may cause
eighth nerve and renal damage. When using
chloramphenicol, the reticulocyte count should
be checked every four days.

No complications occurred when tetracyclines
were used for over one year. In less severe in-
stances, smaller doses given orally may suffice.
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1967.

LUMBAR DISCOGRAPHY AND ITS CLINICAL APPLICATIONS*

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DISCOGRAPHY is a method of defining the condition of an intervertebral disc by the injection of a radio-opaque substance. The technique and its clinical applications were initially described by Lindblom[1] in 1948. Much interest was stimulated in many centres but in the many papers that followed[2-12] there was little agreement as to the indications for the procedure, its value or indeed its safety. For this reason, we describe our experience with the method at the Toronto General Hospital where discography has been employed in the preoperative investigation of patients with pain thought to be due to intervertebral disc disease. From January 1958 to September 1966, this technique was used to examine 1092 patients at 2517 disc levels.

We believe that this examination should be done in the following instances: firstly, in patients with low back pain who have no evidence of root irritation and have no localizing signs on clinical examination or on conventional radiological examination; secondly, in those with bizarre radiation of back pain, for example, pain radiating to the groin; and thirdly, in those with obvious evidence of disc herniation in whom it is proposed to combine discotomy with spinal fusion. Most authorities agree that vertebral segments should not be fused if the disc above or below the level of prolapse shows any evidence of degeneration. Discography was done in this last group to determine the integrity of the adjacent discs. Finally, this examination was done in patients who developed recurrent or continuous back pain after laminectomy and discotomy, to determine whether the pain originated from that level or from an adjacent level.

TECHNIQUE

The examination is done in the Department of Radiology. The patient, adequately premedicated, is placed in the prone position with two pillows under the hips. Although some workers[13-15] favour the posterolateral approach, we use this only when a high lumbar level is being investigated. At other levels a midline transdural approach is routinely employed. Following infiltration of the skin with local anesthetic, a 22-gauge spinal needle is pushed in to the approximate depth of the disc space. A definite change in resistance will be apparent when the needle enters the intervertebral disc. After a needle has been placed in each of the desired levels, anteroposterior (AP) and lateral films of the lumbar spine are made to ensure that the needles are accurately centred at the junction of the middle and posterior thirds of the intervertebral disc space. Adjustments are made and checked as necessary.

The patient is warned that the procedure may be painful but he is not told when the injection will be made. Using a 2-c.c. syringe, 0.5 to 2 c.c. of 50% sodium diatrizoate (Hypaque) is injected. The operator records his impression of the resistance to injection, the amount injected and the distribution of the subsequent pain. It is the distribution of the pain that is important rather than its severity, even though the latter may be quite distressing. The pain associated with injection subsides within a few minutes.

After the injection of the contrast medium, with the patient still prone and before the needles are removed, a lateral film of the lumbar spine is made using a cross-table horizontal beam. The needles are removed. Then, with the patient in the lateral decubitus position, a lateral film is again made using an overhead tube. With the patient supine and the knees drawn
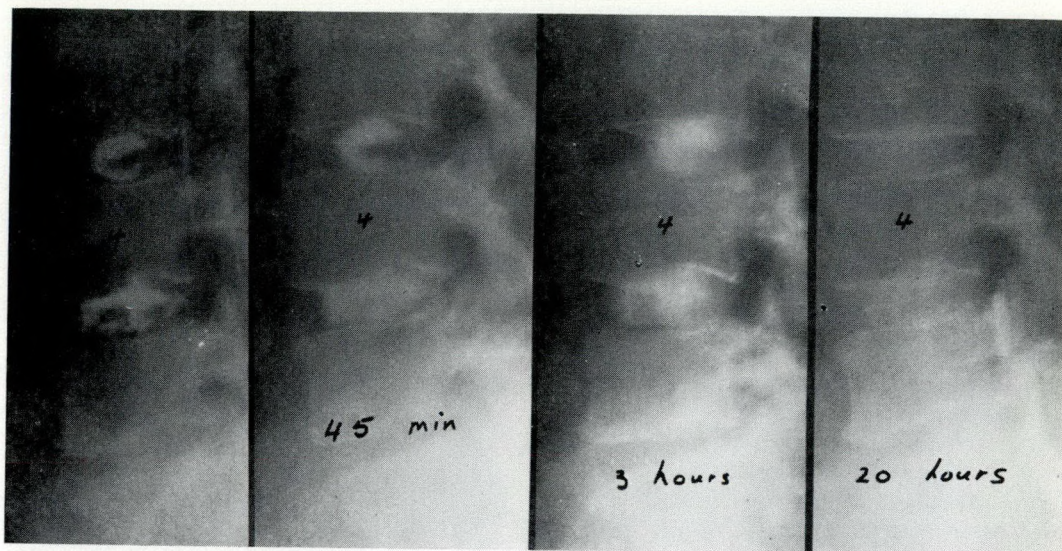
Fig. 1.—Resorption of the contrast medium. A normal disc pattern is seen at L3/4, a degenerated disc pattern at L4/5. There is progressive fading of the contrast material within the disc.

up, an AP view is then made followed by a second AP view with the tube tilted 20 to 25° cranially and centred on L4/5. The additional lateral view is taken to detect leakage of contrast medium along the needle tract; we have not encountered such leakage but other authors have reported it. The patient is returned to the ward and, within the limits of his pre-existing complaints, is allowed full activity. Unlike myelography, the patient rarely has headache after this examination.

RESULTS

The interpretation of a discogram depends on four basic factors: the resistance to injection, the amount injected, the pain reaction and the radiographic appearance. Each of these will be discussed in detail below. It is not possible to state accurately how many of the 2517 discs examined were abnormal. "Abnormality" must be assessed in relation to the four factors mentioned above, and to the adequacy of injection and the clinical problem under investigation. We are primarily interested in the interpretation of the results and their clinical application.

Significance of the Resistance to Injection

Injection into normal discs encounters firm resistance. Contrast medium injected into the annulus or into the cartilaginous end plate (as has occurred in a few patients) also meets firm resistance. Injections into degenerated discs meet diminished resistance; this is also noted in epidural leakage and inadvertent injections into the vertebral body. Although the normal disc gives firm resistance, such resistance on injection does not invariably mean that the underlying disc is normal because, in approximately 15% of our patients who gave firm resistance, the radiographic patterns suggested degenerated discs.

Significance of the Amount Injected

Approximately 0.5 c.c. of contrast medium can be injected into the normal lumbar disc. Larger quantities can be injected into the degenerated disc. Excessive quantities can be injected when there is epidural leakage and, in these patients, the pain pattern may be atypical—usually an intense midline burning pain. We do not inject more than 2 c.c. of contrast medium in any one disc injection. With the water-soluble material, good radiographic detail can be obtained for the first 10 to 15 minutes after injection. Blurring of the outlines begins after 15 minutes and by 24 hours there is a vague haze throughout the nucleus and the annulus (Fig. 1).

Interpretation of Pain Reactions

When the injection into a disc reproduces the typical pain pattern that the patient has been experiencing before injection, this disc is assumed to be producing the patient's symptoms. The exact mechanism of pain production during discography is unknown. We believe that both discogenic and neurogenic pain can be reproduced. During operation under local anesthetic, back pain has been produced by electrical needle stimulation of the annulus and the nucleus, and by palpation of the dorsal surface of the annulus.[16-19] The injection of saline or contrast material into the disc increases intradiscal pressure. Normally the annulus confines the effects of this increased pressure within the nucleus and no pain is produced. In an abnormal disc, the pressure is not confined and may be transmitted through an already weakened or torn annulus producing pain by stretching the fibres of the annulus. At operation, it has been demonstrated that injection and distension of a degenerative disc may produce gross hyperflexion of the posterior joints. This excessive movement may also be a source of pain. Finally, diffuse bulging of the annulus on distension may irritate nerve roots and produce root pain. According to Hirsch and Schajowicz,[17] a pathological disc has a larger number of nerve fibres than a normal disc because vascular granulation tissue has invaded the disc. Stimulation of these fibres (? pain fibres) by the increase in intradiscal pressure may explain the discogenic pain.[20]

The clinical significance of reproducing the patient's typical pain by the injection of a disc is difficult to establish. According to Lindblom,[1] if the pain produced during discography is similar to the patient's previous pain, the disc is herniated and the nerve root is compressed. This has not been our experience. In many patients undergoing exploration we have been unable to demonstrate herniation or root compression by discs that were abnormal on discography. Similar findings have been reported by Fernström.[21]

A disc that appears normal on plain films may still be degenerated and the source of symptoms.[22, 23] Also, a disc in which degenerative changes are demonstrated radiologically may not produce symptoms. In our series, 30% of patients with evidence of disc degeneration on plain films had no pain when contrast medium was injected into the disc. Of the remainder, only one-half had typical (previously experienced) pain, and the other half had atypical pain. Atypical pain was described in the back and legs, and in bizarre sites such as the groin, testicle, lower abdomen, epigastrium and even the head.

Because discography is painful, it has been suggested that the competence of the disc be assessed at operation by observing the resistance offered to the injection of saline into the nucleus.[24, 25] However, our findings and those of others suggest that even the apparently normal disc many not furnish the resistance expected and may also accept more fluid than usual. Secondly, a not-uncommon technical error is injection into the annulus. This error is recognized only by examining the discographic pattern. Therefore, saline injections into the annulus may simulate a normal disc even in the presence of degeneration. Even when successful, the saline test does not differentiate between the symptomatic and asymptomatic degenerative disc.

Radiographic Interpretation

The normal intervertebral disc consists of the nucleus pulposus within the concentric fibres of the annulus fibrosus which are attached to the adjacent vertebral bodies. When outlined by contrast medium, the nucleus pulposus is seen as a well-circumscribed shadow which may show several patterns—bilocular, ovoid, horseshoe, collar-button, square or rectangular. Regardless of the pattern, the contrast medium is confined to the anatomical position of the nucleus pulposus (Fig. 2).[4, 26]

As the disc degenerates, the radiographic pattern varies depending on the severity and type of annular involvement. With simple degeneration without an annular tear, the normal discographic pattern will be lost with shredding, irregularity and spreading of the contrast medium. With annular tears, the contrast medium spreads beyond the confines of the annulus, and extends under the ligaments, through a torn posterior longitudinal ligament into the
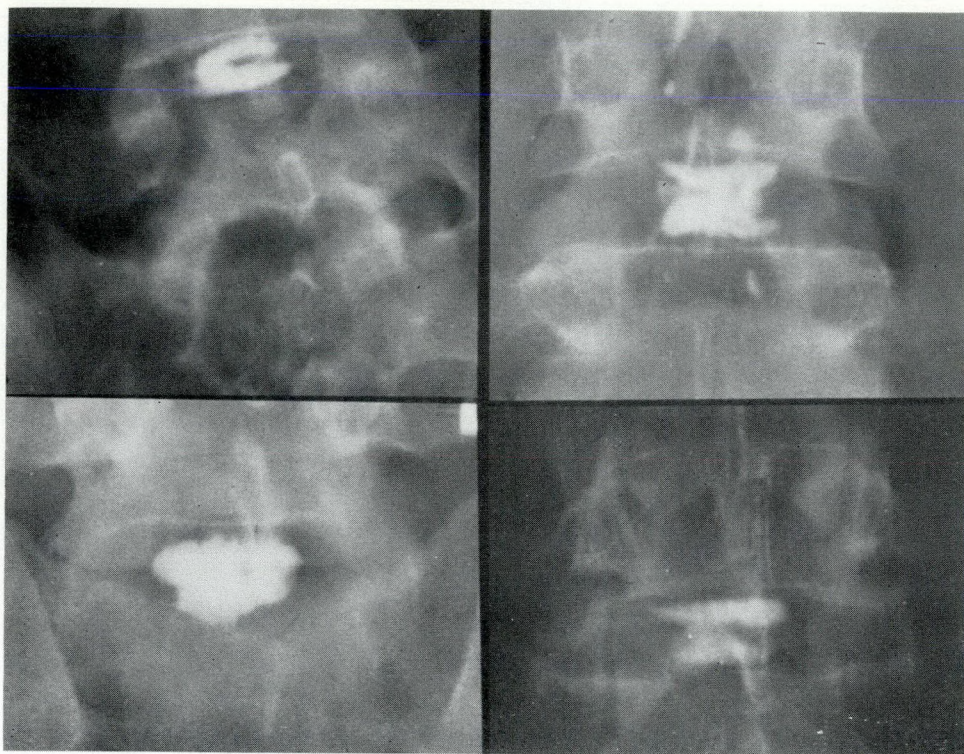
Fig. 2a   Fig. 2b

Fig. 2.—Composite AP (a) and lateral (b) discograms showing normal variations of pattern including bilocular, ovoid, collar-button and rectangular.

epidural space, or along any adjacent tissue plane (Fig. 3). A "sequestrum" of nucleus can occasionally be seen under a ligament or in the epidural space as a radiolucent shadow with an encircling ring of contrast medium (Fig. 4).
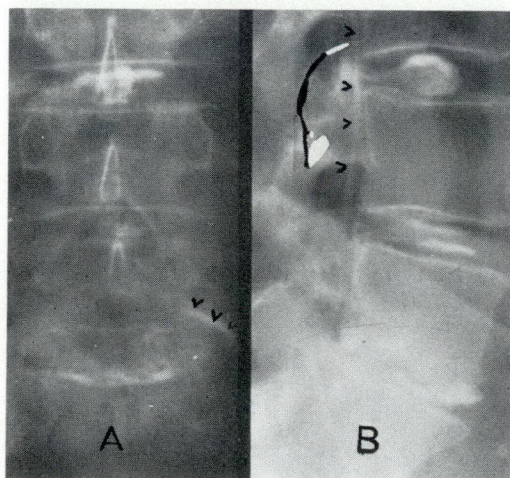

Fig. 3.—(A) Perineural leak to the left at L5/S1. (B) Epidural leak posteriorly at L3/4.
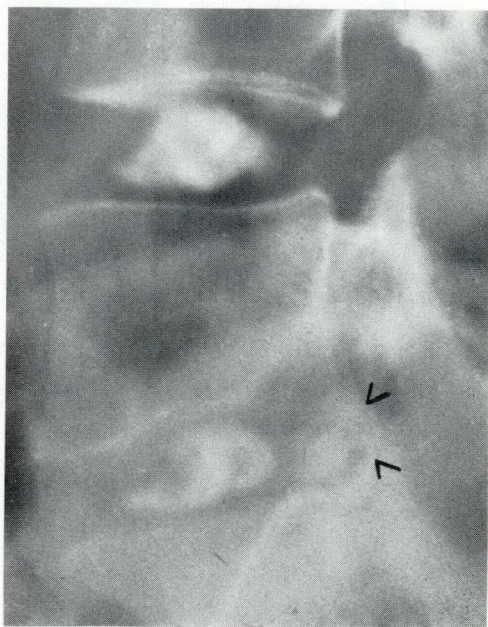

Fig. 4.—A collection of contrast medium outlines a posterior disc sequestration at L5/S1. This was later proved at operation. Myelography failed to demonstrate this sequestration because the subarachnoid space was narrow at this level.
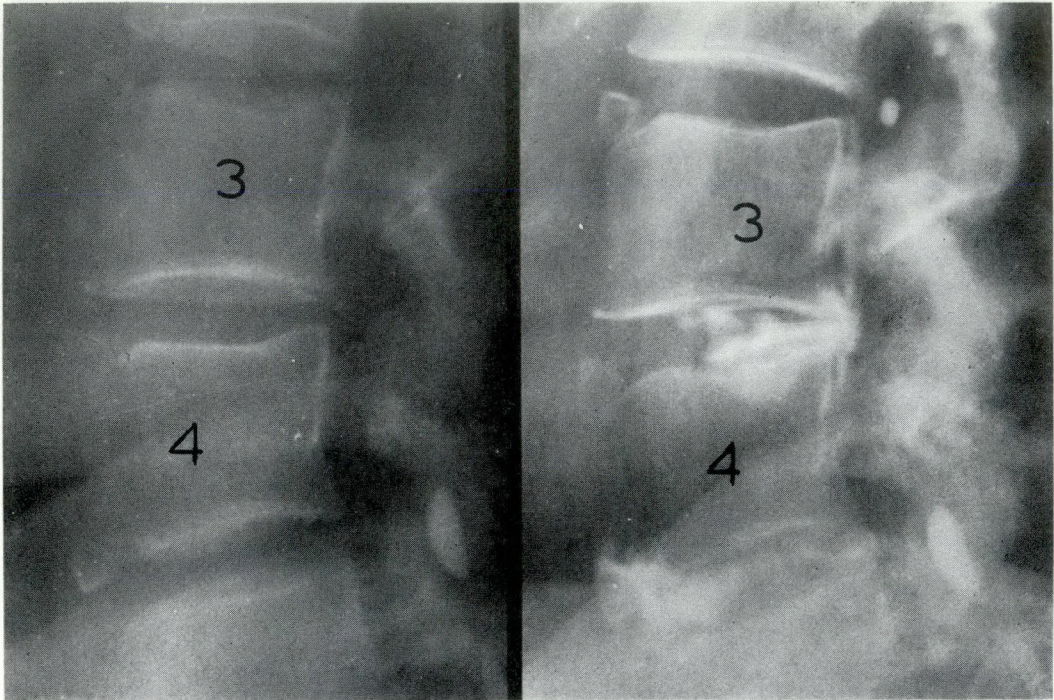
Fig. 5.—Multiple transosseous disc ruptures. The contrast medium crosses the defect at the anteroinferior border of the L4 vertebral body. The defect at the anterosuperior border of the L4 vertebral body was not outlined, probably because the medium leaked epidurally tracking up behind the body of L3.

Fig. 6a   Fig. 6b

Fig. 6.—(a) Anteroposterior and (b) lateral views showing contrast medium entering a Schmorl's node in the inferior surface of L3.

Fig. 7a   Fig. 7b

Fig. 7.—Annular injection at L5/S1. If only the lateral view (b) was seen, the error in technique would not be recognized. The eccen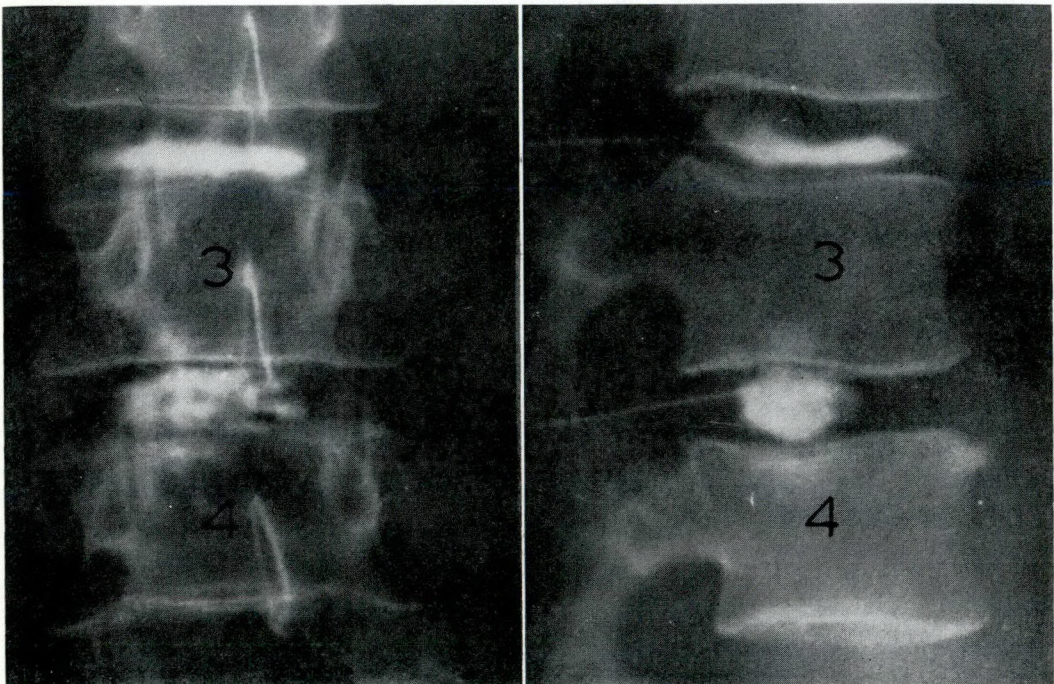tric position of the contrast medium on the AP view (a) indicates an annular injection. When the injection was repeated at the L5/S1 level, the disc was normal.

Other types of disc degeneration can also be demonstrated: (a) A transosseous disc rupture. This occurs when the disc tissue is driven out through the vertebral body rather than through the annulus. This rupture produces a separate fragment (Fig. 5). This phenomenon was first described by Schmorl and confirmed by Lindblom.[27] It has been erroneously termed a "limbus vertebra", a chip fracture, a persistent secondary epiphysis or an intercalary body. (b) A central herniation of disc material, termed a Schmorl's node (Fig. 6).

Unusual patterns produced by technical errors must be recognized: (a) Injection into the annulus (Fig. 7).[28] This can be avoided by checking the needle position radiographically before injection. (b) Intraosseous injection of the contrast medium (Fig. 8).

CLINICAL APPLICATIONS

When the diagnosis of disc herniation is made clinically, a myelogram rather than a discogram is advised if radiological confirmation is desired. Although myelography

is highly accurate in disc herniation,[29] if the prolapse is unusually far lateral, if the subarachnoid sac is short or narrow, or if the sac is not applied closely to the back of the L5/S1 disc space, the herniation may not be visualized. In these cases, discography is a valuable secondary procedure.

The chief value of discography is in the investigation of the patient with symptoms arising from disc degeneration without herniation.[30] The radiographic pattern identifies the level or levels involved, and the character of the pain produced on injection differentiates symptomatic from asymptomatic disc degeneration.

Myelography in patients with back pain due to disc degeneration rather than herniation is valueless. Plain radiographs often appear normal at the involved level. Discography before spinal fusion ensures that the symptomatic level of disc degeneration is included in the fusion.

When the fusion is planned and discography indicates that a single level is producing symptoms, only that level is fused (Fig. 9). If discography produces
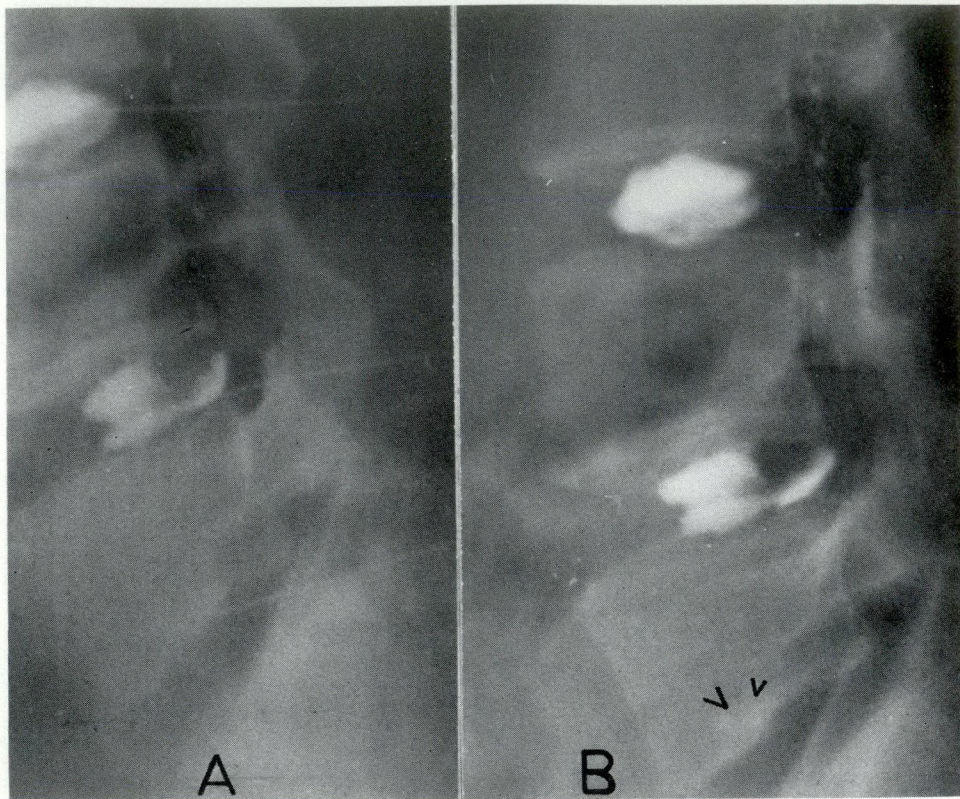
Fig. 8.—(A) Intraosseous injection. Lateral view with needles in place. Normal disc at L3/4, degenerated disc at L4/5. No contrast medium was seen in the disc space at L5/S1 even though 1 c.c. of contrast medium was injected against diminished resistance. The tip of the needle is in the cartilaginous end plate or in bone. (B) Lateral view after removal of the needles. The contrast medium is in the lower border of the L5 vertebral body.
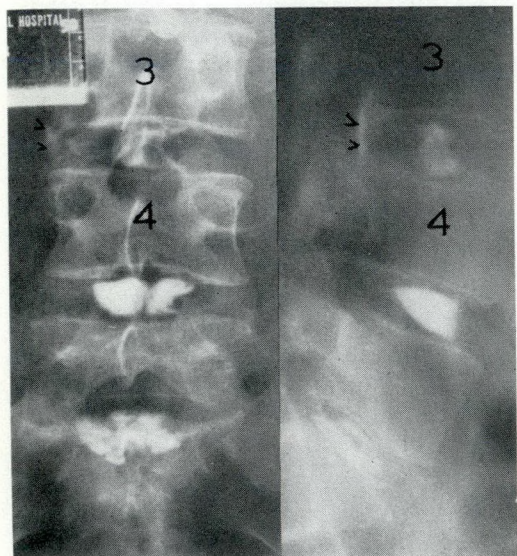

Fig. 9.—This patient presented with back and right leg pain without root tension. On discography, the contrast medium extravasated to the right and posteriorly at L3/4 and reproduced his

pain in an adjacent level or if degeneration is demonstrated without pain, this level is also included in the fusion (a "two level" fusion). In the small group of our patients who had multiple degenerated but asymptomatic discs, we fused only the symptomatic level. This avoided a long multisegment fusion, an operation which has not given encouraging results (Fig. 10). However, a long multisegment fusion cannot be avoided if the L3/4 disc is producing symptoms and the discs at the lower two levels are also degenerated. Fortunately, the combination of circumstances is rare but full information must be obtained if the fusion is to be planned intelligently. When discography produces pain at more than three levels, spinal fusion is usually

previous pain. Minor degenerative changes were seen at L5/S1; an unusual normal pattern at L4/5. Single segment fusion was done at L3/4 with complete relief of symptoms. The patient is symptom free 1½ years later.
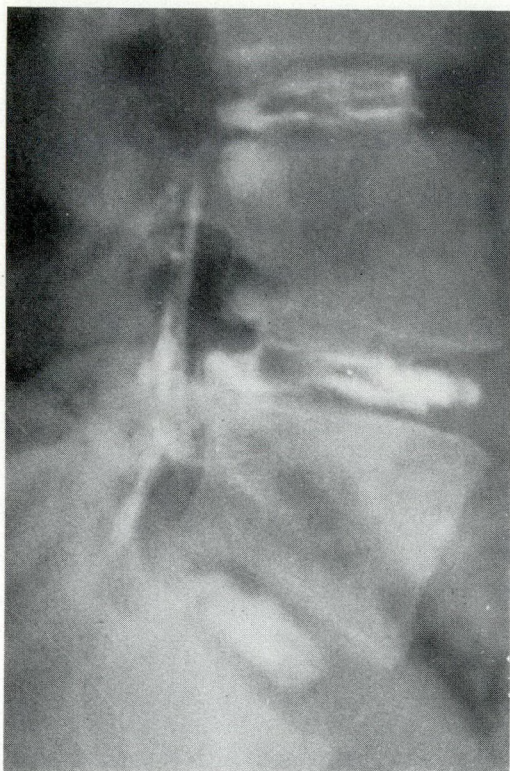
contraindicated; indeed, the demonstration of degeneration at three levels should make the surgeon hesitate because "three level" fusions have had limited success.

Discography is also valuable in the investigation of recurrent back pain following previous spinal operations, particularly spinal fusion. It can demonstrate that the vulnerable "level-above-the-fusion" is not necessarily the cause of the recurrent symptoms in these patients (Fig. 11).

COMPLICATIONS OF DISCOGRAPHY

Minor complications have been reported such as headaches, febrile reactions, meningismus, accentuation of symptoms and allergic reactions.[16] Major complications are relatively rare. Meningitis is unusual and is no more common than with myelography. A review of reports of complications following discography[31-36] shows that of over 5000

Fig. 10.—Discography showed degenerative changes at all three lower lumbar levels. A myelogram done previously was negative. Injection at L4/5 reproduced the patient's previous pain and this single level was fused. The patient has had no symptoms for two years.
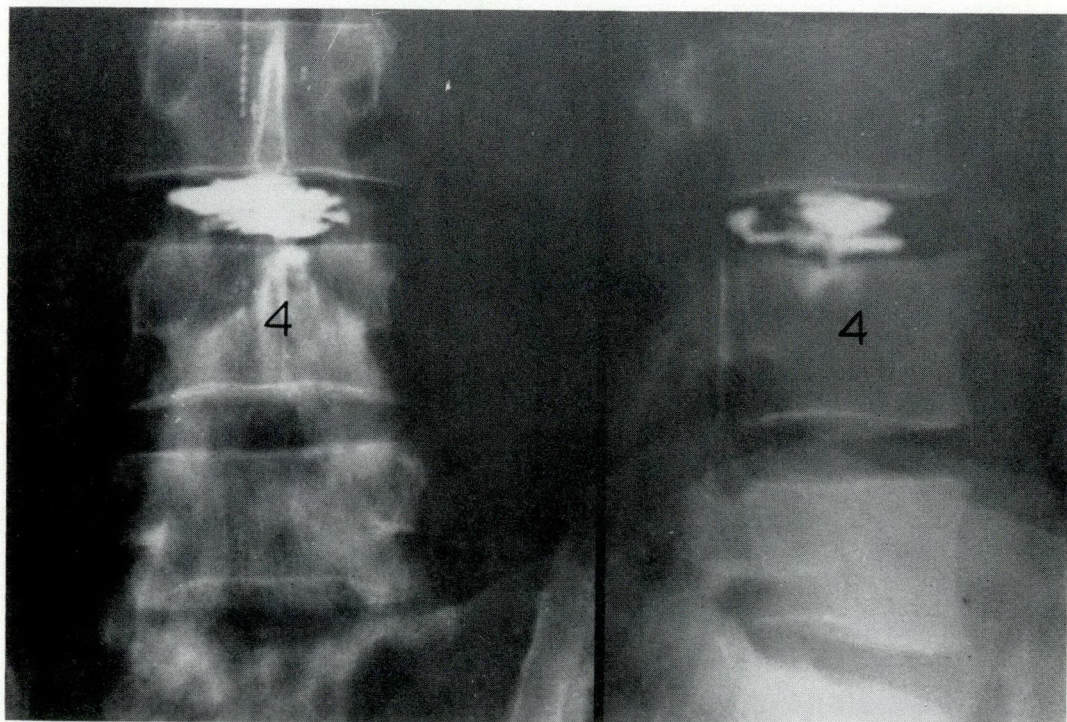

Fig. 11.—Intact L4-to-sacral fusion. An L3/4 discogram shows a Schmorl's node and extravasation of contrast medium posteriorly. Discography produced no pain. In view of these findings, the fusion was not extended.

discs injected, approximately 30 discs became infected; 19 of these, however, were from one group of 88 patients reported by a single author. Disc space infection has also been reported after diagnostic lumbar puncture. Gardner et al.[5] found that 37 of 58 patients with disc space infection had meningitis at the time of lumbar puncture.

Some authors have expressed concern that discography may damage a normal disc or further damage a degenerate disc. In a one-year follow-up of his cases, Ribbing[36] reported that 3 of 11 normal discs and 18 of 49 degenerated discs had lost 1 mm. in height. Collis and Gardner[37] reviewing some 4000 injected discs concluded that discography did not produce any mechanical or clinical damage to the disc. Cloward and Buzaid,[4] discussing a 10-year experience with discography, reported that they do not know of a single disc that has ruptured and become symptomatic because of discography.

When we examined disc material removed at operation, we found no change that could be attributed to injection. These findings agree with those of Gardner et al.[5] but others have described changes. Goldie[38, 39] found abnormal droplets in disc material that were "refractile to light but anisotropic". He thought these were due to the contrast medium (35% Dijodan) but, on analysis, found the droplets were composed of chondroitin sulfuric acid. He found no evidence of necrosis or tissue degeneration.

In over 1500 disc injections, Fernström[21] described six herniated discs produced by discography but did not specify whether normal or degenerate discs had herniated. After puncturing the annulus of fresh cadavers with a 4.7 mm. trochar, Friberg[40] could not produce extrusion of disc material by spinal motion. At operation, Hirsch[41] demonstrated that an intervertebral disc when punctured did not release disc material.

In our series of 1092 patients examined at 2517 disc levels, only one developed disc infection. As far as we know, none developed disc herniation or collapse following discography. Approximately 10% of patients had some headache after this procedure. Two patients had meningismus.

Conclusions

The major indications for discography are to establish the level of the painful disc, to distinguish the symptomatic from the asymptomatic degenerative disc, and to establish the extent of the spinal fusion necessary to relieve pain.

When there is nerve root tension and the clinical diagnosis of herniated disc has been made, myelography is the procedure of choice to confirm that diagnosis. It is emphasized that discography is not an alternative to myelography but is an ancillary investigation which furnishes information that cannot be obtained in any other way. While, in many of our patients, myelography was done before discography, in many of those with low back pain without sciatica, only discography was done.

The overall morbidity is low and should not deter any physician from asking to have the procedure done. However, the pain elicited, although essential to the interpretation of the test, may be quite severe. The physician should always bear this in mind in selecting patients for this procedure.

Discography promises to enlarge our knowledge of low back pain that originates within the intervertebral disc.

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RÉSUMÉ

Il appartient surtout à la discographie de déterminer à quel niveau est situé le disque douloureux, de distinguer la forme symptomatique de la forme de dégénérescence discale asymptomatique et de préciser l'amplitude de l'arthrodèse spinale nécessaire pour soulager la douleur.

Dans le cas où existe une tension radiculaire et une fois obtenu le diagnostic de hernie discale, c'est avant tout à la myélographie qu'il faut s'adresser pour confirmer ce diagnostic. Il est bon de souligner que la discographie ne remplace pas la myélographie, mais n'est qu'un auxiliaire précieux permettant de fournir des renseignements qui ne pourraient être obtenus par un autre moyen. Même si, chez nombre de nos malades, nous avons procédé à la myélographie avant la discographie, chez de nombreux malades souffrant de lombalgie sans sciatalgie, nous n'avons pratiqué que la discographie.

La morbidité globale est faible et ne devrait décourager aucun médecin de demander cet examen radiologique. La douleur qu'il entraîne, même si elle est indispensable à son interprétation, peut être très vive. Aussi, le médecin devra-t-il toujours s'en souvenir pour choisir les malades candidats à cet examen.

La discographie pourra élargir le champ de nos connaissances sur la lombalgie qui a son origine dans le disque intervétébral.

NEOPLASMS OF THE ATLAS AND AXIS

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and V. LAWSON, M.D., *Toronto, Ont.*

NEOPLASMS of the atlas and axis are rare and even metastases in this location are uncommon. In 619 biopsies of the spine for the diagnosis of a multitude of lesions, Ottolenghi, Schajowicz and De Schant[1] found only four tumours of C1 or C2. The present communication describes our experience with 12 patients with this condition. In it, three main points will be emphasized: that diagnosis is often difficult; that laminectomy may be hazardous; and that, in most patients, operative stabilization of the involved area should be considered.

Most patients with neoplasms of the atlas and axis present with acute neck pain of sudden onset, usually after a minor traumatic episode. Loss of rotation is the most significant physical finding. Because these patients are usually in the older age group, radiographs often show degenerative changes in the lower cervical spine. These may draw the examiner's attention away from the more important problem and tend to obscure the diagnosis. Furthermore, radiographs of the atlantoaxial joints are often difficult to interpret, especially if the films are of poor quality. As the lesion progresses, the vertebrae become unstable, eventually subluxate and compress the spinal cord. Extradural spread of tumour is less likely to produce cord compression. Laminectomy, because it tends to increase the instability, may aggravate or precipitate quadriplegia.

In many patients with metastatic tumour, the physician adopts an attitude of *laissez-faire*. A few of these patients, however, may have a life expectancy of two or more years; in these, surgical stabilization may prevent cord compression and relieve pain and disability to a significant degree. Our experience suggests that a more active approach to many of these patients is indicated.

The 12 cases described in this paper were collected from the Toronto General, Wellesley and Princess Margaret Hospitals, all teaching hospitals of the University of Toronto. Eight were examined and managed by the authors. The remaining four were under the care of orthopedic colleagues. Only 1 of the 12 had a benign tumour, 3 had multiple myeloma and the remainder had metastatic tumours (Table I).

TABLE I.—PRIMARY DIAGNOSIS AND ITS RELATION TO NECK PAIN

Primary diagnosis	Number of patients	Neck pain as initial symptom	Neck pain appearing after disease apparently controlled
Osteoid osteoma.	1	1	
Multiple myeloma......	3	2	
Carcinoma:			
Breast........	3	1	1
Bladder.......	1		1
Tongue.......	1		1
Thyroid......	1	1	
Lymphosarcoma.	1		
Anaplastic carcinoma (? lung).......	1		

These 12 patients all had neck pain. In five this was the presenting symptom (Table I). Three other patients developed neck pain long after the primary tumour was thought to have been eradicated (Table I). Two patients developed such pain six years after treatment of the primary lesion.

CASE REPORTS

Seven patients from this group are described to illustrate the major problems associated with neoplasms of the atlas and axis.

Case 1.—D.H., a 19-year-old man, had the only benign lesion in the series. He presented with a one-year history of painful torticollis. Radiographs showed a lesion of the spinous process of C2 (Fig. 1). Total excision of this spinous process relieved his symptoms. On histological diagnosis, the lesion proved to be an osteoid osteoma.

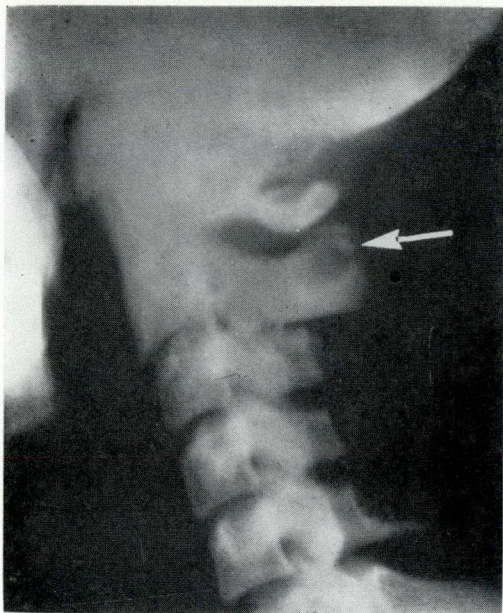*Department of Orthopedics, Wellesley Hospital, Toronto, Ont.

Fig. 1.—Case 1, D.H. Osteoid osteoma, spinous process of the axis.

Case 2.—R.L., a 61-year-old man, developed neck pain of sudden onset while drying his hair after a shower. Radiographs showed a pathological fracture of C2 (Fig. 2) which subsequently proved to be due to multiple myeloma. The vertebrae were fused from the
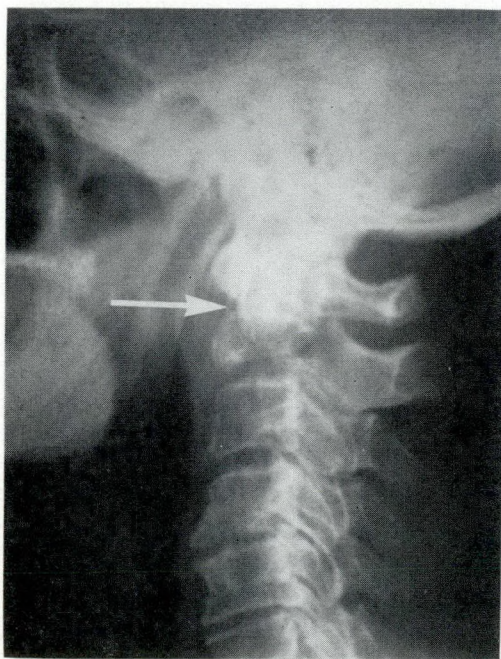

Fig. 2.—Case 2, R.L. Pathological fracture of the body of the axis.

occiput to C4 with a posterior cancellous bone graft. The patient returned to work.

Apart from one episode of neck pain he continued at work for the next four years. During this time his upper cervical spine was destroyed to an alarming degree until ultimately his neck was supported only by the posterior graft (Fig. 3). Eventually, after a
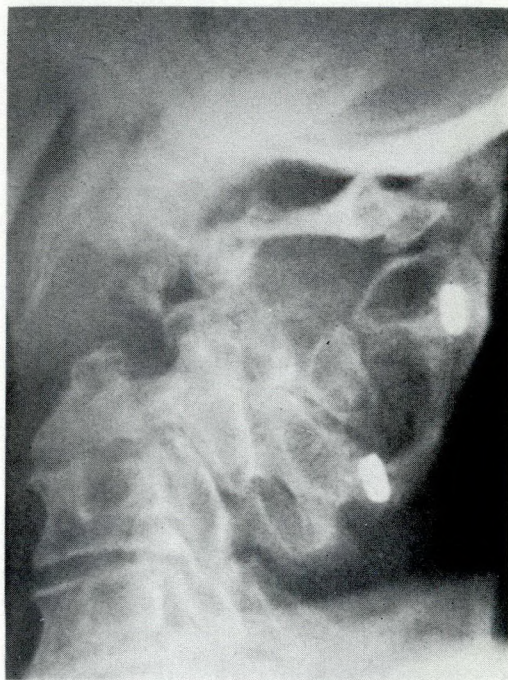

Fig. 3.—Case 2, R.L. Appearance two years after fusion. Note that the axis has almost disappeared.

pathological fracture through the femoral neck confined him to bed, he steadily deteriorated and now, five years after the onset of his disease, his condition is terminal.

Case 3.—W.B., a 48-year-old man, developed neck pain after a minor fall. Radiographs were interpreted as showing only degenerative changes (Fig. 4). After six months of unsuccessful conservative therapy, further investigation established the diagnosis of multiple myeloma. The key physical finding of grossly restricted rotation, mainly a function of the atlantoaxial joint, could not be explained by the degenerative changes in the lower cervical spine and, in retrospect, lytic changes in the left articular mass of the atlas were apparent on earlier films.

Eight months after the onset of neck pain, he developed lateral subluxation of C1 on C2

Fig. 5.—Case 3, W.B. Anteroposterior view showing lateral displacement of the axis.

Fig. 4.—Case 3, W.B. Film interpreted as showing degenerative changes. Arrow indicates osteolytic lesion in the atlas, recognized in retrospect.

(Fig. 5). He has now carried on for two years with disabling neck pain. His generalized disease appears to be under control but he has

refused the offer of operative stabilization, to date.

Case 4.—C.M., a 56-year-old woman, had known multiple myeloma of two years' duration. She presented with acute neck pain and radiographs showed a pathological fracture of the axis (Fig. 6). A film of her cervical spine,

Fig. 6.—Case 4, C.M. Pathological fracture of the body of the atlas.

Fig. 7.—Case 4, C.M. Anterior subluxation of the atlas on the axis.

done as part of a skeletal survey two months before this, had been normal.

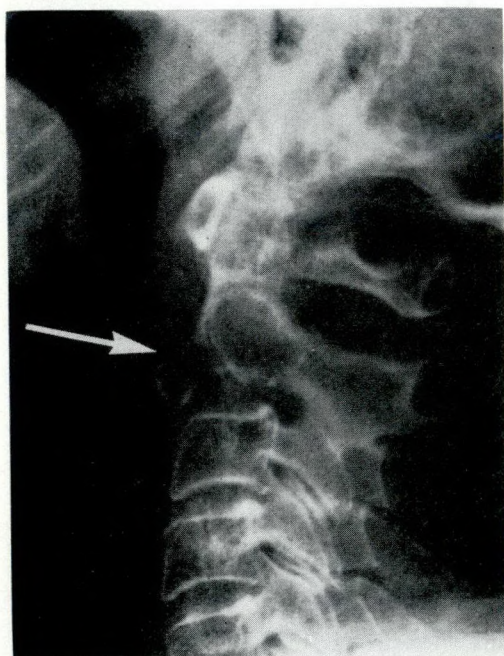Over the next four months, after progressive subluxation of C1 on C2, she became quadriplegic (Fig. 7). Skeletal traction reduced the displacement and she recovered. A posterior fusion from C1 to C3 was then done (Fig. 8). She is now active, free of neck pain and has no neurological deficit.
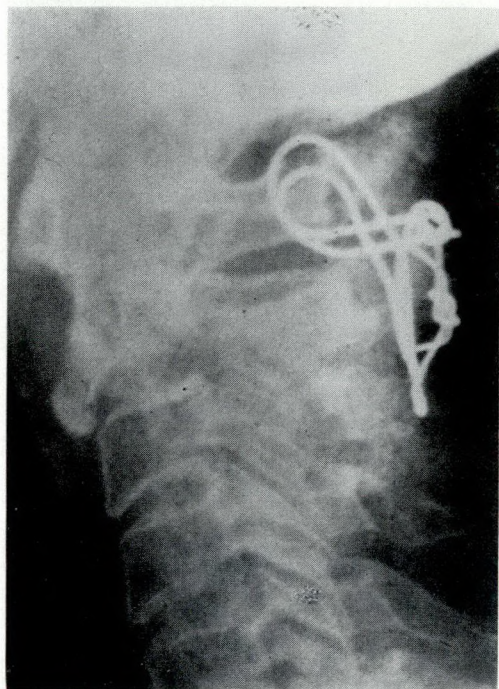

Fig. 8.—Case 4, C.M. Position after closed reduction and fixation. The wire passes around the lamina of the atlas.

Case 5.—J.H., a 65-year-old man, fell and fractured his odontoid process six years after receiving radiation therapy for a cancer of the bladder (Fig. 9). Over the next month he developed progressive subluxation with quadriparesis (Fig. 10) and we suspected that the fracture was through an area weakened by metastasis. A specimen obtained by transpharyngeal needle biopsy showed papillary transitional cell carcinoma.

On skeletal traction the subluxation was easily reduced and the neurological deficit was reversed. He was fused from C1 to C3 and initially did well. Four months later, on a follow-up radiograph, a second lytic lesion was demonstrated in C5. Shortly after this he developed recurrent neck pain, but remains ambulant and free of neurological symptoms.
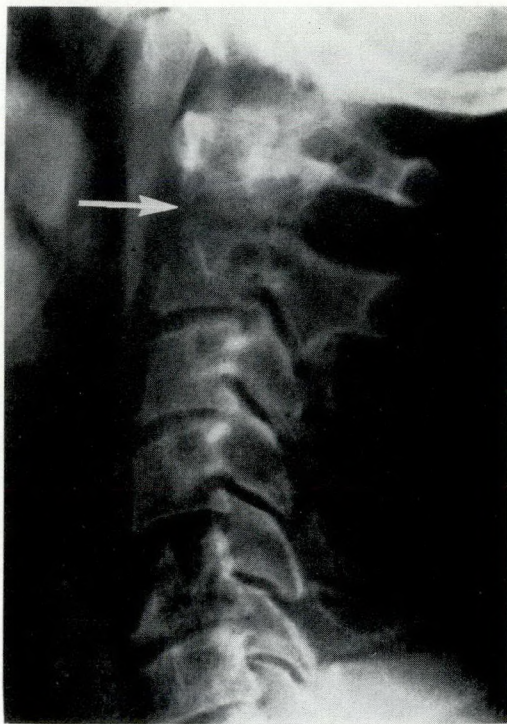

Fig. 9.—Case 5, J.H. Film interpreted as showing a fracture of the odontoid, possibly old.
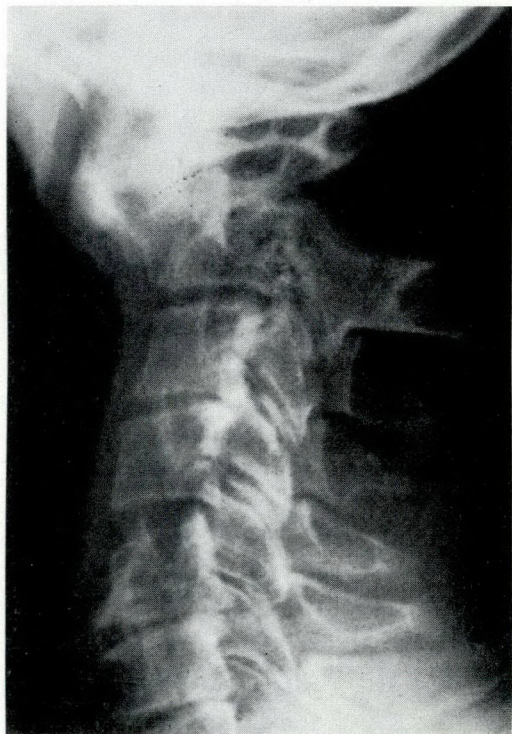

Fig. 10.—Case 5, J.H. Subsequent anterior subluxation of the atlas on the axis.

Fig. 11.—Case 6, M.W. Pathological fracture of the axis with minimal subluxation.
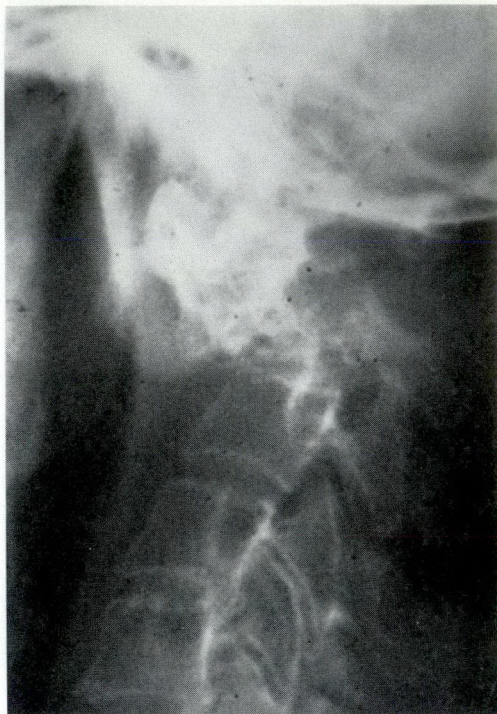

Fig. 12.—Case 6, M.W. Increased subluxation following laminectomy.

Case 6.—Over a two-year period, M.W., a 53-year-old single woman, was intensively investigated for neck pain but no cause was found. The striking clinical finding was loss of rotation in the cervical spine. Her personality suggested that she was subject to hysteria.

Two years after the onset of symptoms, she was found to have a pathological fracture of C2 (Fig. 11). A biopsy of the involved area by the posterior route showed that the lesion was a low-grade papillary carcinoma of thyroid origin. During the biopsy, the laminae of C1 to C3 were removed. A thyroidectomy was also done. After operation, she developed progressive subluxation with quadriplegia (Fig. 12).

With skeletal traction the subluxation was reduced and the neurological deficit receded. Because the posterior elements had been resected, an anterior fusion (C3 to the occiput) was carried out. She was stabilized with a "halo" and body plaster. In five months, fusion was solid (Fig. 13). Treatment with radioactive iodine kept the metastatic disease under clinical control. The patient has been free of pain and has remained at work for the past 18 months.

Case 7.—Mrs. A.M., a 52-year-old woman, consulted her physician because of neck pain. She was found to have carcinoma of the breast
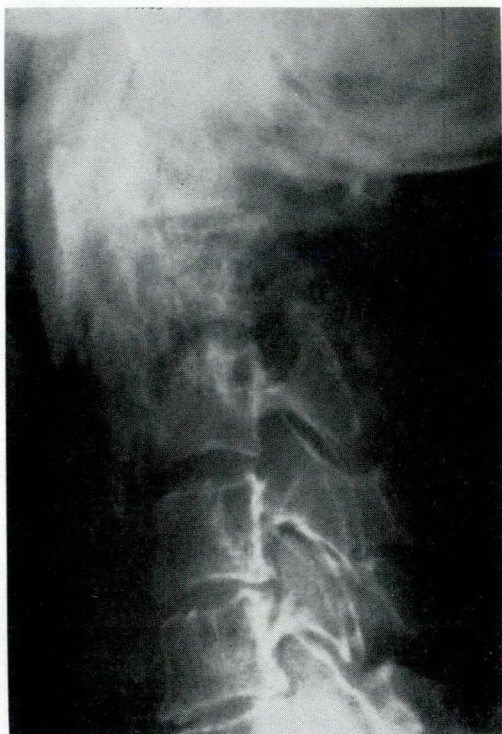

Fig. 13.—Case 6, M.W. Position after closed reduction and anterior fusion.
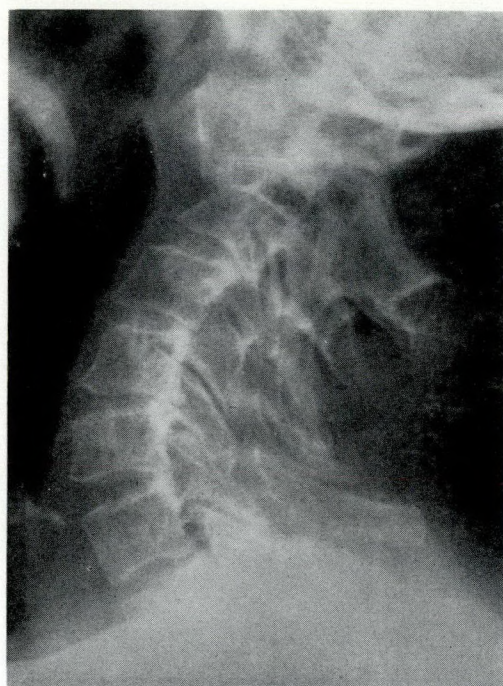
Fig. 14.—Case 7, A.M. Gross displacement due to a pathological fracture of the axis.

TABLE II.—SURVIVAL TIME AFTER ONSET OF NECK PAIN

Patient	Disease	Survival time	
		Alive	*Dead*
R.L.	Myeloma......	5 years*	
W.B.	Myeloma......	2 "	
C.M.	Myeloma......	1½ "	
	Carcinoma:		
H.T.	Breast......	1½ "	
D.J.	Breast......	2½ "	
A.M.	Breast......		7 years
J.H.	Bladder.....	1 "	
J.K.	Tongue	1½ "	
M.W.	Thyroid.....	3½ "	
C.M.	Lympho-sarcoma.....		2 months
L.S.	Anaplastic carcinoma...		1 month

*Terminal at the time of writing.

with a metastatic lesion in the axis. Radiation therapy gave dramatic relief of symptoms and she remained well for four years until the odontoid process fractured and C1 subluxated on C2 (Fig. 14). For the next three years, she was disabled with severe neck pain, but had no significant neurological symptoms. She suddenly died after a minor fall. No autopsy was done but we believe that acute spinal cord compression caused her death. Perhaps a fusion might have protected her from this complication.

DISCUSSION

Most of the 12 patients reported here sought medical attention because of acute neck pain that developed after relatively minor trauma. In five, this symptom was the first manifestation of their disease (Table I). The cause of such acute pain is usually a pathological fracture. Since the main movement of the atlantoaxial joint is rotation, restriction of this movement is an important physical finding. Radiographs of these vertebrae are difficult to interpret because most such patients are in an age group where degenerative changes in the lower cervical spine are common. This may

be misleading. Tomograms are particularly useful and occasionally cineradiographs may reveal abnormal movement at the atlantoaxial joint.

In most of these patients, the tumour was in the more anterior portions of C1 or C2, most commonly the articular mass of the atlas, and the body of the axis below the odontoid process; with pathological fracture, progressive subluxation was common and, when advanced, resulted in cord compression.

In these 12 patients the posterior vertebral structures were spared, relatively speaking. Cord compression was secondary to subluxation rather than the effects of extradural tumour. In each, symptoms of cord compression were relieved by reducing the subluxation with skeletal traction. Laminectomy, whether done for biopsy or decompression, often makes the situation worse because it removes the only stable portion of the upper cervical spine and subluxation may progress more rapidly. In one of these patients, laminectomy actually precipitated cord compression. Moreover, absence of the posterior elements makes stabilization of the cervical spine extremely difficult.

Because of the poor prognosis in metastatic disease, the surgeon often hesitates to advise operative stabilization. Survival time in these 12 patients (Table II) suggests that operative stabilization should be

considered in some of them. Patients with multiple myeloma, metastatic cancer of the thyroid and carcinoma of the breast in particular survived for relatively long periods.

Since some lesions can be relatively well controlled with radiation and chemotherapy, it is essential to establish a specific tissue diagnosis. The danger of subluxation following a posterior approach (laminectomy) makes this route inadvisable. Lesions of the atlas and axis are readily accessible to an anterior transpharyngeal approach. A biopsy needle introduced under radiological control with the image intensifier is a simple and relatively safe procedure and does not carry the risk of increasing the instability.

Of the 11 patients with metastatic disease, 5 were not considered candidates for operation. Two had widespread metastatic disease and died a few months after they were first examined. In three others, symptoms were controlled adequately by a brace. Of the remaining six, four were stabilized by open operation. In two, posterior fusion was done to stabilize a subluxation that had produced quadriplegia. In these patients the deformity was reduced by traction, and the reduction was maintained by wiring the laminae together at the time of fusion. Both recovered from their quadriplegia. One patient with myeloma and intractable pain had a posterior fusion. He worked for the next four years in relative comfort. The patient who developed progressive subluxation and quadriparesis following laminectomy was fused using the anterior approach. Reduction and fixation were maintained by the halo apparatus. She recovered from her neurological deficit. When considered in retrospect, two more patients should have been stabilized by posterior spinal fusion. One patient with myeloma, whose main disability was neck pain, carried on for $2\frac{1}{2}$ years. The other, who had metastatic cancer of the breast died suddenly after three years of disabling neck pain.

Posterior fusion of the upper cervical spine is not hazardous and is not accompanied by a high morbidity rate. The fusion must include the vertebra above and below the one involved by tumour.

In lesions of C2, interlaminar wiring of C1 to C3 produces sufficient stability to allow early mobilization of the patients. Lesions of C1 require fusion to the occiput. The use of a halo for postoperative immobilization in this less common site (3 of 12) allows relatively early ambulation. Anterior cervical fusion of the upper cervical spine is a more formidable procedure. Furthermore, the fusion technique does not provide any immediate stability. It should be reserved for those patients in whom the posterior elements are unsuitable for arthrodesis, either because of tumour involvement or because of previous laminectomy.

SUMMARY

Neoplasms of the atlas and axis are uncommon and the majority are metastatic tumours. The patients frequently present with acute neck pain as the result of a pathological fracture. Restriction of rotation is an important sign.

The diagnosis may be difficult, largely because it is difficult to evaluate radiographs of this region. Laminectomy may be hazardous. In those patients with a life expectancy of one year or more, operative stabilization should be considered for the relief of disabling pain or cord compression.

REFERENCE

1. OTTOLENGHI, C. E., SCHAJOWICZ, F. AND DE SCHANT, F. A.: Aspiration biopsy of the cervical spine; techniques and results in thirty-four cases, *J. Bone Joint Surg.* [*Amer.*], **46A**: 715, 1964.

RÉSUMÉ

Les néoplasmes de l'atlas et de l'axis sont rares et la majorité d'entre eux sont des tumeurs métastatiques. Les malades se plaignent souvent d'une douleur cervicale qui résulte d'une fracture pathologique. La limitation du mouvement de rotation est un signe clinique important.

Le diagnostic peut être difficile, surtout en raison de la difficulté d'interpréter les radiographies de cette région. La laminectomie peut présenter de gros risques. Chez les malades qui sont susceptibles d'avoir une survie d'au moins un an, on peut envisager une opération ayant pour objet de stabiliser et de soulager la douleur ou la compression de la moëlle.

FRACTURES OF THE FEMORAL NECK ASSOCIATED WITH FRACTURES OF THE SAME FEMORAL SHAFT

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INJURY to the proximal end of the femur can easily escape detection if it is associated with a fracture of the same femoral shaft. The clinician confronted with a patient with multiple injuries, directs his attention to the more serious and life-threatening injuries. The obvious deformity begs for recognition. Even the most junior surgeon recognizes shortening and external rotation of the leg as signs of femoral neck fracture. However, when this deformity is masked by another due to fracture of the same femoral shaft, only the surgeon who is aware of this complication and thoroughly assesses the patient and the radiographs will make the correct diagnosis.

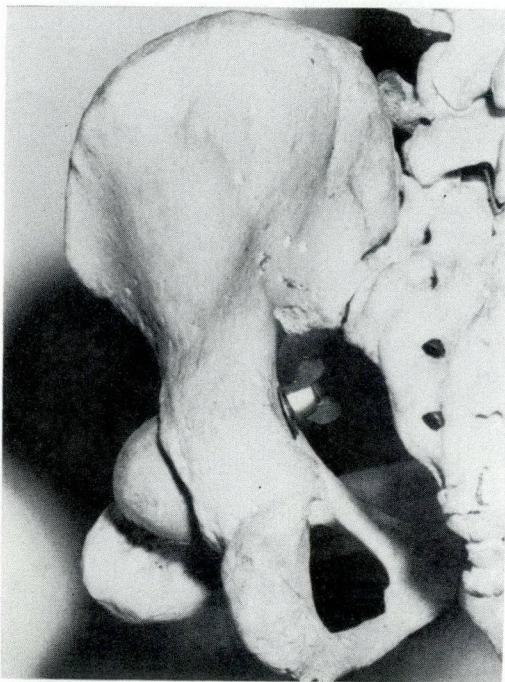The pathomechanics of this double fracture suggests that it is a distinct clinical

Fig. 2.—The hip is flexed and abducted. The femoral head is well seated in the acetabulum.

Fig. 1.—The hip is flexed and adducted. The femoral head is well out of the acetabulum.

entity which should be anticipated from an analysis of the mechanism of injury. Fracture of the femoral neck associated with a fracture of the same femoral shaft usually results from longitudinal compression of the femur; for example, when the knee of a front-seat automobile passenger strikes the dashboard following sudden deceleration. If the femur is in adduction when it is longitudinally compressed (as in the passenger sitting with his legs crossed), the femoral head is well out of the acetabulum and a posterior dislocation or fracture-dislocation may result (Fig. 1). However, if the femur is in abduction, the femoral head is well seated in the acetabulum and cannot dislocate (Fig. 2) and, if all the energy has not been dissipated in shattering the femur, a fracture-dislocation of the hip joint or fracture of the femoral neck results.

As is common practice in orthopedic surgery, we have used Pauwel's well-

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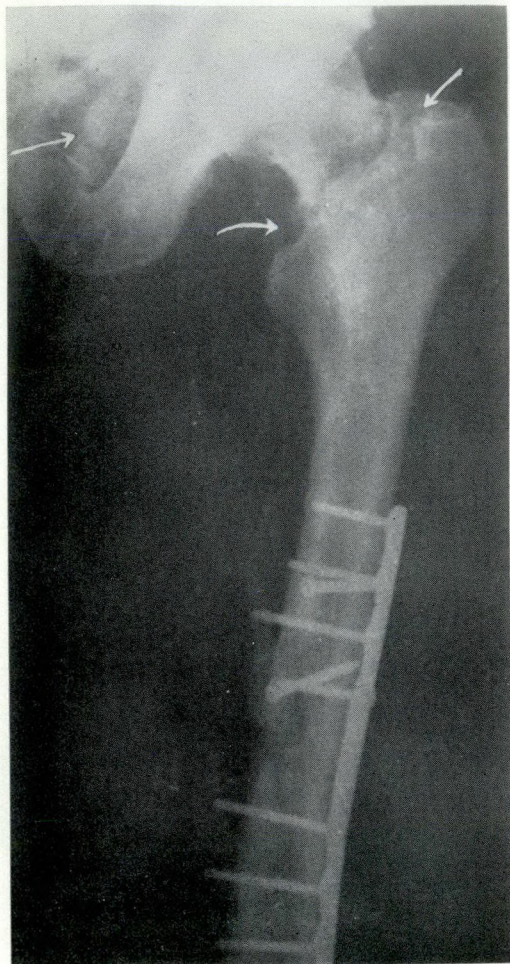
Fig. 3.—Case 1. Check radiograph two months after injury. Note the fracture of the femoral neck.
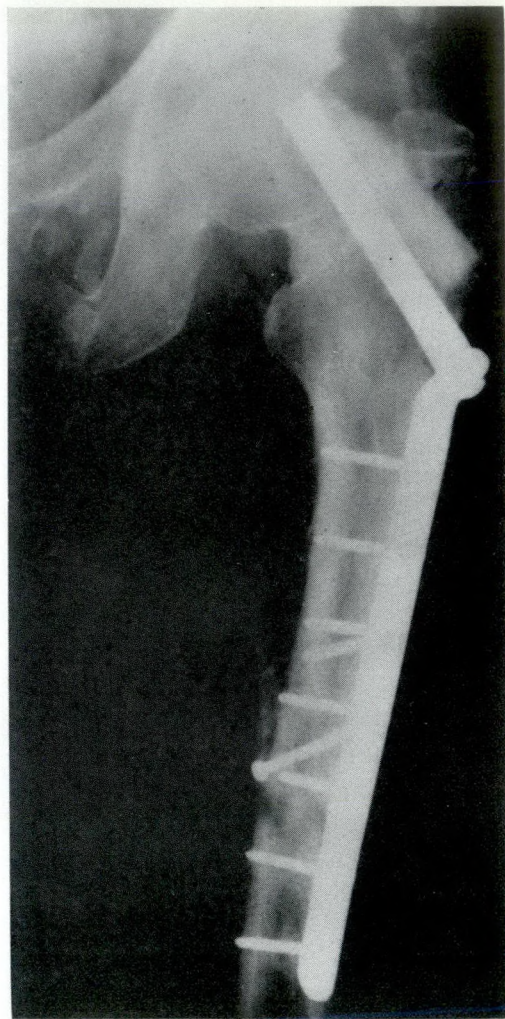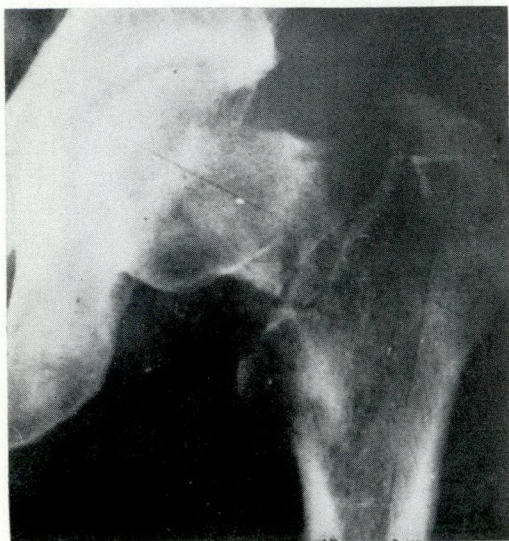
Fig. 5.—Case 1. The varus displacement has been corrected by internal fixation and bone grafting.

known classification of femoral neck fractures, which briefly is as follows: Type I— the plane of the fracture approaches the horizontal, i.e. less than 30°; Type II—the plane of the fracture is 30 to 50°; Type III—the plane approaches the vertical, i.e. 70° or more.

CASE REPORTS

Case 1.—A 49-year-old Flight-Sergeant, who was driving at 50 m.p.h. when his car skidded and hit a bridge, sustained a compound fracture of the left femoral shaft. The wound was debrided, the fracture plated and the limb

Fig. 4.—Case 1. Four months after injury. There is non-union and varus displacement of the femoral neck.

immobilized in a hip spica. Two months later follow-up radiographs revealed for the first time that the femoral neck on the same side was also fractured (Fig. 3). This break was thought to be impacted and uniting in a satisfactory position. The patient was free of pain and had a good range of hip movement. Four months after injury, however, there was considerable varus displacement and evidence of delayed union of the shaft fracture (Fig. 4). The varus displacement of the neck was corrected, and both fractures were immobilized with a Smith-Petersen nail and long side plate (Fig. 5). A cortical graft was inserted into the neck above the nail, and cancellous chips were placed about the shaft fracture. The patient was immobilized in a hip spica for three more months. Eleven months after injury, when both fractures had united, full weight bearing was allowed. The patient was free of pain but was disabled because his hip and knee movements were restricted.

Case 2.—S.K., a 35-year-old man, was in the front seat of an automobile that was involved in a "head-on" collision. He sustained extensive facial lacerations and an obvious fracture of the right femur. An open reduction was done and the femoral fracture fixed with

Fig. 7.—Case 2. The femoral neck fracture has been reduced and immobilized with three Moore pins. The cancellous bone graft is not seen.

Fig. 6.—Case 2. Note the fracture of the femoral neck.

a Küntscher nail. Two months later he still limped, but was bearing full weight and did not complain of pain. A radiograph at this time showed a hitherto unrecognized fracture of the femoral neck (Fig. 6). At operation four months after injury, the fracture gap was found to be filled with dense fibrous tissue. There was no evidence of union. The fracture was fixed with three Moore pins and a cancellous graft was inserted anteriorly (Fig. 7). The patient was kept in a hip spica for 3½ months. Four months after operation, when the fracture of the neck had united, the Moore pins were removed. When reviewed 14 months after injury, the patient had no symptoms, and was ready to return to work, although he still walked with a limp and had moderately restricted hip and knee movement.

Case 3.—A.M., a 40-year-old labourer, was working inside a steel tank 100 feet above the ground when the scaffolding on which he was standing collapsed. He fell to the floor of the tank, landing on both knees and smashing his face against the inner wall. The most obvious of his many injuries were grossly displaced compound fractures of both femurs. The initial radiographs also disclosed severely comminuted fractures of both patellae (Fig. 8). He was taken to the operating room
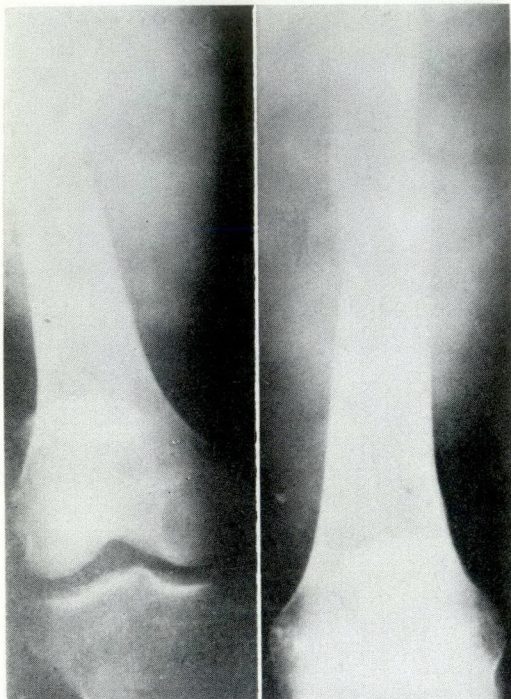
Fig. 8.—Case 3. The hip joints are not included in the film which is also of poor quality, a frequent fault with emergency portable films.

where, after thorough debridement, an intramedullary Küntscher nail was placed in the right femur and the left femur was immobilized on a Thomas splint.

Five days later, because of constant groin pain and spasms of the leg muscles, the pelvis was radiographed. Both femoral necks were fractured (Fig. 9). These fractures were reduced under direct vision, bone grafts were inserted and the bones were internally fixed using lag screws (Fig. 10). Biopsy of both femoral heads at this time showed no evidence of avascular necrosis.

The patient recovered with remarkable speed. One year after injury, all his fractures had united and he was walking with two canes. He had no pain but complained of knee stiffness, particularly of the right which had a 10° extensor lag and would flex only to 50°. A quadricepsplasty was attempted but the incision became infected and the operation failed. Three months later, while quite drunk, he tripped, felt a tearing sensation in his right thigh and could then bend his knee to 90°— a range of motion he has since maintained.

Case 4.—B.D., a 25-year-old man, was in the front seat of an automobile during a head-on collision. He sustained a compound fracture of the left femur and a comminuted fracture of the left patella. He also lacerated his forehead and left shoulder, and abraded his left knee. As primary treatment his lacerations were debrided and closed, the left patella was removed and the femoral fracture was reduced under direct vision and held with an intramedullary Küntscher nail. On survey of the initial films, the attending surgeon and radiologist discovered no other bony injury. Later the same day a post-reduction film of the femur showed a fracture of the femoral neck in the vertical plane (Pau-


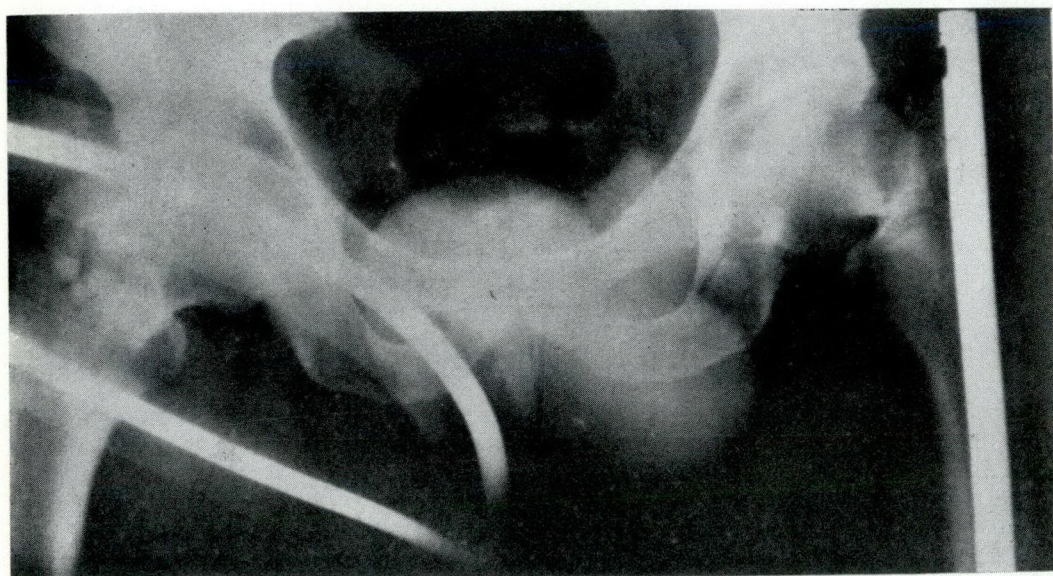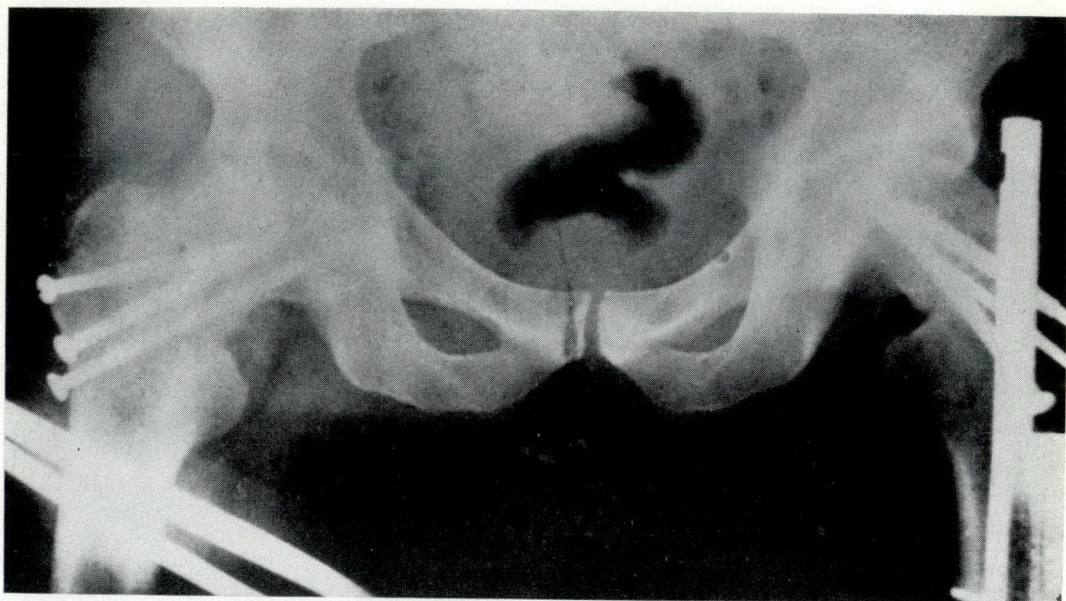Fig. 9.—Case 3. Bilateral femoral neck fractures.

Fig. 10.–Case 3. The femoral neck fractures have been reduced and immobilized with three lag screws.

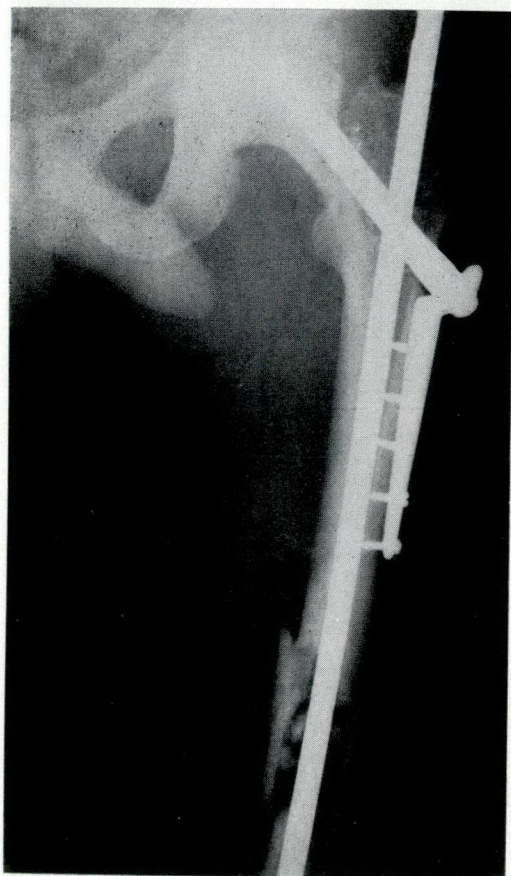**Fig. 11.**–Case 4. A Smith-Petersen nail has been introduced anterior to the Küntscher nail.
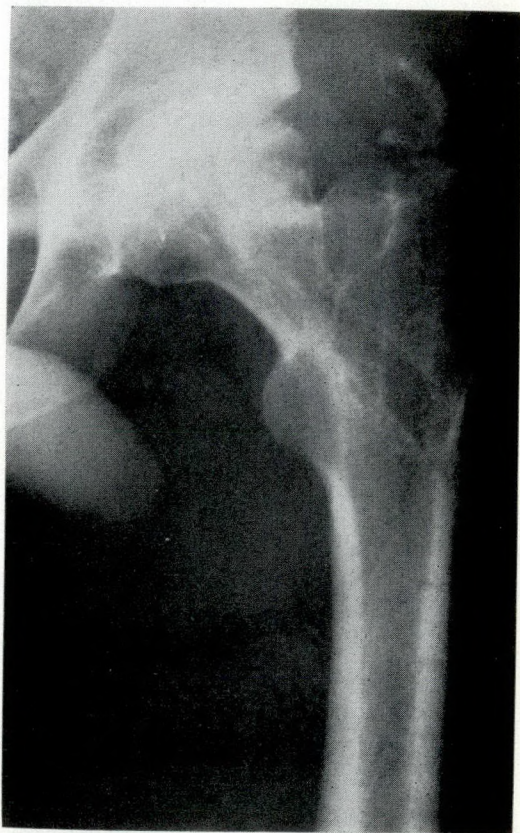
Fig. 12.–Case 4. Eighteen months after injury all fixation devices have been removed. Union is solid and there is no evidence of avascular necrosis.

wel's Type III subcapital fracture). Four days later the surgeon did a closed reduction of the neck fracture and attempted to fix it by inserting a Smith-Petersen nail and plate. He had great difficulty in introducing the nail which kept striking against the Küntscher nail. Finally, he introduced the nail through a V-shaped slot cut in the front of the neck (Fig. 11). The femoral neck united in 6 months and at 18 months radiographs showed no evidence of avascular necrosis (Fig. 12). The patient, however, still complains of weakness in the left knee and has a noticeable limp on walking.

Case 5.—T.J., a 54-year-old woman, was in the front seat of an automobile during a head-on collision. She fractured her femoral shaft (Fig. 13). An open reduction was carried out
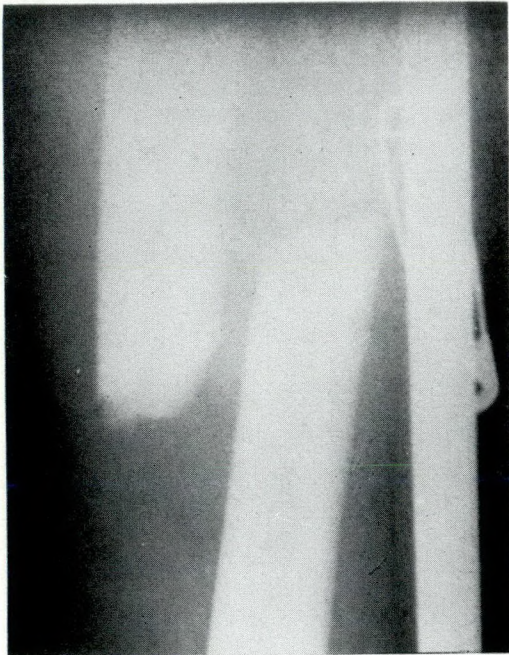
Fig. 13.—Case 5. On the basis of this radiograph, treatment was undertaken.

and the fragments fixed with a plate. A long leg plaster was applied. Two months later the plaster was removed and a radiograph of the femur was obtained which, for the first time, included the hip (Fig. 14). The surgeon noted that the femoral neck was fractured but chose to treat it expectantly. He allowed partial weight bearing when the radiograph indicated that the femoral shaft was united.

Fourteen months after the injury, the patient suddenly developed a sharp pain in her
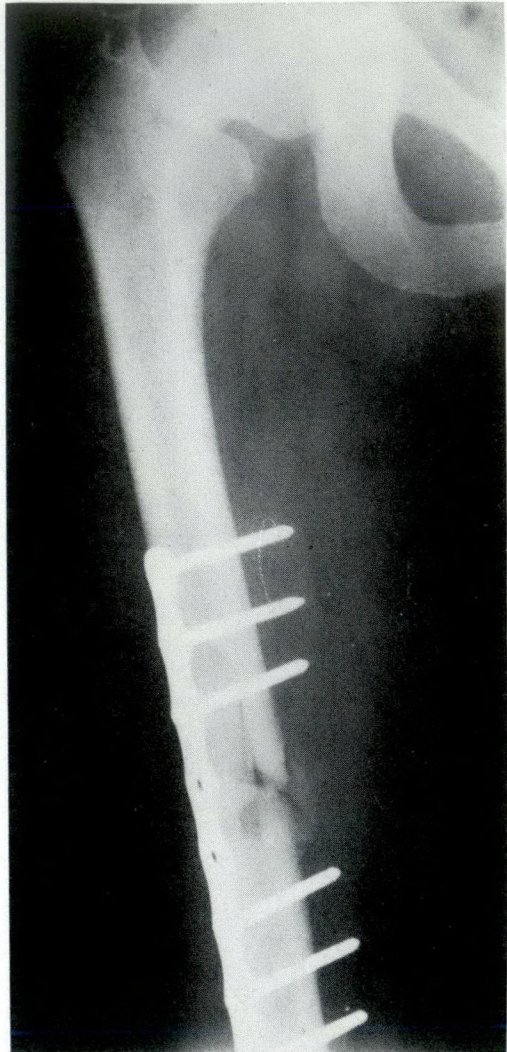
Fig. 14.—Case 5. Note fracture of the femoral neck and the varus displacement.

thigh and returned for advice. A radiograph showed that the femoral neck had united in the varus position. The femoral shaft had not united and the plate had broken. The femoral shaft was fixed using an intramedullary nail and bone graft. Four years after injury, the patient still complains of easy fatigability, limp and shortening of her leg.

Case 6.—S.M., a 59-year-old woman, was in the front seat of an automobile during a head-on collision. The initial radiographs revealed an undisplaced Pauwel's Type III subcapital fracture and a fracture of the femoral shaft (Figs. 15 and 16).

The shaft fracture was reduced under direct vision and fixed with a plate. Skeletal traction
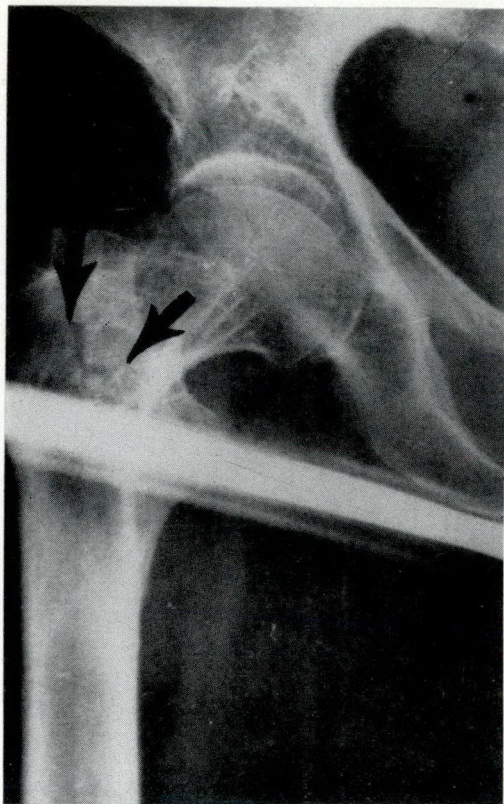
Fig. 15.—Case 6. Fracture of the femoral neck.

was continued and knee movement begun. Ten days later the femoral neck fracture was reduced and fixed using a sliding nail (Fig. 17). Two months after injury, she has regained a good range of hip movement but, in spite of early mobilization, the knee is still stiff.

DISCUSSION

The fracture of the femoral neck in these double injuries is most commonly a Pauwel's Type III intracapsular fracture. Initially, it may be undisplaced and hence difficult to recognize (Fig. 18). Displacement, when it occurs, is due to the proximal femoral shaft fragment going into external rotation. This produces a characteristic narrowing of the femoral neck on the anteroposterior film. This displacement must be recognized and corrected to prevent the subsequent external rotation deformity of the leg.

Dencker[1] described four intertrochanteric fractures associated with femoral shaft fractures in people who had been knocked
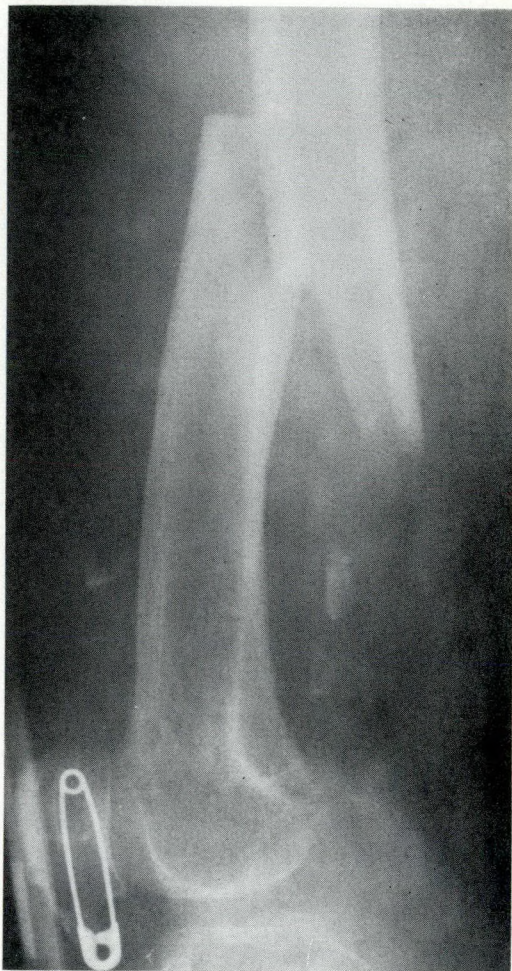
Fig. 16.—Case 6. Fracture of the femoral shaft.

to the ground. The mechanism of injury in these individuals was, of course, different from that seen in traffic accidents.

An analysis of our patients and others[1-3] in whom the femoral neck fracture went unrecognized suggests some important guiding principles for the treatment of such fractures.

In our series.—Case 1. The fracture went unrecognized for two months while the patient was in a hip spica. At four months the fracture was still not united and had drifted into varus. *Case 2.* The fracture was unrecognized for two months while the patient was bearing weight. At four months there was no sign of union. *Case 3.* The fracture was recognized after two months of non-weight bearing. At the end of a year, when partial weight bearing was
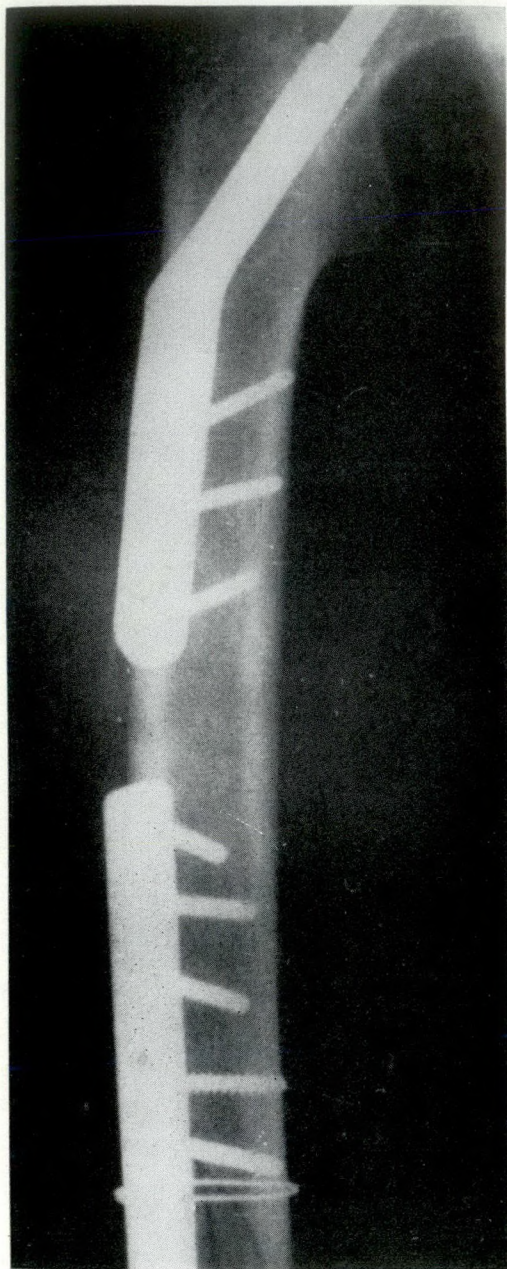
Fig. 17.—Case 6. Internal fixation of both fractures.

Fig. 18.—An undisplaced Pauwel's Type III fracture of the femoral neck.

allowed, the fracture united in varus. *Case 5*. The fracture was recognized at five months and ultimately united in varus.

In Kimbrough's series.[3]—*Case 1*. The fracture was first recognized at 14 months. It had not united and avascular necrosis was present. *Case 2*. The fracture was recognized at four months when the patient came out of a hip spica. It had united in

varus. *Case 3*. The fracture was recognized at two weeks, and then treated in traction for five months with the hip and knee both flexed at 90°. At the end of that period, there was still no sign of union.

In Dencker's series.[1]—*Case 3*. The fracture was recognized at 10 months. It had not united.

Thus, of the eight patients in whom the fracture went unrecognized for varying periods of time, only two united, but both had drifted into significant varus. In Kimbrough's series[3] one patient developed avascular necrosis and a Moore arthroplasty, done as a late reconstruction, was not successful. All such non-unions can be treated successfully by open reduction, internal fixation and bone grafting. None developed avascular necrosis. All femoral neck fractures that were recognized early and treated by internal fixation united without problems.

Early recognition is of paramount importance. Definitive treatment should never be begun until a complete radiological examination has been carried out. Radiographs of the femur should include both hip and knee joints, particularly the hip, because injuries here are so easily overlooked.

Femoral neck fractures should be treated by internal fixation with or without open reduction. The method of fixation depends on the treatment chosen for the femoral shaft fracture. In the present series, lag screws or Knowle's pins were used in conjunction with an intramedullary nail. Küntscher's double nail combination (an interdigitating intramedullary and cervical nail) seems to provide excellent fixation for these two fractures, although we have had no experience with this method. Internal fixation of the femoral shaft facilitates subsequent reduction of the cervical fracture, permits early joint mobilization and more rapid return of function, particularly of the knee.

Femoral neck fractures that have not united should be reduced under direct vision and held by internal fixation and bone grafting. If the internal fixation is rigid, a hip spica is probably unnecessary.

In these double femoral fractures, the ultimate prognosis of the hip fracture appears to be good. There are often associated knee injuries,[4] especially comminuted fractures of the patella. Both Ritchey, Schonholtz and Thompson[4] and Kimbrough[3] emphasized that knee injuries are frequently overlooked; this is also our experience. In the patients described in this paper, residual disability was due, not so much to the femoral shaft fracture, but to concomitant knee and, to a lesser degree, hip injuries.

Summary

Fractures of the femoral neck associated with fractures of the same femoral shaft often escape detection. Six patients with such double fractures are described and some of the current literature is presented. In the treatment of these femoral neck fractures the following principles are recommended: (1) Definitive treatment of a fractured femur must never be undertaken until both the hip and the knee joint have been examined clinically and radiologically. (2) Internal fixation of the femoral shaft should be done first to facilitate subsequent reduction and fixation of the cervical fracture. (3) Internal fixation of the femoral neck fracture is mandatory. (4) Unrecognized femoral neck fractures in which non-union develops, should be reduced under direct vision and held by internal fixation and a bone graft. If internal fixation is rigid, a hip spica is unnecessary.

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Résumé

Il arrive souvent que des fractures du col fémoral coïncidant avec des fractures du même fémur échappent à l'attention. Les auteurs présentent six malades souffrant de cette double fracture et ils passent en revue la littérature. Pour traiter ces fractures du col fémoral, ils conseillent de s'inspirer des principes suivants: (1) Le traitement définitif d'un fémur fracturé ne doit jamais être entrepris sans avoir au préalable examiné, cliniquement et radiologiquement, et la hanche et le genou. (2) La fixation interne du fémur devrait être faite en premier lieu, en vue de faciliter la réduction subséquente et la fixation de la fracture du col. (3) Il est impérieux de procéder à la fixation interne de la fracture cervicale. (4) Les fractures du col fémoral restées ignorées et où il y a absence de cal devraient être réduites en vision directe et maintenues en place par fixation interne et greffe osseuse. Si la fixation interne est rigide, il est inutile de poser un spica de la hanche.

ROTATIONAL SUBLUXATION OF THE SCAPHOID*

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ROTATIONAL subluxation of the scaphoid is an uncommon but interesting wrist injury. Of nine such cases described in the present communication, five have been followed for at least two years and will be reviewed in detail. The characteristic radiological features of this injury are: (1) the vertical position assumed by the scaphoid in relation to the long axis of the radius (Fig. 1);

Fig. 2.—Widening of the space between the scaphoid and lunate, and foreshortening of the scaphoid in the anteroposterior view.

Fig. 1.—The vertical position of the scaphoid in the lateral view (lunate shown in broken lines).

(2) the variable widening of the space between the scaphoid and the lunate bones (Fig. 2); (3) the foreshortened appearance of the scaphoid in the anteroposterior view (Fig. 2). This subluxation usually follows an injury that produces an intercarpal ligament tear.

Russell,[1] in 1949, described 59 patients with intercarpal dislocation, 4 of whom had rotational subluxation of the scaphoid,

i.e. 6.8% of the intercarpal dislocations in his series.

The carpal bones of each row are held together by strong fibrocartilaginous interosseous ligaments (Fig. 3). In addition,
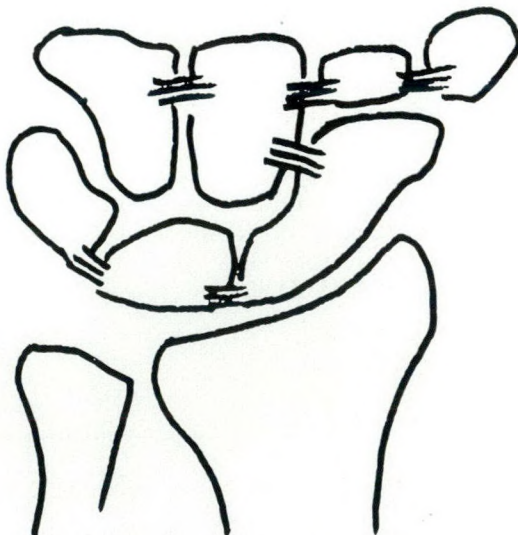

Fig. 3.—Interosseous ligaments of carpal bones.
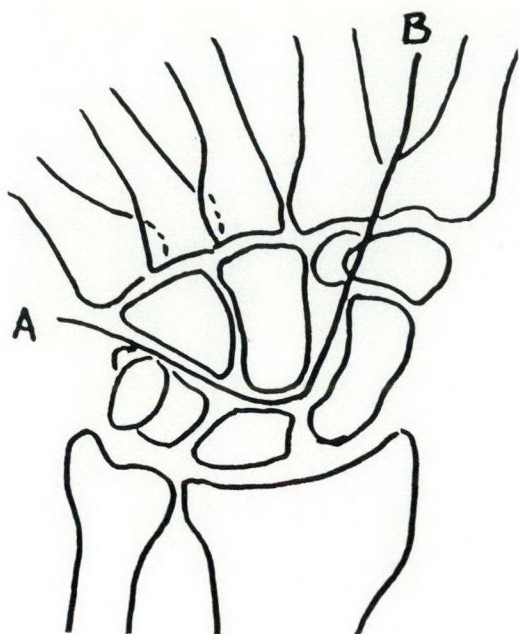
Fig. 4.—Normal plane of intercarpal motion.

there are fibrous dorsal and palmar intercarpal ligaments which assist in maintaining stability. A single interosseous ligament passes between the proximal and distal carpal rows—between the scaphoid and capitate. When the ligaments between
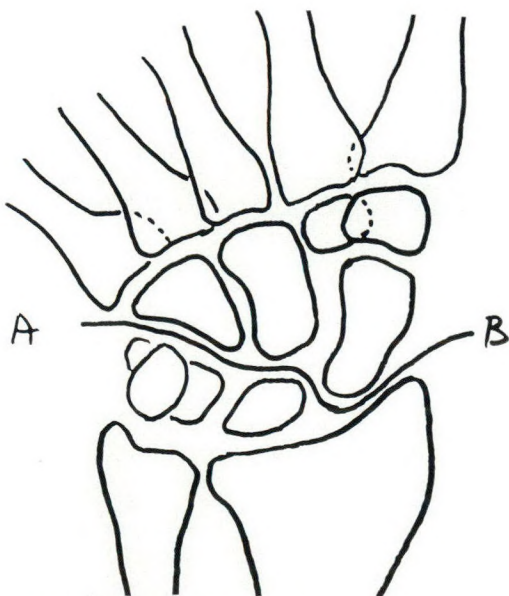
Fig. 5.—Plane of intercarpal motion after division of the scapholunate ligament.

the scaphoid and lunate are torn, the proximal pole of the scaphoid is allowed to rotate dorsally and move with the distal carpal row.

An analysis of movement at the wrist joint shows that 54% of flexion and extension occurs at the radiocarpal joint, and the remainder at the midcarpal joint.[2]

The midcarpal joint normally moves through a plane medial to the scaphoid (A-B, Fig. 4), and the plane extends distally between the first and second metacarpal bases. After the ligaments between the scaphoid and lunate are disrupted, the plane of movement is radically altered to run between the scaphoid and lunate bones (A-B, Fig. 5). Ulnar deviation of the hand normally takes place by movement at the radiocarpal joint. When deviation is forced, the proximal row of carpal bones is shifted laterally pressing against the scaphoid and closing the space between the scaphoid and lunate. Radial deviation occurs at the midcarpal joint and, to avoid the radial styloid during this movement, the scaphoid rotates into the palm. Forced dorsiflexion of the wrist presses the carpal bones of the proximal row together.

Rotational subluxation of the scaphoid may be either primary or secondary. The latter is seen after incomplete reduction of a major carpal dislocation and may be difficult to diagnose.[3]

Case Reports

Case 1.—N.H., a 53-year-old laboratory technician, was tilting a 200-lb. machine when it suddenly slipped. He received its full weight on his supinated left hand, which "spread and snapped". Following this, he had wrist pain that persisted for one year. On examination he had a swelling over the dorsum of the left wrist, occasional clicking on movement, tenderness over the proximal carpal row and moderate pain. Radiographs showed marked widening of the interosseous space between the scaphoid and lunate, and foreshortening of the scaphoid in the anteroposterior view; in the lateral view the scaphoid was rotated vertically (Fig. 6). Deviation of the hand to the ulnar side tended to close the space between the scaphoid and the lunate (Fig. 7).

At operation, the anterior approach was used. On opening the capsule of the wrist joint, the proximal pole of the scaphoid was seen

Fig. 6a
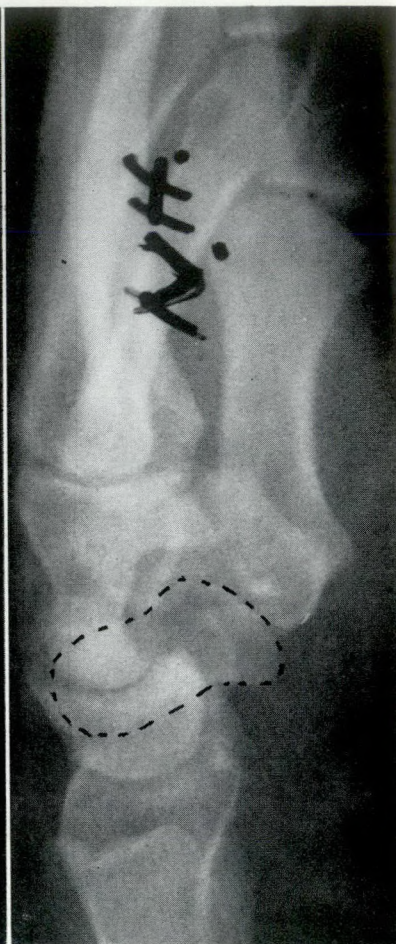
Fig. 6b

Fig. 6.—Case 1. (a) Anteroposterior view shows widening of the space between the scaphoid and lunate, and foreshortening of the scaphoid. (b) Lateral view shows the vertical position of the scaphoid (dotted lines).

to be rotated dorsally. Remnants of the intercarpal ligament remained but there was a wide gap between the scaphoid and the lunate. The scaphoid was levered into its normal position in relation to the lunate and, after curettage of the adjacent surfaces, a threaded wire was passed through the scaphoid into the lunate. A trough was then cut in the scaphoid and lunate distal to the articular surfaces and, into this, a cancellous iliac graft was inserted. The wrist was then immobilized for three months (Fig. 8). Following this, the scaphoid and lunate were clinically stable and the patient's symptoms were relieved. Radiographs of the scaphoid and lunate, however, did not show solid bony continuity (Fig. 9).

Fig. 7.—Case 1. Anteroposterior view shows narrowing of the space in ulnar deviation.
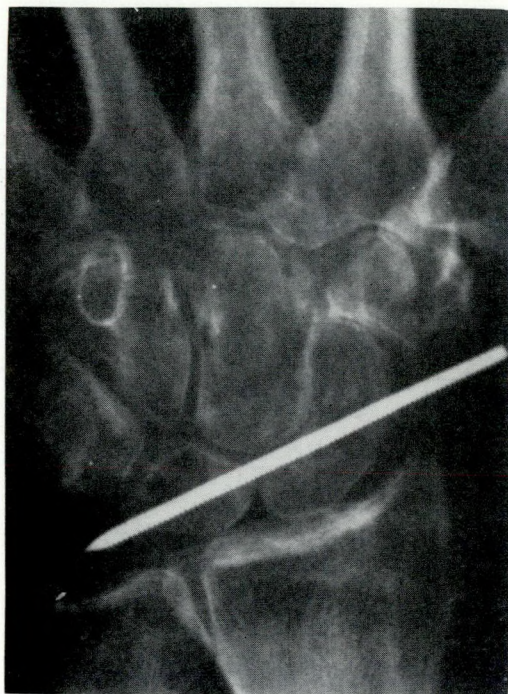
Fig. 8.—Case 1. The scaphoid now held in a normal position by a threaded wire.

Case 2.—After a fall on his outstretched left hand, S.W., a 48-year-old man, had pain, swelling and limitation of movement. He was examined on the same day and, on the radiographs, the scaphoid was in a vertical position and the space between the scaphoid and lunate was widened (Fig. 10).

At operation through a dorsal incision, a wide gap was seen between the scaphoid and lunate. It was quite evident that the scapholunate ligament was completely disrupted, allowing the scaphoid to move with the distal carpal row in palmar flexion. The scaphoid was reduced and fixed with a Kirschner wire passed from the scaphoid into the lunate (Fig. 11). The wire was left in place for six weeks. The end result was quite satisfactory.

Case 3.—A.B., a 33-year-old man, gave only a sketchy description of his original injury, but was known to have had symptoms when his right wrist was radiographed three years before (Fig. 12). On analysis, the symptoms suggested rotational subluxation of the scaphoid. He continued to have pain in the wrist and some weakness. Radiographs showed that there were some degenerative changes in the scaphoid and nearby bones (Fig. 13).
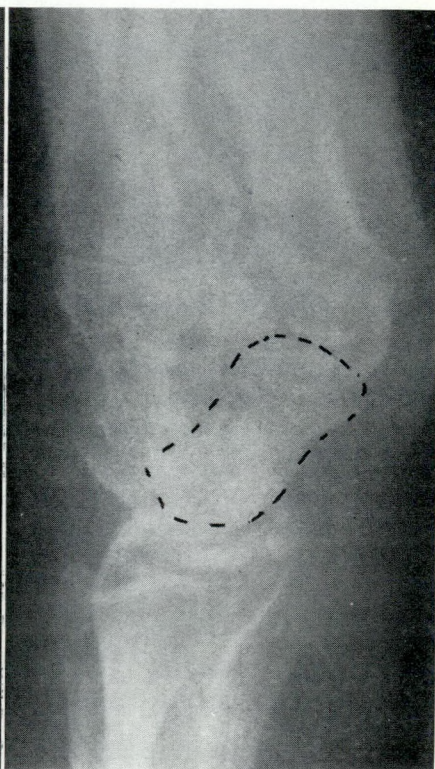
Fig. 9a   Fig. 9b

Fig. 9.—Case 1. (a) Anteroposterior and (b) lateral views show the normal alignment of the scaphoid.
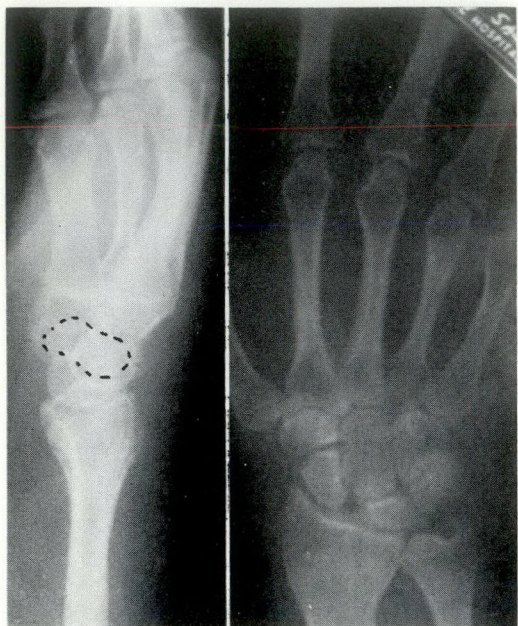
Fig. 10a   **Fig. 10b**
Fig. 10.—Case 2. (a) Lateral and (b) anteroposterior views show three characteristic findings of rotational subluxation of the scaphoid.
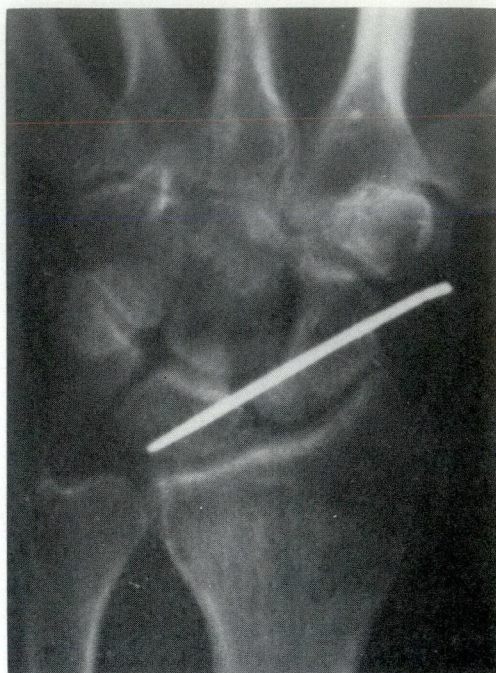
Fig. 11.—Case 2. The scaphoid realigned and held with a Kirschner wire.
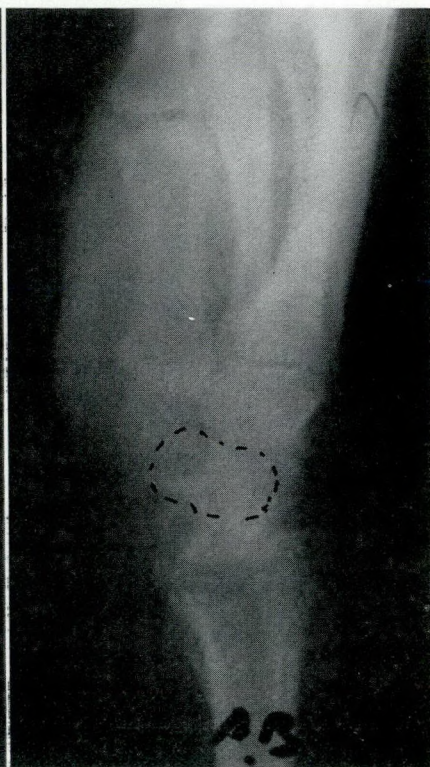
Fig. 12a   **Fig. 12b**
Fig. 12.—Case 3. (a) Anteroposterior and (b) lateral views show findings suggestive of rotational subluxation of the scaphoid.
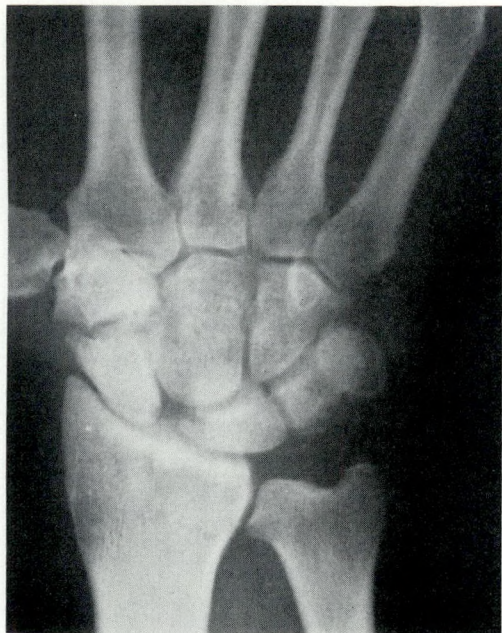
Fig. 13.—Case 3. Anteroposterior view three years later shows more obvious widening of the space between the scaphoid and lunate, and marked degenerative changes in the wrist.

At operation, extensive erosion (down to subchondral bone) was seen on the radial surface of the scaphoid.

Case 4.—F.M., a 48-year-old worker in a stone-crushing plant, severely injured his right hand when a large stone fell from a height. He had a compound dislocation of the hand, and the scaphoid and first metacarpal ray were displaced laterally (Fig. 14). In addition, he had a midcarpal dislocation and a fracture of the radial styloid. The bones were fixed with multiple Kirschner wires but he was left with a gap between the scaphoid and lunate (Fig. 15). In this particular case, the fixation was inadequate and the deformity recurred. Subsequently a proximal-row carpectomy was done.

The next patient illustrates a contentious point, namely, the scaphoid may subluxate following excision of the lunate, a situation in which the scapholunate ligament is no longer functioning.
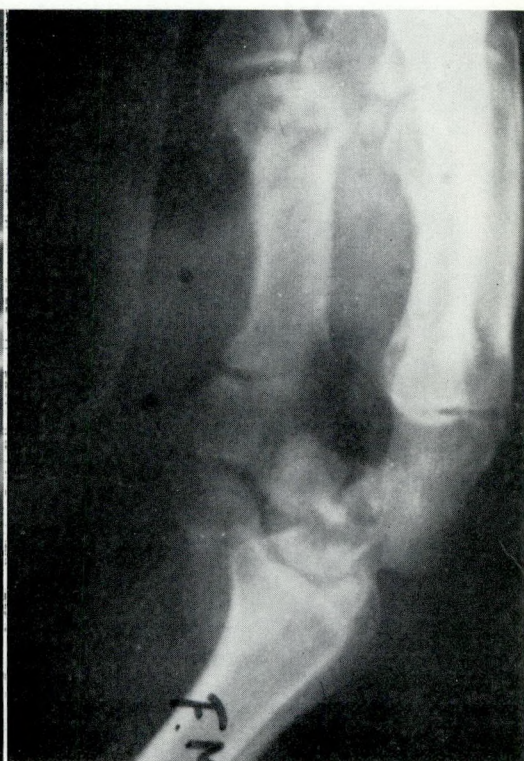
Fig. 14.—Case 4. Compound dislocation: the scaphoid and first metacarpal ray are displaced radially and inferiorly.
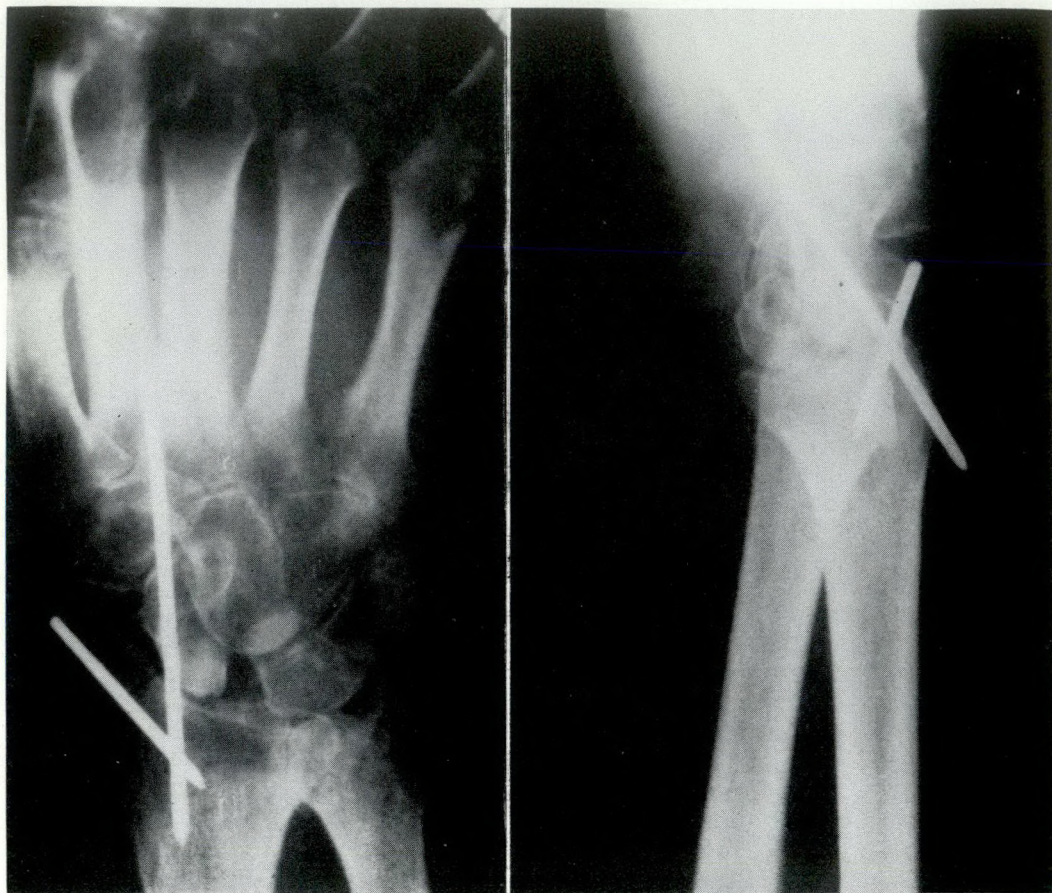
Fig. 15.—Case 4. Incomplete reduction of severe carpal dislocation.

Case 5.—R.C., a 41-year-old woman, had the lunate from her left wrist excised 6½ years earlier. She had limited movement and pain over the dorsum of the wrist. The scaphoid was rotated and lying in a vertical position; there were marked degenerative changes in the joint (Fig. 16).

DISCUSSION

Vaughan-Jackson[4] described a patient who, after an injury described as "a strain", could voluntarily sublux the scaphoid. I have done further studies on this point on a wrist from a freshly amputated arm. After the scapholunate ligament was divided, the scaphoid, being untethered, could be rotated dorsally during palmar flexion. Recently, I saw four other patients with rotational subluxation of the scaphoid. The results of treatment in these patients are still pending. Two were involved in car accidents. One recalled putting his hand forward towards the seat in front before being forcibly thrown against it. The other was driving the car but could not recall any specific details of the injury. Another, a teenage girl, was playing tennis when the ball struck the frame of the racquet. She felt a sudden jarring pain which radiated up her arm. The fourth, a 56-year-old alcoholic, complained of pain in his wrist but refused treatment. He had no recollection of earlier injury but radiographs showed degenerative changes in the radiocarpal joint.

With respect to complications, if no arthritic changes have developed, late injuries are treated by scapholunate fusion using the anterior approach. Proximal-row resection or, alternatively, wrist fusion can be done when arthritis is present.

Recent injuries producing rotational sub-
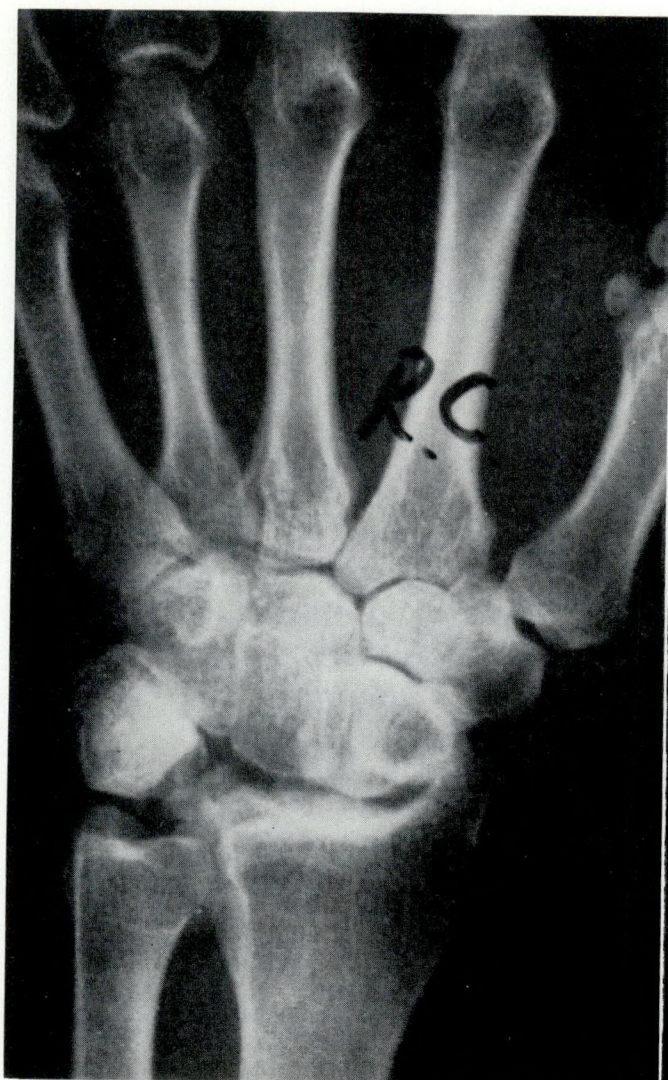
Fig. 16a   Fig. 16b

Fig. 16.—Case 5. (a) Anteroposterior view shows marked foreshortening of the scaphoid after lunate excision. (b) Lateral view shows the vertical position of the scaphoid.

luxation of the scaphoid can be treated adequately by Kirschner wire fixation which transfixes the scaphoid and lunate. This is best accomplished by open reduction, the end of the wire being left in a subcutaneous position. Plaster immobilization is continued for six to eight weeks and the wire removed under local anesthetic after three months.

SUMMARY

Rotational subluxation of the scaphoid occurs following an intercarpal ligament tear. This injury should be considered in the differential diagnosis of wrist pain and following the reduction of a major carpal dislocation. It has developed after excision of the lunate and has been blamed for the bad results of this operation. Arthritis may develop in the wrists of those with untreated dislocations. For this reason, such injuries should be actively treated as soon as they are recognized.

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RÉSUMÉ

La subluxation rotatoire du scaphoïde survient après déchirement d'un ligament intercapien. Il importe de songer à cette lésion au moment du diagnostic différentiel d'une douleur du poignet et après réduction d'une luxation majeure du carpe. Cette subluxation s'était produite après excision de l'os semi-lunaire, qui avait été incriminée pour les résultats médiocres de cette opération. L'arthrite peut s'installer dans les poignets des malades dont la luxation n'a pas été traitée. C'est pourquoi il importe de traiter adéquatement ces lésions dès leur découverte.

CLOSED INTRAMEDULLARY NAILING

An attempt is made to assess the value of primary intramedullary nailing of tibial shaft fractures by comparing the early and late results in nailed and conservatively treated fractures of comparable severity. In all instances, the nail was inserted by a closed technique. Reduction of the fracture and subsequent introduction of the intramedullary nail were controlled by the aid of an image intensifier mounted on a C arch allowing controls in two planes.

The result obtained in 32 fractures of the tibial shaft in 31 patients treated by closed intramedullary nailing in the years 1963 to 1964 are presented. The medullary cavity was reamed out and the fracture stabilized by a heavy nail with a mean diameter of 11 mm. All patients were operated upon within six weeks after the trauma.

For comparison, a series of 53 fractures of the tibial shaft in 52 patients were selected out of 112 fully documented patients treated by conservative methods during the same period. The two groups under observation were comparable in regard to mechanism of trauma, site of fracture, and severity of displacement, comminution, and wound. The series comprised more severe fractures than the average unselected series; 50% of the fractures were compound, and the conservative control group included 10 fractures which needed bone grafting because of delayed union.

There were no fractures in the upper third of the tibial shaft. In 69% of the nailed fractures and 68% of the conservatively treated ones, the fracture was in the middle third; in the remainder it was located between the middle and lower thirds. Each group included two patients with double fractures.

There were no significant differences between the two groups in length of time elapsing before the patient could walk without a stick or the interval between trauma and return to work. Two years later, the residual joint stiffness in the knee and ankle joints were of the same degree, whereas tarsus movement was better in the nailed group of patients. The similarity of the results in the nailed and conservatively treated patients indicates that, basically, severity of the trauma and the type of fracture determine the end result.

Rigid internal fixation makes unnecessary the use of external splinting and thus allows early mobilization of the knee, ankle and tarsus joints. This is an important argument in favour of operative treatment. The results in this comparative study do not, however, reveal any significant difference between the nailed and conservatively treated fractures, although in the nailed group the good range of movement in the tarsus was impressive.

The functional result, as measured by the ability to walk without a stick and to return to work, was similar in both groups under observation.

In the authors' opinion, most fractures of the tibial shaft can be adequately treated by conservative measures combined, if necessary, with early subcortical bone grafting.

It is concluded that in most instances conservative treatment of tibial shaft fractures gives good results. In selected instances where fracture retention is difficult, in fractures combined with skin problems, and in multitrauma patients presenting nursing problems, closed intramedullary nailing may offer an alternative to conservative treatment.—Slätis, P. and Rokkanen, P.: Closed intramedullary nailing of tibial shaft fractures; a comparison with conservatively treated cases, *Acta Orthop. Scand.*, **38**: 88, 1967.

CARPOMETACARPAL DISLOCATIONS OTHER THAN THE THUMB: A REPORT OF 11 CASES

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SUBLUXATION or dislocation of the bases of the metacarpals other than the thumb are uncommon and may be overlooked in a patient with multiple injuries from a traffic or industrial accident. These dislocations, which are often accompanied by fractures of the bases of the metacarpals or the adjacent surfaces of the carpal bones, are best described as "fracture-subluxations" or "fracture-dislocations".

This communication describes the case histories and radiographs of 11 patients with this injury treated over the past 10 years. Three of these were the patients of other orthopedic surgeons.

In referring to this injury, Watson-Jones[1] notes that "manipulative reduction by traction on the fingers and strong pressure over the back of the metacarpal is difficult but, fortunately, the imperfect reductions seldom interfere with the function of the hand, despite the bony thickening". He also notes that isolated dislocation of the fifth carpal metacarpal joint is rare. In his chapter on unusual and instructive cases, he shows radiographs of dorsal dislocation of the fourth and fifth metacarpals, palmar dislocation of the second, third, fourth and fifth metacarpals and isolated palmar dislocation of the base of the fifth metacarpal. He draws attention to the difficulty of making the diagnosis of dorsal dislocation from an anteroposterior radiograph and recommends that this injury be treated by closed or open reduction.

According to Boyes,[2] carpometacarpal dislocations are less common than radiocarpal and midcarpal dislocations. Such dislocations upset mechanics and muscle balance so seriously that they should be reduced surgically. In his paper, he describes a patient with dorsal dislocation of the fourth and fifth metacarpal bases on the carpus in whom loss of finger flexion made open reduction and fixation with a nail and

wire necessary three months after injury.

Waugh and Yancey[3] reviewed the literature on carpometacarpal dislocation up to 1948 and found 79 cases. They described two additional patients with simultaneous dorsal dislocations of the bases of the fourth and fifth metacarpals. One, of 3½ months' duration, required open reduction and transverse percutaneous wire fixation. The other was treated successfully soon after injury by closed reduction and plaster fixation.

In a paper in which they referred to 100 cases collected by Picchio in 1954, Shephard and Solomon[4] described four additional patients with carpometacarpal dislocation other than the thumb. Three of them were motorcyclists in whom the impact of the handlebars on the palm dislocated the metacarpal bases. In one patient who died soon after injury, the joint capsule was stripped from the dorsum of the carpus and from the front of the base of the metacarpal. The three surviving patients, treated by closed reduction, had mild residual deformity but no detectable impairment of function or grip.

Ker[5] described isolated palmar dislocation of the base of the fifth metacarpal in a patient who sustained a direct blow over the base of this bone. Closed reduction was

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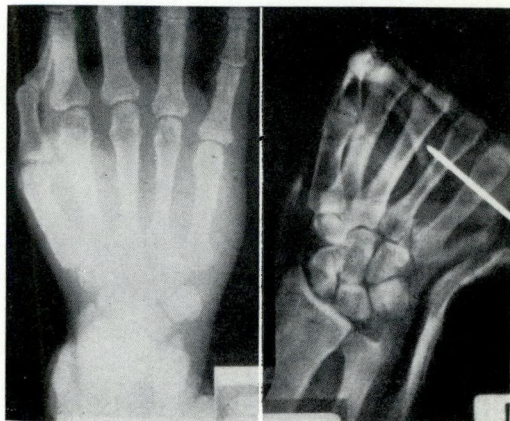
Fig. 1.—Case 1. Fracture-subluxation of the base of the fifth metacarpal before and during treatment.
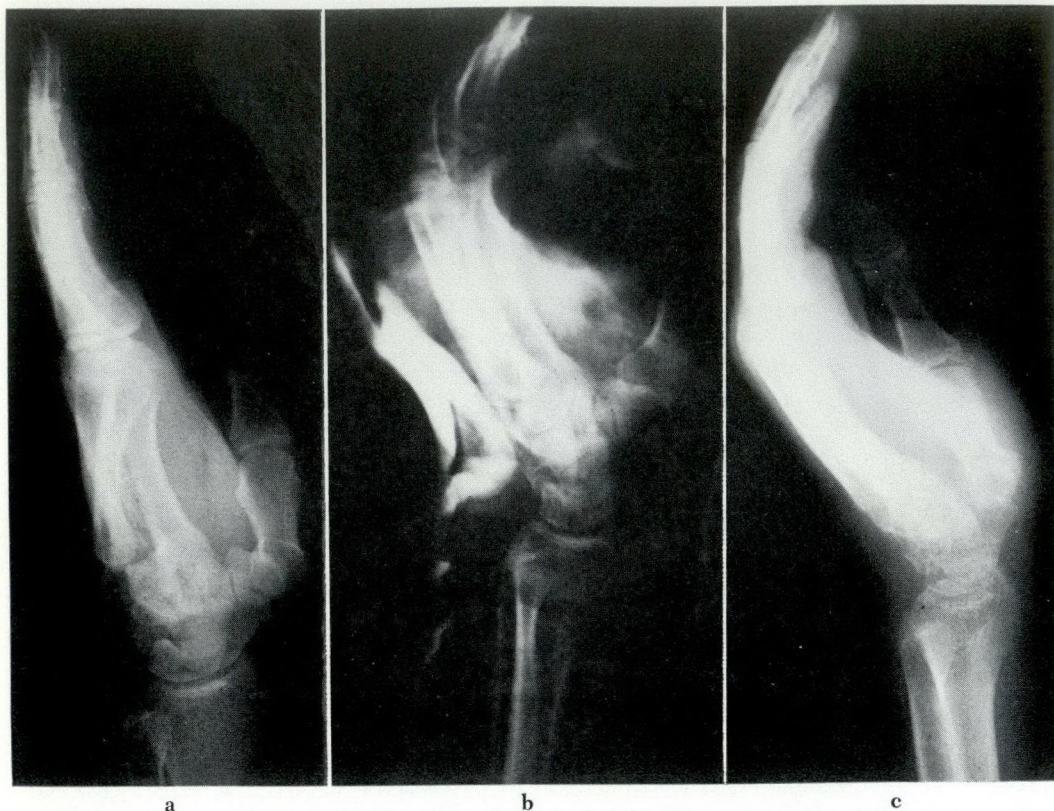
Fig. 2.—Case 2. (a) Dorsal dislocation of the base of the fifth metacarpal with a fracture of the dorsum of the hamate. (b) Reduced in a moulded plaster cast. (c) Six weeks after injury.

unsuccessful and open reduction was necessary to hold the unstable metacarpal base in place with a catgut suture.

CASE REPORTS

Case 1.—A 23-year-old man sustained a blow on the dorsum of his hand and had pain and swelling over the base of the fifth metacarpal. Radiographs showed an ulnar fracture-subluxation of the base of this bone. Closed reduction was unstable but was maintained by a wire passed through the shafts of the fifth and fourth metacarpals and incorporated in a plaster cast (Fig. 1). Early finger motion was encouraged and the fixation was removed in six weeks. The joint was stable and satisfactory.

Case 2.—A 32-year-old man fell down a flight of stairs with a small can in the palm of his hand. The radiographs showed that he had a partial dorsal subluxation of the base of the fifth metacarpal and a fracture of the dorsum of the hamate (Fig 2a). A closed reduction was done and maintained with a

moulded plaster cast (Fig. 2b). Two weeks after injury he returned to his work as a painter. Six weeks after the reduction the cast was removed and the affected bones appeared to be stable (Fig. 2c). For a few months he had an occasional sharp pain in his palm but now has a comfortable and strong hand.

Case 3.—A 23-year-old man injured his right hand in a fight. There was pain and swelling on the dorsum of the hand at the bases of the metacarpals. Radiographs showed that the bases of the third, fourth and fifth metacarpals were dislocated, and the base of the fourth metacarpal was fractured (Fig. 3). Closed reduction was done and maintained with a plaster cast for six weeks. This man was again seen three years later because of an injury to his right thumb. A radiograph of his hand at this time showed no residual deformity or abnormality at the bases of the metacarpals. The end result was satisfactory (Fig. 4).

Case 4.—A 46-year-old man caught his right hand and forearm in the feeder mechanism of
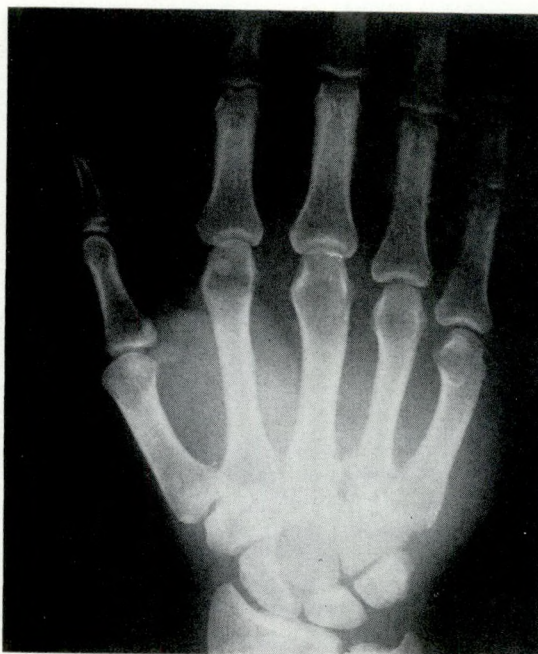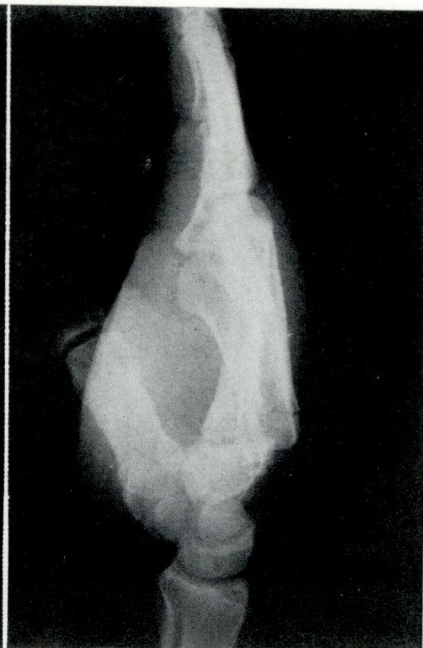
Fig. 3a   **Fig. 3b**

Fig. 3.—Case 3. (a) Dorsal dislocation of the bases of the third, fourth and fifth meta-carpals with a fracture of the base of the fourth metacarpal, styloid process of the third metacarpal and dorsum of the capitate. (b) Lateral view of the same injury.
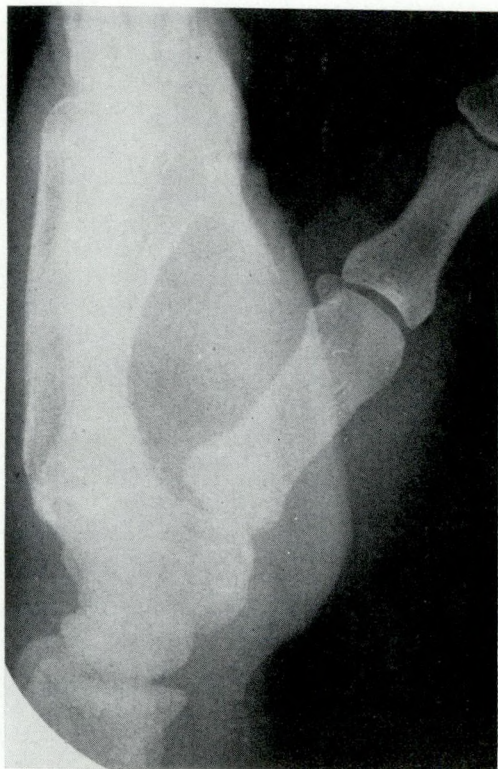
Fig. 4a   **Fig. 4b**

Fig. 4.—Case 3. (a) Lateral and (b) anteroposterior radiographs showing satisfactory position and healing of the metacarpal bases three years after dislocation.

a   b   c
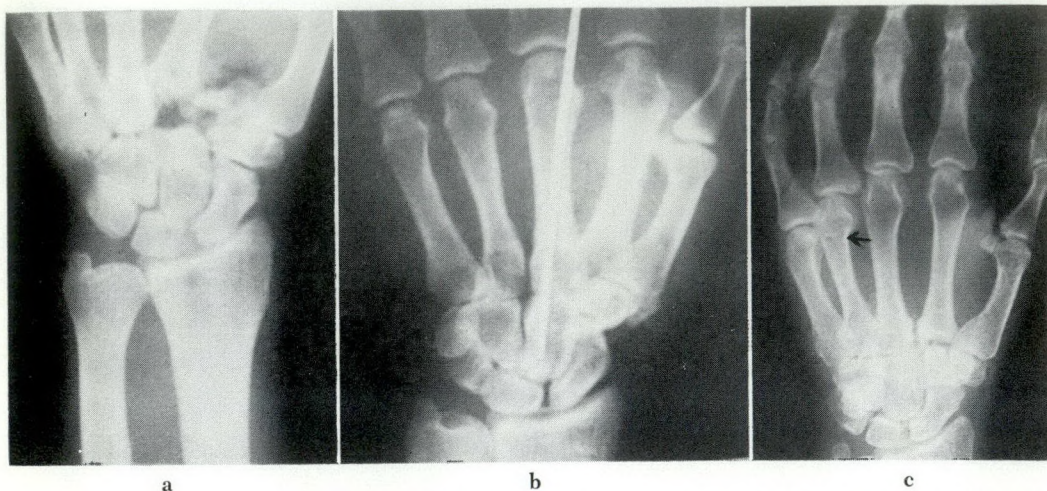
Fig. 5.–Case 4. (a) Open palmar dislocation of the second and dorsal dislocation of the third and fourth metacarpal bases. (b) Percutaneous fixation after primary arthrodesis of the second carpometacarpal joint and closed reduction of the other affected joints. (c) Eighteen months later with a new fracture of the neck of the fourth metacarpal.

a garbage truck. He sustained lacerations of the dorsum of the hand and base of the right thumb, volar dislocation and fracture of the base of the second metacarpal, and dorsal dislocation of the bases of the third and fourth metacarpals. He also had associated fractures of the trapezoid and the lower shaft of the radius (Fig. 5a). The open wound was cleansed, the articular cartilage removed and the base of the second metacarpal was fused to the trapezoid. A wire was passed through the base of the third metacarpal and into the capitate to maintain the reduction (Fig. 5b). The man returned to work in four months and had few complaints. Eighteen months later, he was seen because of a fracture of the neck of the fourth metacarpal of the same hand. At this time a radiograph showed that the metacarpal bases were in good position but the fusion between the second metacarpal and the trapezoid was not solid. He had no symptoms related to his original injury (Fig. 5c).

Case 5.—A 33-year-old man, while inebriated, fell down a flight of stairs and injured the bases of the metacarpals of his right hand. The radiographs showed that he had complete dorsal dislocations of the bases of the second, third and fourth metacarpals (Fig. 6). After closed reduction and plaster fixation, satisfactory function was restored.

Case 6.—In a motorcycle accident in England in 1958, a 20-year-old man had sustained dorsal dislocation of the bases of his right sec-

ond, third, fourth and fifth metacarpals. Closed reduction and plaster fixation for six weeks had given an initial satisfactory result. (This man may be Case 4, in Shephard and Solomon's report.[4]) Four years after injury, however, he developed a painful swelling on the dorsum of the same hand and associated weakness of the grip. There was slight bony prominence at the bases of the second, third and fourth metacarpals, and evidence of residual
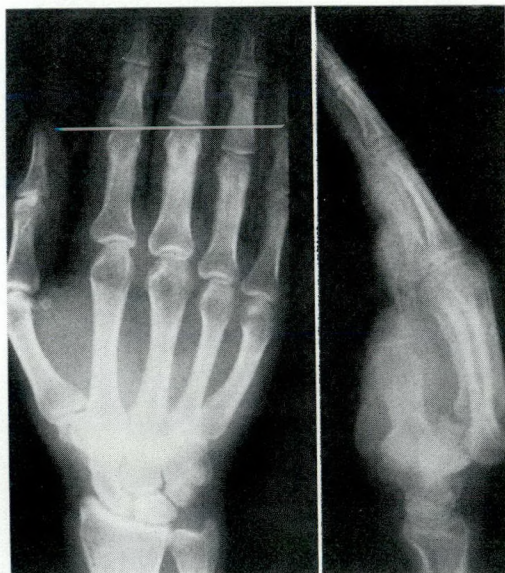
Fig. 6.–Case 5. Two views of dorsal dislocation of the bases of the second, third and fourth metacarpals without any apparent fracture.

a   b   c
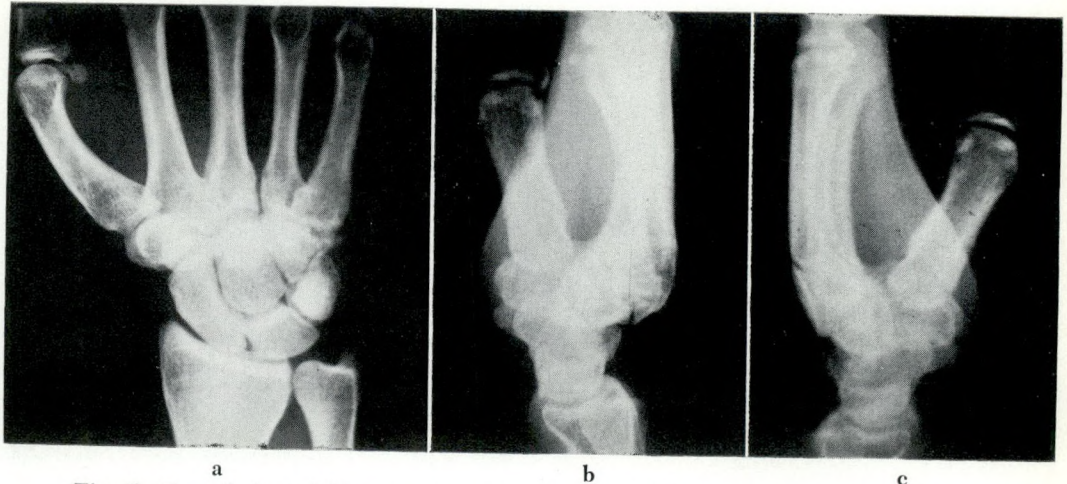
Fig. 7.—Case 6. (a and b) Two views of a four-year-old dorsal carpometacarpal dislocation with a bony prominence and joint space changes. (c) Lateral view four months after excision of the prominence and iliac-crest grafting.

subluxation and joint space narrowing on the radiographs (Fig. 7a and b). At operation, the prominent bases of the metacarpals were resected and the second, third and fourth carpometacarpal joints were fused using an iliac-crest graft. Four months later he had a strong grip and only 5 to 10° limitation of palmar and dorsal flexion in that wrist (Fig. 7c).

Case 7.—A 22-year-old workman was struck on the back of the right hand by an automobile jack and fractured the shaft of the fourth metacarpal, dislocated the base of the fifth metacarpal dorsally, and fractured the dorsum of the hamate (Fig. 8). Closed reduction of the dislocation and the fracture was difficult but was finally obtained. A wire was passed across the bases of the fifth, fourth and third metacarpals to hold the dislocation, following which the fracture of the fourth metacarpal was manipulated (Fig. 9). The cast and wire were removed at six weeks. The end result was satisfactory.

Fig. 9.—Case 7. After closed reduction and transverse percutaneous wire fixation of the ulnar three metacarpal bases.

Fig. 8.—Case 7. Fracture of the shaft of the fourth metacarpal. On the lateral view note the dislocation of the base of the fifth metacarpal and fracture of the dorsum of the hamate.

Case 8.—A 25-year-old barber fractured and dislocated the bases of the right fourth and fifth metacarpals (Fig. 10). These were treated satisfactorily by closed reduction and plaster fixation for six weeks. The end result was satisfactory (Fig. 11).
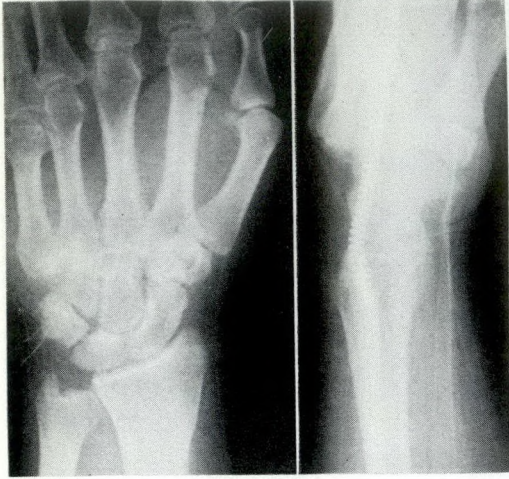
Fig. 10.—Case 8. Fracture-dislocations of the bases of the fourth and fifth metacarpals with the hand in an air splint.

Fig. 11.—Case 8. Satisfactory reduction and progress of healing six weeks after injury to the fourth and fifth metacarpal joints.

Case 9.—While playing ball, a 9-year-old boy struck his left wrist on some boards and sustained a fracture-subluxation of the base of the second metacarpal. This was treated by closed reduction and the end result was satisfactory (Fig. 12).

Case 10.—In an automobile accident, a 19-year-old girl sustained multiple injuries including a fracture of the base of the fifth metacarpal with dorsal subluxation of the bases of the second, third and fourth metacarpals (Fig. 13). Four weeks after injury, open reduction was done and the bases of the second and third metacarpals were fixed to the carpus with wire to obtain good reduction (Figs. 14 and

15). On follow-up by the referring doctor, she had no residual bony deformity and no complaints.

Case 11.—A 16-year-old boy, the driver of a motorcycle, was involved in a collision. In addition to other major injuries, he had a volar fracture-dislocation of the base of the second metacarpal and a dorsal displacement of the base of the third metacarpal (Fig. 16). When his open wounds were treated, a short hooked rod was passed through the base of the index

Fig. 12.—Case 9. Fracture-subluxation of the base of the second metacarpal in a 9-year-old boy.
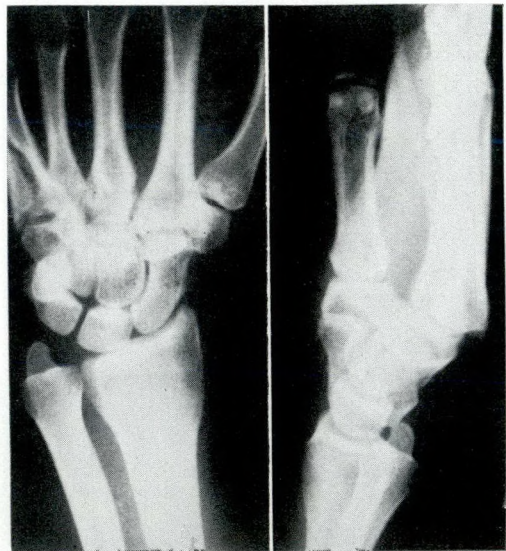
Fig. 13.—Case 10. Oblique fracture of the base of the fifth metacarpal with dorsal subluxation of the bases of the second, third and fourth metacarpals.
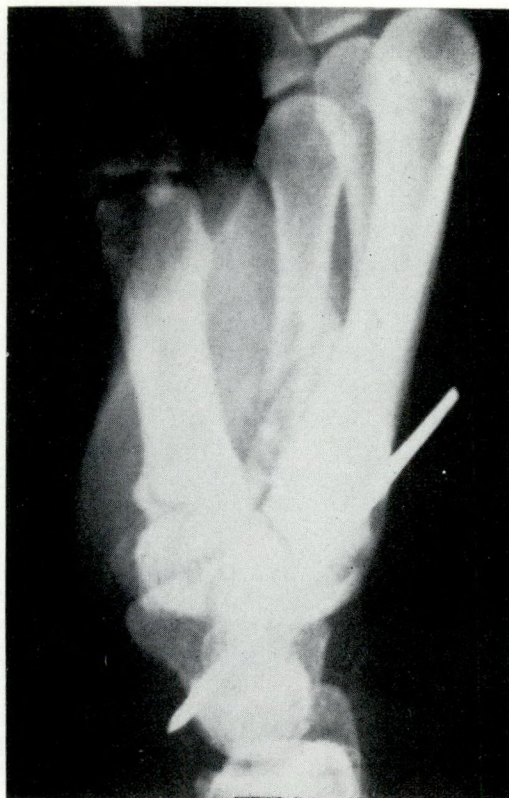
Fig. 14.—Case 10. Lateral view of the hand at the time of open reduction with two-wire fixation.

Fig. 15.—Case 10. Later view of the hand in a plaster cast shows satisfactory reduction.

finger was eventually amputated. One year after injury, the boy still had an "aching" pain at the base of the second metacarpal, and prominence and soreness of the base of the third metacarpal.

DISCUSSION

An analysis of this series suggests that these injuries are most common in the right

metacarpal into the adjacent carpal bone (Fig. 16). Residual posterior subluxation of the base of the third metacarpal remained. The index

a   b   c
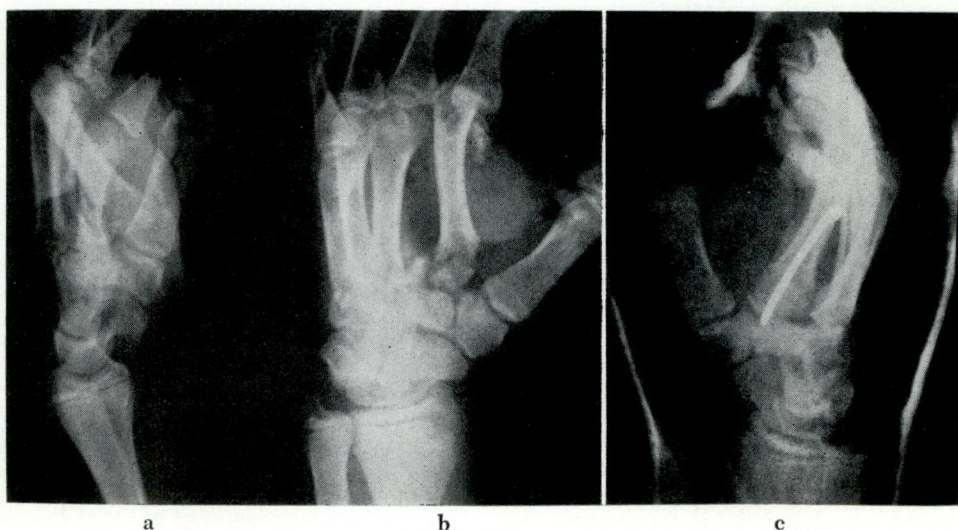
Fig. 16.—Case 11. (a) Lateral and (b) oblique views showing open palmar dislocation of the base of the second metacarpal with a dorsal fracture-dislocation of the base of the third metacarpal. (c) Oblique view after open reduction and insertion of a short hooked rod into the second metacarpal.

hand of young adult men. The subluxation or dislocation of the metacarpal bases is is usually posterior and the most common mechanism of injury seems to be a blow to the metacarpophalangeal joint, the head of the metacarpals or the dorsum of the hand, probably with the wrist in palmar flexion (Cases 1-3, 5 and 7). The palmar pressure exerted by the motorcycle handlebar (Cases 6 and 11) was not common in this series.

Two distinct patterns of injury involved the bases of the metacarpals on either the radial or ulnar sides of the hand. Only one patient had injury to all four metacarpal bases. Each individual metacarpal base was involved 6 times in the 11 patients.

It is of interest to compare the distribution of carpometacarpal joint injuries in these 11 patients with the cases discussed by Waugh and Yancey.[3] They found that the bases of the ulnar four metacarpals were most commonly involved; the base of the fifth alone, and of the fourth and fifth together or second and third together were the next most common types. In the present series, in only one patient were the ulnar four metacarpals injured, in three only the fifth and in one each the fourth and fifth together, and second and third together. The relatively uncommon pattern —involvement of the bases of the fourth, third and second metacarpals—was seen in three patients in this series.

In the two patients who had open wounds (Cases 4 and 11), the second metacarpal base dislocated forwards and others backwards, probably indicating a twisting injury.

Associated fractures included that of the dorsum of the hamate, crush fractures of the trapezoid and capitate, and associated fractures of the metacarpals, especially the bases of the second or third, and the shaft of the fourth or fifth.

The injury is best demonstrated in true lateral radiographs of the hand. Closed reduction is usually successful and the bones can often be held in position with a plaster cast moulded closely against the dorsum of the hand at the bases of the metacarpals. Instability or recurrent subluxation can be prevented by transverse wire fixation through the metacarpal bases.

Longitudinal wire fixation was used in the two patients with associated open wounds and the one in whom late reduction was done by open operation. Open reduction may be necessary to obtain a satisfactory positioning if the true nature of the injury is not realized at once or closed reduction is not done in the first few weeks.

Both patients with residual subluxation of a metacarpal base (Cases 6 and 11) have had bony deformity, associated weakness of grip and tenderness. These subluxations should be reduced completely as early as possible. Residual deformity and subluxation (Case 6) can be corrected by excision of the bony prominence and local arthrodesis. This gives comfort and a strong grip with only slight loss of wrist motion.

SUMMARY

Eleven patients with carpometacarpal dislocation other than the thumb are described. These injuries are seen chiefly in the right hand of young adult men and are due to a variety of mechanisms which affect chiefly the ulnar or the radial metacarpals.

Early diagnosis and closed reduction, with or without percutaneous wire fixation, are advised because failure to obtain complete reduction often leaves the patient with a weak grip, tender bony prominence and a persistent "aching" pain over the fracture site. If treatment is delayed, open reduction with skeletal fixation may be necessary. Old unreduced subluxations can be treated by partial excision of the metacarpal base and carpometacarpal fusion.

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RÉSUMÉ

L'auteur présente 11 cas de luxation carpométacarpienne intéressant d'autres doigts que le pouce. Ces blessures se voient surtout à la main droite de jeunes adultes du sexe masculin et dépendent de mécanismes divers qui affectent principalement les métacarpes cubitaux et radiaux.

On conseille de poser un diagnostic précoce et de pratiquer une réduction fermée, avec ou sans fixation percutanée d'une suture, parce qu'une réduction incomplète laisse souvent le malade avec une force de préhension diminuée, une proéminence osseuse hypersensible et un endolorissement au niveau du siège de la fracture. Si le traitement est retardé, il peut être nécessaire de pratiquer une réduction ouverte avec fixation au squelette. D'anciennes subluxations qui n'ont pas été réduites peuvent être traitées par excision partielle de la base du métacarpe et arthrodèse carpométacarpienne.

CHEST WALL TUMOURS

Tumours of the thoracic skeleton are uncommon and can present difficult problems in diagnosis and management, particularly in the performing of adequate radical excision and subsequent reconstruction of the chest wall. The wide variety of cell types in chest wall tumours makes classification of these lesions difficult.

In one description of the malignant chest wall tumours, these lesions are divided into those that are radiosensitive and those that are radioresistant. The radioresistant tumours include chondrosarcoma, osteogenic sarcoma, neurosarcoma, fibrosarcoma, liposarcoma, angiosarcoma, and anaplastic sarcoma. Radiosensitive malignant tumours include Ewing's sarcoma, reticulum cell sarcoma, plasma cell myeloma, tumours in Hodgkin's disease, and lymphosarcoma. A collective review of benign, malignant, and metastatic tumours of the chest, as well as a brief review of sternal, clavicular, and scapular tumours is presented by the authors.

The patient with a chest wall tumour usually presents himself to the physician with a mass in the chest wall which may or may not be painful. In view of the high rate of malignancy in chest wall tumours, histologic diagnosis should be obtained in all instances.

The preferred method of treatment for benign and malignant nonradiosensitive tumours of the chest wall is surgical excision. Ewing's sarcoma, reticulum cell sarcoma, myeloma, granulomas of Hodgkin's disease, and lymphosarcoma fall into an intermediate group and may be treated by either resection or irradiation.

If the treatment of the tumour is to be surgical excision, the surgeon should not hesitate to resect a wide margin of chest wall surrounding the tumour. A large portion of the chest wall may be removed and satisfactory reconstruction done with little compromise of pulmonary function. The chest wall resection should include at least 5 cm. of rib on either side of the tumour and, if malignancy is suspected, large segments of normal ribs above and below the tumour. Resection should include pleura, intercostal muscles, and neurovascular bundles. If there is fixation to the overlying chest wall muscles or skin, these also should be resected *en bloc*. Involved areas of lung may be resected, removing a segment or lobe as necessary. Portions or all of the sternum may be resected, including the medial aspects of the clavicles if necessary. Resection of upper ribs for a chest wall tumour may be accomplished by dividing or resecting a portion of clavicle and freeing the tumour from the subclavian vessels and brachial plexus using an anterior approach; some tumours may be approached posteriorly as in a thoracoplasty.

Reconstruction of the chest wall must provide enough rigidity in the area of resection so that intolerable paradoxic motion does not occur. Resections of the anterior chest wall result in more paradoxic motion and respiratory embarrassment than do excisions of the lateral and posterior chest, where the scapula and vertebral column increase stability. Tracheostomy and positive pressure ventilation through a cuffed tube will support pulmonary function when it may be compromised during the early postoperative period. The use of pedicle grafts of skin and chest wall musculature and the use of prosthetic materials such as stainless steel mesh, Marlex mesh, stainless steel struts, and malleable plastic, which can be hardened at the operating table, are all being used successfully in chest wall reconstruction.

Fear of not being able to reconstruct the chest wall should not lead the surgeon to perform an inadequate radical excision of the primary tumour. Careful planning and combinations of various methods and techniques should enable the resourceful surgeon to successfully reconstruct large defects of the chest wall.—Groff, D. B., III and Adkins, P. C.: Chest wall tumors, *Ann. Thorac. Surg.*, **3:** 260, 1967.

ARTHROGRAPHY OF THE KNEE

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ARTHROGRAPHY of the knee was introduced in Europe at the beginning of the twentieth century. In 1948, Lindblom[1] published a definitive account of single positive contrast arthrography. He used only a single positive contrast technique introducing 35% Perabrodil into the joint. In 1951, van de Berg and Crevecoeur[2] described a double contrast technique that has since been modified by others.[3] In this procedure, both a positive contrast substance and air are introduced into the joint. Techniques involving the use of gas[4] or vacuum have been tried but are not now popular.

This paper will describe the results obtained in 452 single positive contrast arthrograms done in 433 patients at St. Joseph's Hospital, Hamilton, Ontario and Joseph Brant Memorial Hospital, Burlington, Ontario, from 1959 to 1966. The patients, 348 males and 85 females, ranged in age from 7 to 66 years. In these patients, arthrography was used primarily to assist in the diagnosis of meniscal lesions. Occasionally, cruciate ligament tears, cartilagenous loose bodies, polypoid tumours and local articular cartilage thinning were also demonstrated. The interpetation of these arthrograms will be compared with the findings at operation and on follow-up examination.

Arthrography was done on both knees in six patients, twice on the same knee in 12 patients and three times on the same knee in one patient. Each examination was assessed separately. The right knee was examined 244 times and the left 208 times.

TECHNIQUE

After an initial development period, this examination was done uniformly throughout the series. A radiograph was made in the lateral projection to assure satisfactory exposure of the joint space and to measure the tilt of the tibial plateau on the tibial shaft. This tilt, which is backward and downward and is usually 6 to 12°, must be taken into account if good "shoot-through"

films are to be made of the joint space. After the site of injection has been infiltrated with local anesthetic, 10 c.c. of aqueous iodine salt, usually 50% sodium diatrizoate, is injected into the joint using aseptic technique. If there is any joint effusion, it is aspirated completely to prevent dilution of the dye. Care is taken to avoid introducing air into the joint. An elasticized bandage is applied to occlude the suprapatellar pouch. The knee is then manipulated through its full range of motion and is rocked and rotated in both flexion and extension. The patient may have pain lasting up to one minute after this examination, usually over the area of clinical symptoms.
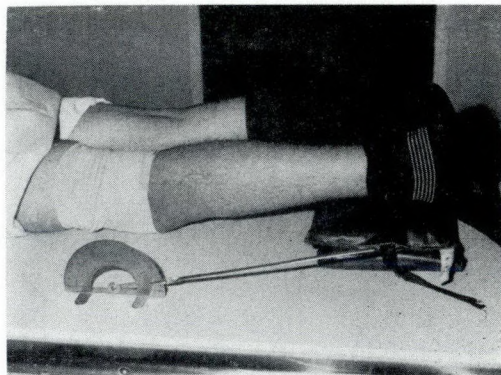
Fig. 1.—On a PA projection with the x-ray tube vertical, the foot is elevated on a sand-bag, 6 to 12° as measured by the protractor to compensate for the tilt of the tibial plateau.

After a single anteroposterior film and stereo lateral films are made, the rest of the views are taken in the stereo posteroanterior (PA) projection under the radiologist's direction (Fig. 1). With the x-ray beam vertical, the patient's foot is elevated until the lower leg is at an angle to the cassette equal to the angle of tilt of the tibial plateau. This angle is halved for rotational or oblique views. With the leg in this position, the rest of the views are taken in the stereo PA projection in the following order: (a) direct PA, (b) internal rotation 45°, (c) external rotation 45° and (d) intercondylar with the knee flexed 80°.

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To show a particular area of the meniscus, additional films in any degree of rotation may be made. All films should be completed within 10 minutes of the injection to ensure sharp detail. After this time, the dye is absorbed into the surrounding synovium and is diluted in the joint.

INTERPRETATION OF ARTHROGRAMS

Interpretation of arthrography requires a knowledge of the normal gross and radiological anatomy of the knee. The physician must have personal interest in and experience with this technique if his interpretations are to be reliable.

The shape and width of the menisci in the radiographs should be studied thoroughly. Lindblom[1] gives the average width as follows: for the medial meniscus—6 mm. for the anteromedial area, 6 mm. for the mid-portion, and 14 mm. for the posterior horn; and for the lateral meniscus— 10 mm. anteriorly, 10 mm. mid-laterally and 9 mm. for the posterior horn. The meniscal outline should be sharp and smooth. A meniscus narrow in one view but not narrow throughout is probably abnormal. After the meniscus is removed, it may regenerate as a blunter wedge. Tears in retained fragments (Fig. 2) or tears in a regenerated meniscus are readily demonstrated by arthrography.

The meniscus is attached to the capsule throughout its entire circumference except posterolaterally where the popliteus tendon separates it from the capsule for an average distance of 13 mm. There is a dispute about the presence of a bursa[5] between the superficial and deep portions of the medial collateral ligament in the normal knee. Regardless of this bursa, the presence of dye in this location indicates that the deep portion of the medial collateral ligament has ruptured. Normal sulci at the capsular attachment of the meniscus are shallow (1 to 2 mm.) and stereo views will show that the dye is outside the meniscus. A deep or complete line of dye between the meniscus and the capsule indicates a detachment at the periphery (Fig. 3).
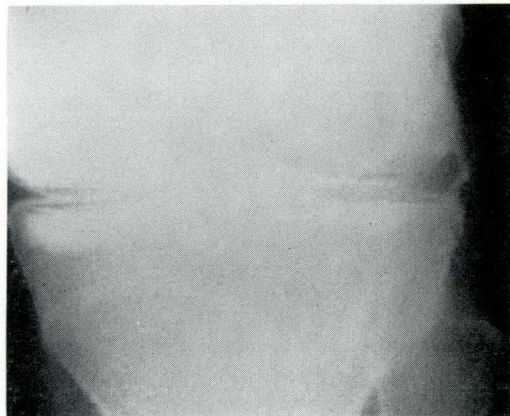
Fig. 3.—The PA view shows a peripheral detachment of the lateral meniscus.

In 32, or 7%, of the knees examined in this series, the semimembranosis-gastrocnemius bursa communicated with the knee joint (Fig. 4). In 20% of the knees examined, the space about the popliteus tendon communicated with the superior tibiofibular joint (Fig. 4). On arthrography, there was a discoid meniscus on the lateral side in eight patients (Fig. 5) and on the medial side in one. A cyst or ganglion of the meniscus was rarely demonstrated.

In this series, one patient had an interarticular polypoid soft-tissue tumour and another a cartilagenous loose body which were not seen on the regular films. In nine patients the films suggested fat-pad hypertrophy.

Horizontal and vertical tears in menisci stand out clearly on arthrography. Transverse tears, however, are difficult to recognize. Films taken from the intercondylar

Fig. 2.—One year after meniscectomy, a 45° external oblique view shows retained posterior horn of the medial meniscus with a vertical tear.
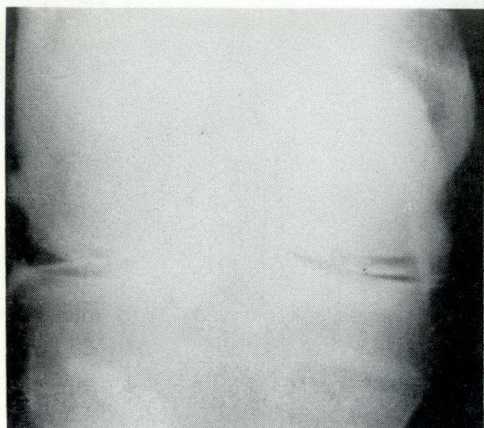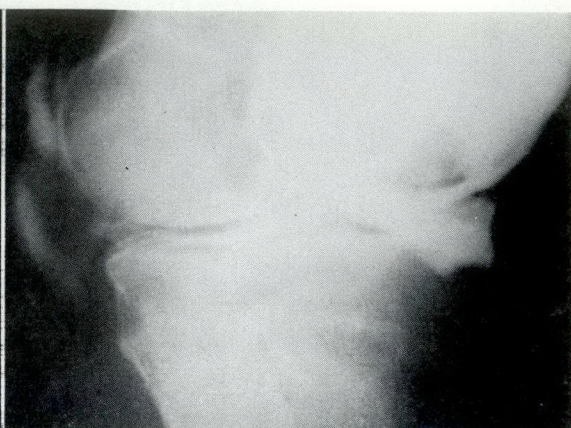
Fig. 4a   Fig. 4b

Fig. 4.—On the PA view (a) the filled semimembranosis-gastrocnemius bursa obscures the tear in the medial meniscus. This tear is seen clearly in the external rotation view (b). The popliteus bursa communicates with the superior tibiofibular joint.

projection best demonstrate these, because flexion of the knee tends to separate the margins of the tear. Fine degenerative meniscal tears or tears within the meniscal substance may not fill with dye. "Bucket-handle" tears may be missed if the width of the cartilagenous wedge is not considered because the displaced fragment is rarely outlined well.

FINDINGS

In 11 of 452 arthrograms (2.5%), the examination was poor and a reasonable interpretation was impossible; these films were excluded. This left for study 441 arthrograms in 422 patients.

Subsequently, 241 arthrotomies were done

in this group of patients and the following procedures were carried out: 225 meniscectomies, 8 loose bodies removed, 4 synovectomies, 1 anterior cruciate ligament repair, and 3 osteochondritis dissecans fragments drilled and curetted. Meniscectomies were done in 191 males (85%) and 34 females (15%). Lateral meniscectomy (17% of all meniscectomies) was relatively more common in females (35%) than in males (14%).

The interpretation of the arthrogram was confirmed at operation in 91% of 241 arthrotomies (Table I). In 169 arthrotomies (70%), a tear of the meniscus indicated by arthrography was found at operation. In 24 arthrotomies (10%), the

Fig. 5.—A discoid lateral meniscus is demonstrated on the internal rotation view.

TABLE I.—DIAGNOSIS MADE AT ARTHROGRAPHY COMPARED WITH THAT MADE AFTER EXPLORATION IN 241 PATIENTS

Arthrogram	Arthrotomies	Percentage
"Positive for tear" and confirmed..........	169	70
"Negative for tear" and confirmed..........	24	10
"Suspicious for tear" and confirmed.......	26	11
	219	91
"Positive for tear"— none found..........	1	0.4
"Negative for tear"— tear found..........	21	8.6
	22	9.0
Total..........	241	100.0

meniscus was normal at operation and on arthrogram. In an additional 26 arthrotomies (11%), a meniscal tear found at operation had only been suggested by arthrography.

Interpretation of 22 arthrograms was proved to be incorrect at arthrotomy (Table I). From one patient in whom the arthrogram suggested a tear, an intact medial meniscus was removed. This patient had extensive synovitis, which on biopsy proved to be due to rheumatoid arthritis. In 21 arthrotomies, a meniscal tear was found that had been missed on arthrography. Twelve men over 40 years of age had degenerative tears of the posterior medial meniscus that were not delineated at arthrography. Four lateral meniscal tears were also missed.

Of the 200 patients who were not operated upon, 49 could not be located for follow-up. The remaining 151 were contacted, and the diagnosis based upon the interpretation of the arthrograms was compared with that suggested by the patient's continuing symptoms (Table II). The interpretation of the arthrogram conformed to the symptoms in 97 patients (64%) and did not conform in 54 patients (36%).

DISCUSSION

None of these 433 patients had significant complications following single positive contrast arthrography. None developed skin

TABLE II.—DIAGNOSIS ON ARTHROGRAPHY COMPARED WITH THAT SUGGESTED BY SYMPTOMS AT FOLLOW-UP IN 151 PATIENTS IN WHOM NO EXPLORATION WAS DONE

	Patients	Percentage
Symptoms supporting arthrogram		
Arthrogram positive—symptoms unchanged.	20	
Arthrogram negative—symptoms improved..	77	
	97	64
Symptoms not supporting arthrogram		
Arthrogram positive—symptoms improved..	15	
Arthrogram negative—symptoms unchanged.	39	
	54	36
Total............	151	100

or joint sepsis. There was no prolonged synovial irritation as a result of the injection of the contrast medium. As Lindblom[1] pointed out, "in comparison to the consequences of exploratory arthrotomies, irritation by the salt solution arthrography is negligible". Even after extrasynovial injection of dye, there were no sequellae.

A satisfactory arthrogram can only be obtained if the following basic details of technique are carefully observed: (a) Joint fluid must be completely removed. (b) Air bubbles must be carefully excluded. (c) Following the injection the joint must be vigorously manipulated to spread the dye. (d) Radiological exposure must be correct to ensure proper contrast in the films. (e) Correct projections must be made through the joint space. (f) All films must be made within 10 minutes of dye injection.

Clinically, the diagnosis of a gross traumatic meniscal tear is easy especially if the knee is locked. If required, examination under general anesthesia will demonstrate significant ligamentous tears without arthrography although accompanying meniscal lesions can be verified by this procedure.

In the 433 patients in this series, the indications[6,7] for arthrography were: (a) a suggestive history but only minimal signs on clinical examination; (b) in those in whom exact diagnosis was essential soon after injury; (c) in those with industrial injuries, or those in whom litigation was pending; (d) in the very young or very old, where accurate diagnosis is more difficult; (e) in those who had previous knee operations; and (f) to allay the patient's anxiety about significant intra-articular pathology.

Each patient in this series was treated on the basis of the clinical findings. If the patient's symptoms persisted despite a normal arthrogram, the knee was explored.

According to Smillie,[8] his error in the clinical diagnosis of meniscal tears is only 4%. This is exceptional. With single positive contrast arthrography, the correct clinical diagnosis was made in 91% of these patients; this figure is much better than that obtained on clinical examination alone. There is no suggestion from this review that the diagnosis of lateral meniscal tears is less accurate than that of medial tears.

SUMMARY

The results obtained in 452 single positive contrast arthrograms in 433 patients have been presented. The radiological interpretation of the films so obtained was compared with the findings at operation, and with the persistent symptoms elicited at follow-up in those who were not operated upon. The result of this review suggests that arthrography of the knee is helpful and reliable in the recognition of meniscal lesions.

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RÉSUMÉ

L'auteur présente les résultats de 452 arthrogrammes opaques simples effectués chez 433 malades. Il a comparé l'interprétation des films aux constatations opératoires et, dans le cas des malades non opérés, aux symptômes persistants observés durant la période de catamnèse. Il ressort de cette revue que l'arthrographie du genou est une méthode précieuse et sûre pour le diagnostic des lésions des ménisques.

RETROPERITONEAL HEMORRHAGE

Retroperitoneal injury and hemorrhage are often diagnostic and therapeutic problems. Sixty-five patients are described who had retroperitoneal hemorrhage as determined by autopsy, surgical treatment, radiological examination or the finding of a palpable mass. They were selected from 285 patients hospitalized between 1947 and 1966 because of blunt abdominal trauma.

Twenty-six of 65 had associated intra-abdominal organ injuries; 48 had significant additional extra-abdominal trauma to the chest, head or extremities. The overall mortality rate for the 65 patients was 38%. Because of associated intra-peritoneal or extra-abdominal trauma, it was often difficult to determine the exact cause of death. In nine patients, however, death was believed to be a result of, or precipitated by, the retroperitoneal hemorrhage. Those who died from retroperitoneal hemorrhage alone, had multiple retroperitoneal organ injuries.

Factors of diagnostic significance included tenderness, shock, the pressure of a mass, ileus, level of serum amylase, and results of urinalysis, blood counts, and roentgenograms. The last were the most accurate, providing diagnostic data on the site of injury and source of hemorrhage in 41 of 65 patients. Examinations included abdominal films, cystograms, and intravenous pyelograms.

Thirty-nine patients without associated intra-abdominal injuries, were operated upon with a 43% mortality rate. The 26 patients who were managed without operation had a 14% mortality rate. Only patients whose condition was deteriorating and who had rapid bleeding were operated upon which accounts for the high mortality in this group.

The authors recommend early, close monitoring of patients with retroperitoneal hemorrhage, and a high index of suspicion for the lesion, particularly in patients who show signs and symptoms of hemorrhagic shock without an obvious bleeding source. Only in this manner will progressive blood loss be detected and patients selected more judiciously for further diagnostic appraisal. Surgical intervention is indicated only in that minority of patients in whom nonoperative management has failed to halt deterioration.—Nick, W. V., Zollinger, R. W. and Pace, W. G.: Retroperitoneal hemorrhage after blunt abdominal trauma, *J. Trauma*, 7: 652, 1967.

CLOSED INTRAMEDULLARY NAILING OF FRACTURES

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DURING the past three years at St. Joseph's Hospital, Toronto, 30 fractures at various sites have been treated by the closed intramedullary nailing technique described by Küntscher. Seventeen of these fractures were treated by the author and the remainder by the other orthopedic surgeons at this institution. Because this method of fracture fixation has distinct advantages and pitfalls, our experience is described and discussed.

Professor Gerhard Küntscher of Hamburg refined the technique of intramedullary nailing. His treatise, "The Practice of Intramedullary Nailing",[1] which is now available in English, is the standard reference text on this procedure. In it, he outlines in detail the technique of closed intramedullary nailing. A guide wire is inserted in the medullary canal and reamers with flexible shafts are passed over it to enlarge the canal enough to take large "cloverleaf" nails. Earlier reports of the use of intramedullary implants for fixation (Nicolaysen in 1897, Delbet in 1906, Lambotte in 1907 and Hey Groves in 1916) were based on an entirely different concept, "intramedullary splinting" or "intramedullary pinning", which is quite distinct from the stable osteosynthesis obtained by intramedullary nailing.

TECHNIQUE

The patients selected for treatment using this technique are those in whom both fracture fragments are of sufficient length to allow firm fixation. The technique of operation, clearly described by Küntscher, will not be repeated here. However, certain points are worth emphasizing. In fresh fractures, a preliminary period of skeletal traction to maintain limb length is extremely important. Preoperative determination of the required nail size is made by measuring a standard radiograph taken

with the x-ray tube at one metre distance from the limb. To compensate for magnification, a special measuring device is used (Fig. 1). Nail diameter is decided by measurement of the medullary size after reaming an estimated one-third to one-half of the cortex. Nail length is measured directly. The operation is performed on a traction table which allows the image intensifier to swing freely in a two-plane surveillance of the extremity (Fig. 2). After the fracture is reduced and the guide wire inserted, the medullary canal is reamed up to a suitable size and the nail is driven over the guide and across the fracture site to obtain rigid fixation. In fractures of the femur, the guide wire and nail are introduced through a short incision over the tip of the trochanter. Nails to fix tibial fractures are introduced through the patellar tendon, and the humerus is nailed from either the upper or lower end depending on the site of the fracture.

After the operation, patients are encouraged in active mobilization of the limb, and are allowed weight bearing within the limits of comfort. The special instruments required for nailing are illustrated in Fig. 3. All are necessary as well as the image intensifier and the special traction table. A good nail extractor is particularly important.

VASCULAR CONSIDERATIONS

Küntscher stoutly maintains that cortical bone is mainly nourished from without and cites his clinical experience with nailing as proof. In the past, this concept of a periosteal, centripetal arterialization of bone cortex has been popular. More recently, the microradiographic studies of Brookes[2, 3] indicate that normal diaphyseal arterial supply is centrifugal, that is, it originates in the medulla and passes outwards to the periosteum. Obviously, the extensive medullary reaming in Küntscher's technique must destroy the medullary vessels completely. However, this technique, as opposed to

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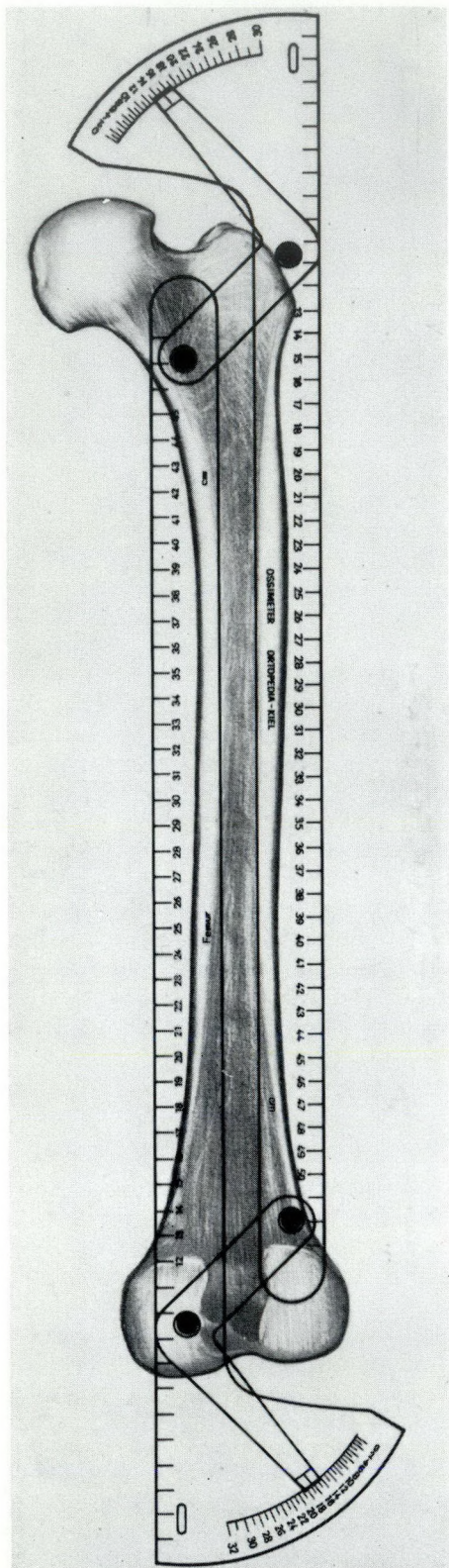
"open" nailing, has the advantage that it is carried out without surgical exposure at the fracture site and coincidental damage to the periosteal blood supply. Clinical experience indicates that fractures of long bones, which have progressed to non-union, heal particularly well after closed intramedullary fixation; perhaps, in these patients, the blood supply at the fracture site has shifted to a periosteal pattern.

However, despite the controversy based on theoretical and experimental considerations of blood supply at the fracture site, a fracture treated by rigid intramedullary fixation regularly heals.

BIOMECHANICAL CONSIDERATIONS

Because the diameter of the nail used after medullary reaming is 14 to 16 mm. in a femur and 9 to 11 mm. in a tibia, the nails do not bend or break. If he is to select patients properly for this procedure, the surgeon must understand the mechanism of fixation of the nail. The fragments are fixed by friction between the nail and bone caused by "interference fit". This may be longitudinal, e.g. a straight nail driven down a reamed medulla which is more or less tortuous (Fig. 4) or circumferential, e.g. the cross-sectional diameter of the nail is greater than that of the reamed medulla and the nail slot closes when the nail is driven. Compression of the slot—the "elastic impingement" of Küntscher—is of questionable importance. In experiments done at St. Joseph's Hospital, it took more than 100 lb. of radial compression to close the slot in any of the stainless-steel cloverleaf nails tested. For this reason, we believe that longitudinal interference fit is the prime mechanism contributing to fixation. Reaming of the medulla, by increasing the area of friction between nail and bone, enhances stability. Clinically, when these nails are removed, most of them have remained quite tight.

Fig. 1.—The appropriate nail size is determined before operation by measuring a standard radiograph taken with the x-ray tube at one metre from the limb. To compensate for magnification, this special measuring device is used. Nail diameter is decided by measuring the medullary cavity after reaming an estimated one-third to one-half of the cortex. Nail length is measured directly.
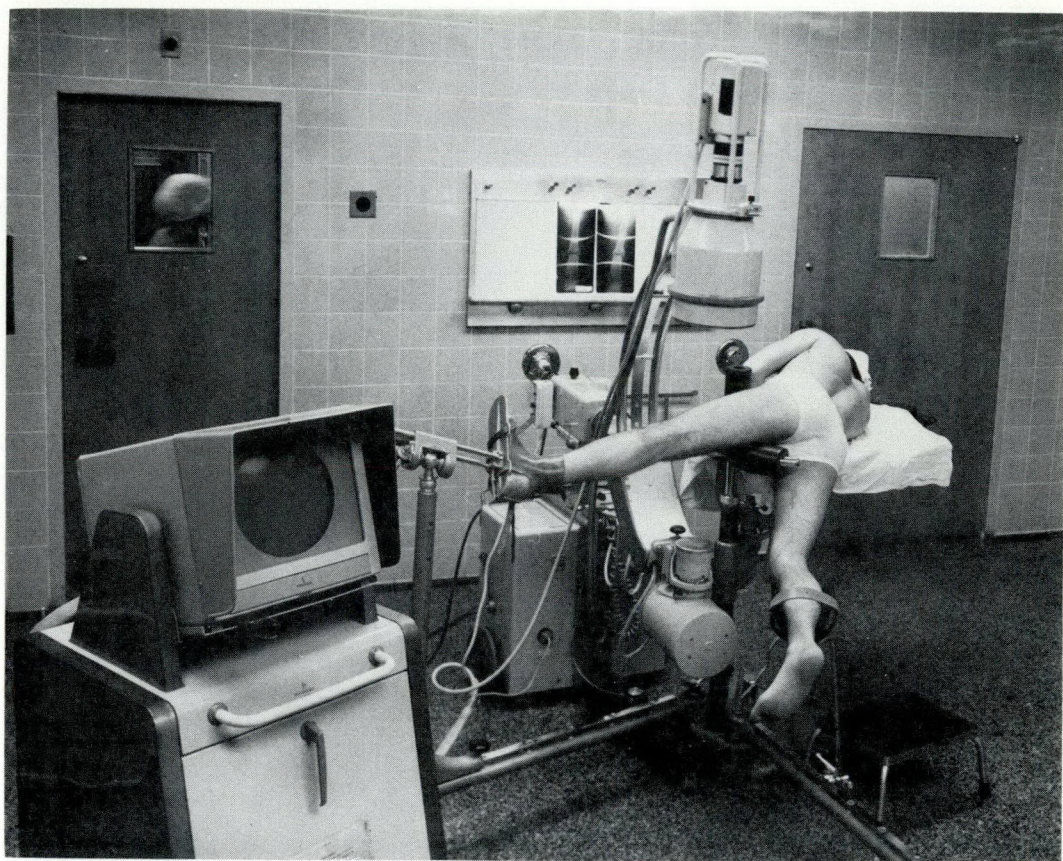
Fig. 2.—Closed intramedullary nailing is done under image intensifier surveillance. Here the "patient" is positioned for nailing of the left femur. The image intensifier can swing to give coverage in two planes.
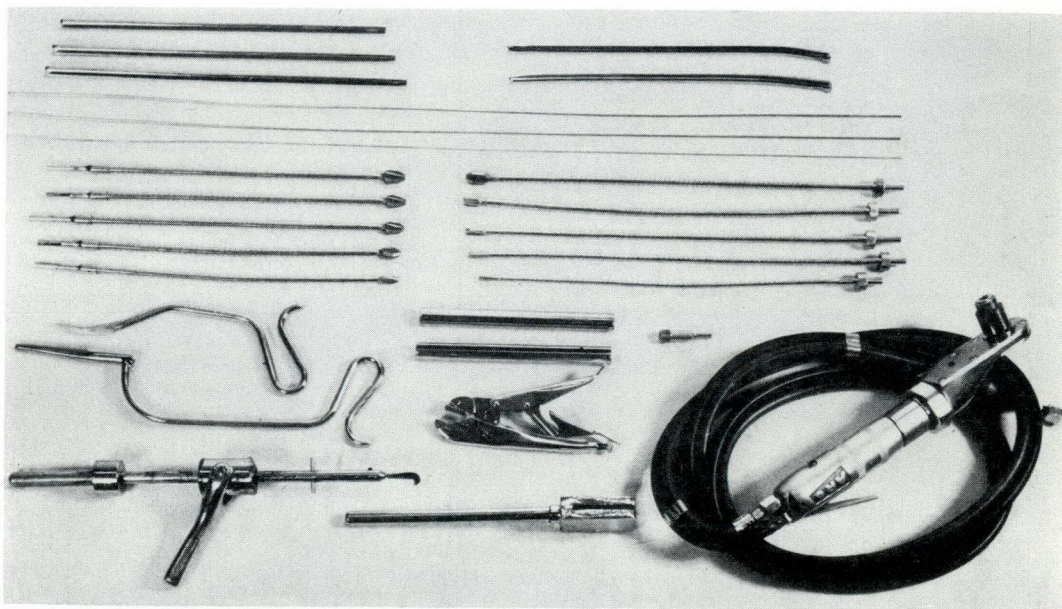
Fig. 3.—Instruments necessary for intramedullary nailing.
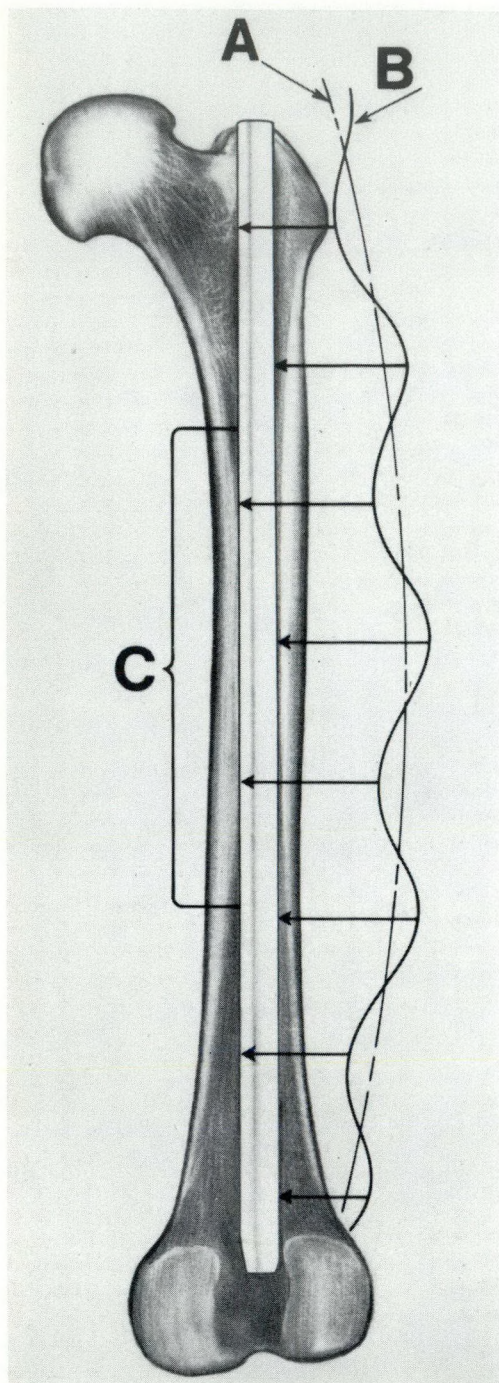
Fig. 4.—Line A—exaggerated contour of the medulla followed by the reamer in a typically undulating path (line B). Fixation is produced by longitudinal interference between the nail and the bone at the areas marked by arrows. The area of maximum nail/contour bone contact developed by reaming is inside area "C".

Results

The site and character of the fractures in the 30 patients described in this paper are shown in Table I. These patients varied

TABLE I.—Fractures Treated by the Kuntscher Method of Intramedullary Nailing

Site	Fresh fracture	Non-union	Pathological fracture	Mal-union
Femur.........	4	6	1 (osteomyelitis)	1
Tibia..........	1	3	—	—
Humerus.......	—	4	—	—
Radius and/or ulna.........	2	8	—	—
Total.......	7	21	1	1

in age from 16 to 60 years. Five were female. In most of the patients, the operation was "closed", that is, the fracture site was not exposed. On five occasions, the fracture site had to be exposed to drill out an obstructed medullary canal to allow passage of the guide wire. Typical examples of some of these fractures are shown in Figs. 5 to 8. In 28 patients, the results have been good. They were able to use the limb soon after operation and obtained all the advantages of early mobilization. There was bony union at the fracture site. In two patients, however, intramedullary nailing failed. In one, the proximal humeral fragment was too short and the cancellous bone too soft to hold the nail. Rigid fixation could not be obtained. The second failure was due to an error of technique—the cortex of the tibia was shattered when the nail was driven. The procedure was abandoned and the patient treated in plaster.

Discussion

Intramedullary nailing as described by Küntscher is effective in the treatment of fresh fractures and non-unions. This technique is most useful in femoral and tibial fractures. In humeral fractures, the technique is more difficult and less successful. These patients tend to develop stiff shoulders when the upper end of the nail protrudes into the rotator cuff. Closed nailing should not be done in forearm fractures because reduction is difficult and reaming almost impossible.
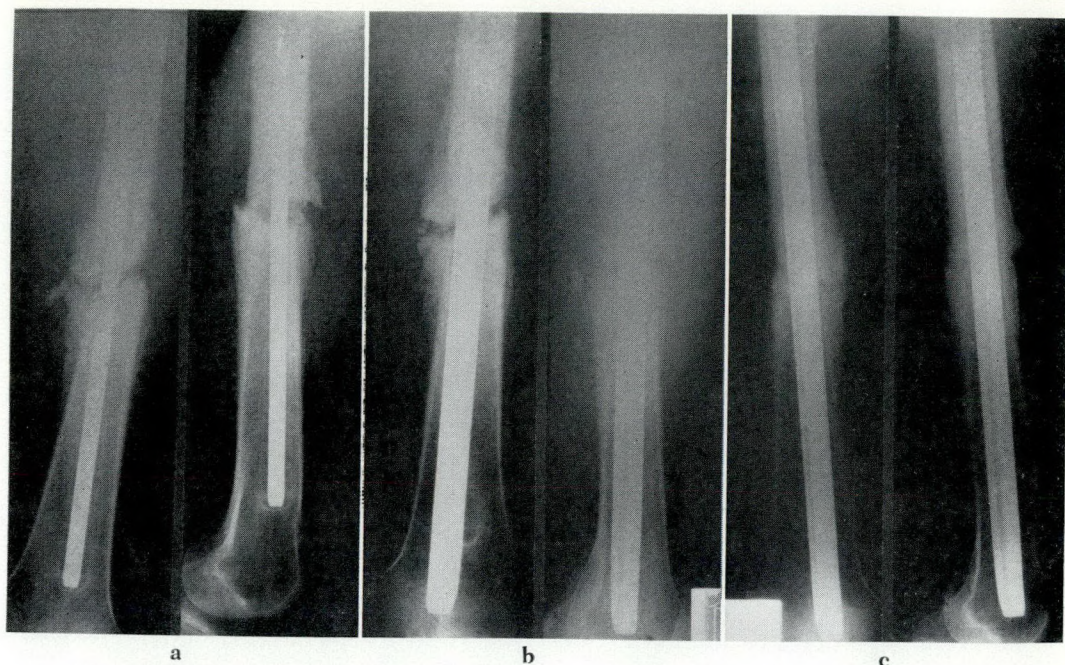
Fig. 5.—(a) Fracture of the femur treated by inadequate intramedullary pinning and ununited. (b) Treated by reaming and insertion of a large (16 mm.) nail. (c) Healed at six months.

In the patients in this series, various technical difficulties were encountered and successfully overcome. In one patient, the nail became impacted in the medullary canal because too large a nail was driven into an insufficiently reamed canal. In other patients, we had difficulty in reducing the fracture to allow passage of the guide wire, because insufficient traction was applied to the limb preoperatively. It is very
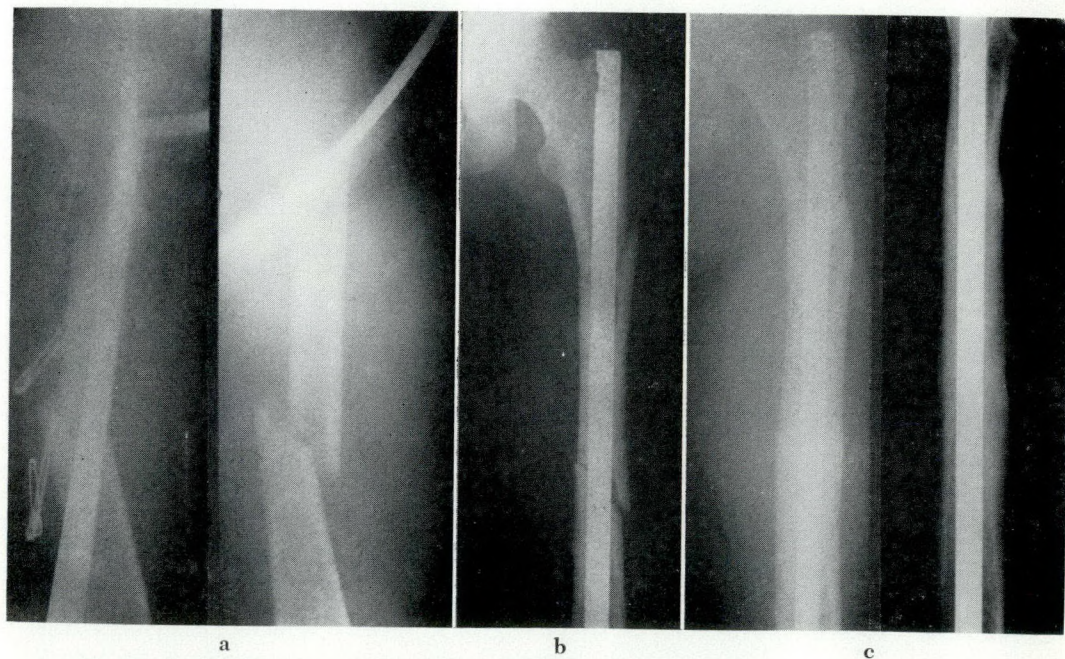
Fig. 6.—Double fracture of the femur. (a) In traction at the time of injury. (b) After closed nailing with a 15-mm. nail. (c) One year later at the time of nail removal. Full joint function.

a   b   c
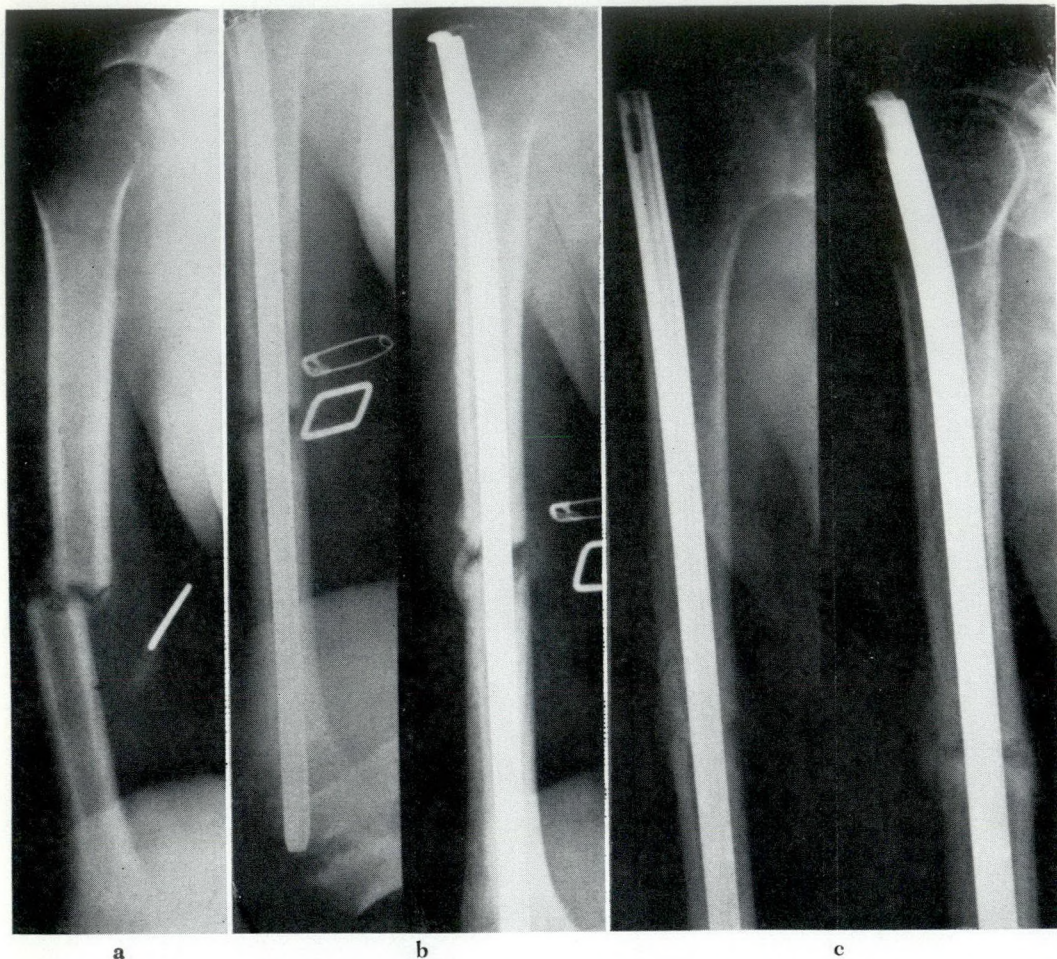
Fig. 7.—(a) Ununited fracture of the humerus at three months. (b) Treated by closed reaming and nailing, and healing progressing at two months. (c) The fracture has healed. Shoulder function was restored.
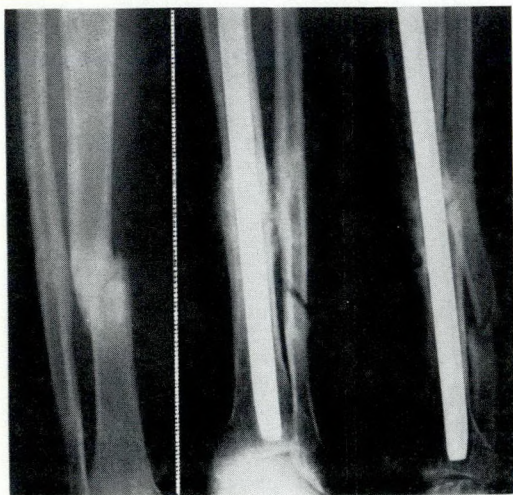
Fig. 8a   Fig. 8b

important to have the limb pulled out to length or even slightly over-pulled before any effort is made at intramedullary nailing.

The Küntscher technique requires a large number of special instruments. A good extractor is of particular importance. Nails that have a threaded end should be used because, with them, manipulation and final extraction are much easier.

The pattern of the blood supply at the fracture site is still a matter of controversy. In any event, clinical experience suggests

Fig. 8.—(a) An ununited fracture of the tibia of 10 years' duration. (b) Treated by open nailing and reaming to allow drilling of the medullary canal at the fracture and osteotomy of the fibula. Healing at six months.

that if the fixation is rigid, healing will follow. Closed intramedullary nailing has the great advantage that it does not require exposure of the fracture site, thus, periosteal stripping and infection at the fracture site are avoided. Reaming of the medullary canal allows the use of much larger nails which do not break. Reaming also gives increased area of nail/bone friction (interference fit) and thus enhances fixation.

Conclusions

Küntscher's intramedullary nailing technique is a superior method of fixing suitable fractures.

The technique is demanding. There are many opportunities for mishap. Instrument requirements are extensive.

Fractures of the femur and the tibia are the most suitable for this procedure. Fractures of the humerus are less suitable. Forearm fractures are not suitable for intramedullary nailing.

Non-unions of the mid-shaft are very well treated by this technique.

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Résumé

Nous avons traité 30 cas de fractures par la technique fermée de Küntscher, soit la mise en place d'un clou intramédullaire. Les résultats en ont été excellents: bonne cicatrisation et excellente récupération fonctionnelle. Pour appliquer ce traitement, nous avons choisi des malades chez lesquels les deux fragments de la fracture étaient suffisamment longs pour obtenir une fixation solide. Les fractures du tibia et du fémur sont celles qui conviennent le mieux à ce genre de traitement. Les cas de pseudarthrose peuvent être bien traités par cette méthode. Celle-ci exige une grande habileté et de très nombreux instruments. L'alésage du canal médullaire permet d'employer de gros clous qui ne risquent pas de se plier ou de casser. Cet alésage se traduit par un contact plus intime entre clou et cortex et, partant, par une fixation plus solide. Cette méthode étant pratiquée sans mettre a nu le siège de la fracture, autrement dit étant "fermée", évite la perte d'un lambeau de périoste et le risque d'infection.

CARCINOMA OF CERVIX

Thirty patients, 14 antepartum and 16 postpartum, had invasive carcinoma of the cervix between 1940 and 1965. Twenty-four patients were classified as having stage I lesions, 5 as having stage II lesions and 1 as having a stage III lesion. Fourteen patients were pregnant and 16 postpartum at the time of diagnosis. Of these 14, 3 were in the first trimester, 9 in the second, and 2 in the third trimester. Among the 16 patients in whom invasive carcinoma of the cervix was discovered post partum, lesions were discovered at delivery in 2; in 3, lesions were diagnosed at 6 weeks post partum; and in 7, lesions were found between 2 and 10 months post partum. Four patients were diagnosed after a spontaneous abortion. In the last five years of study all patients with invasive carcinoma of the cervix were diagnosed antepartum.

Twelve patients were treated by radical hysterectomy and pelvic lymphadenectomy, 11 by irradiation after spontaneous abortion or vaginal delivery, 5 by abdominal delivery followed by irradiation therapy, and 2 by irradiation of the intact pregnant uterus.

Of the 23 patients who were treated over 5 years ago, 16 (70%) survived. All patients treated by radical hysterectomy and lymphadenectomy lived over five years. Five of 11 patients who were diagnosed within 1 year post partum died of recurrent cervical cancer. Of the 18 patients with stage I lesions, 12 survived 5 years, while all 5 patients with stage II lesions lived 5 years.

Vaginal delivery through a cervix containing invasive cancer seems to be associated with an unfavourable prognosis. Radical surgical treatment is proposed because it avoids spontaneous abortion associated with radiation therapy, it affords accurate information regarding the extent of the cancer and it reduces the maternal emotional trauma associated with the termination of the pregnancy.—Mikuta, J. J.: Invasive carcinoma of the cervix in pregnancy, *Southern Med. J.*, **60:** 843, 1967.

HISTORY OF CANADIAN SURGERY

JAMES CAMERON CONNELL: 1863-1947

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WHEN James Cameron Connell died on October 5, 1947, Queen's University, Kingston lost a man who, as a student, teacher, pioneer specialist, surgeon and administrator, had served his community for more than half a century. A grateful university had, however, long before his death recognized his single-minded loyalty by bestowing an honorary degree (LL.D. 1930) upon him at his retirement from active university affairs.

Dr. Connell was born of Scottish parents —James Connell and Elizabeth Cameron— on July 25, 1863 at Ayr, Ontario (Fig. 1).
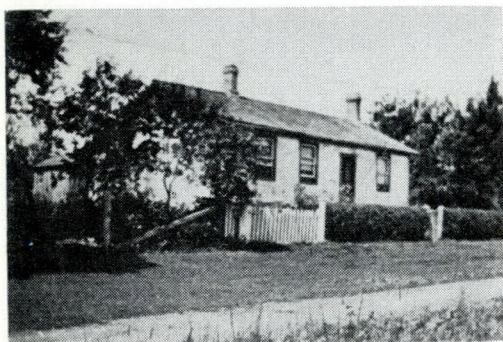
Fig. 1.—Dr. Connell's birthplace in Ayr, Ontario.

He had two sisters, Elizabeth who remained unmarried and Jessie who, like her brother James, also graduated from Queen's University and later married the Reverend James Binnie. The Connell family moved shortly after Dr. Connell's birth to Dundas, Ontario where he completed his high school education. His Scottish-Canadian family were of modest means and reading was a major relaxation. His fondness of reading led to the accumulation of a substantial library and Dr. Connell became a man of wide culture who drew strength from the writings of great men of the past. This, along with his inherited Scottish tenacity, stood him in good stead in later

life. He was a great admirer of Sir William Osler and like him he found a daily spur to action and a philosophy of life in Shakespeare's lines:

"The flighty purpose never is o'ertook
Unless the deed go with it."

From Dundas High School in 1880, at the age of 17, Dr. Connell entered Queen's University with intention of becoming a theologian. He remained in contact with Dundas by working in the post-office there during the summer recess—at a monthly pay of $12! Sometime during his student life, he changed careers and graduated B.A. in 1884, having taken an extra year in mathematics in which subject he received a Gold Medal. He graduated M.A. in 1885 and then entered medicine to graduate M.D., C.M. in 1888. As well as being a distinguished student, he found time to become a leader in many student activities and, in his final year in medicine, was elected to the coveted presidency of the Alma Mater Society.

After postgraduate work in New York, Dr. Connell returned to Kingston to set up practice as the first eye, ear, nose and throat specialist in the district, and indeed the second in Canada to devote his time solely to this specialty. He was appointed professor of this new department at Queen's University in 1891 and remained its head for almost 40 years. His practice grew rapidly and patients came to him from all parts of eastern Canada and New York State. His academic work was of the highest calibre and was recognized by the use for several years of his textbook on his own specialty. Dr. Connell remained active and accomplished in his field until 1937 when, at the age of 74, he retired to devote his time to his family and garden.

In 1903, the venerable Dr. Fife Fowler, who had guided Queen's Medical College through several metamorphoses, died and Dr. Connell was asked to become his successor. The appointment of a relatively young man, 40 years of age, to such a

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responsible position in a medical faculty was not common at that time. The wiseness of this choice was manifest many times during the ensuing 26 years that he remained Dean of the Faculty of Medicine at Queen's. He began a thorough and scholarly development and reorganization of the Medical School, built it up to Grade A standing and, by transforming and rebuilding the Kingston General Hospital, laid the foundations for the present excellence of the Queen's University Faculty of Medicine and its affiliated hospitals. Thus, to his already acknowledged fame as a surgeon, he added that of a skilful administrator with the genius and ability to do and to dare.

He always regarded the Medical School and the Kingston General Hospital as one institution and, in their history, his name is enduringly written. He personally considered the opening of the Douglas Wing and the Richardson Laboratories in October of 1925 as the high point of his career. That day he told the story of the Kingston General Hospital—the second oldest in Ontario—and in handing the buildings over to the Governors stated, "Nothing in my career has given me more satisfaction than to see these buildings come into being." He recounted that when he first knew the hospital its yearly income was less than $3000; in 1925 it was $170,000; would that he could be here today when the budget exceeds $10 million! In such programs as these he showed his breadth of vision, his place as a leader and, by promoting departmental and clinical expansion at Queen's University and the Kingston General Hospital, contributed more than any other man to the development of the medical faculty.

His devotion and loyalty to the medical faculty was forcefully demonstrated in 1913 when he tendered his resignation as Dean of Medicine (Fig. 2). *The Whig* at that time noted that his resignation was "a matter of public concern". Dr. Connell felt that the University policy was "starving" the medical departments, and interfering therefore with the development of the medical faculty which had had largely "to paddle its own canoe". *The Whig* de-

Fig. 2.—J. C. Connell in 1913 at the time of his resignation as Dean of Queen's Medical College.

scribed him as "a wise Dean of the Faculty of Medicine, bringing to his tasks order and system and business capacity, and as a result the medical college has come to occupy a unique and splendid position". Fortunately, the Trustees of Queen's University promised to accord the Faculty of Medicine better treatment in the form of financial support and Dr. Connell was persuaded to withdraw his resignation.

At the outbreak of hostilities in 1914, Dr. Connell was instrumental in organizing the No. 5 Stationary Hospital Unit which later became the No. 7 General Hospital Unit that served with distinction in Egypt and France. Grant Hall on Queen's campus became a convalescent hospital until 1920 and, as a Lieutenant-Colonel, Dr. Connell was consultant in his specialty as well as Dean of Medicine.

As President of the Board of the Kingston Health Association, he was instrumental in the establishment of the Mowat Sanatorium for the treatment of tubercu-

losis on the Van Straubenzie estate in the village of Portsmouth. This location was chosen because it could be easily reached from the Kingston Street railway yet was "sufficiently isolated that the most fastidious could not be annoyed". So successful was this venture that in July 1914 the Kingston Health Association reported that "The work of the hospital has resulted in the City of Kingston being cleaned up of tuberculosis. There is at present no known case of tuberculosis in the City of Kingston not being properly cared for."

After the war the question arose as to whether the medical faculty should remain in Kingston or move to Ottawa where a greater volume of clinical material was available. Dr. Connell was adamant that the Medical School should remain at Queen's and he was largely responsible for it staying. This was one of his finest hours and a time at which his determination and inborn Scottish tenacity was most needed and used. He lost friends over this but he won the respect of many opponents. Thus, in another of its hours of travail, Queen's University Medical School was fortunate in having a man at its helm whose courage and devotion ensured the survival of the Faculty of Medicine.

Dedicated men like Dr. Connell often meet opposition but his fairness and selflessness earned the respect of his opponents who oftentimes found their respect turning into affection. It was this incident that accelerated the remodelling of the hospital and the building of the Douglas Wing. Under his vigorous leadership as the chairman of the building committee, over $1.25 million was raised in the years 1920 to 1925 to complete the project.

In 1929, Dr. Connell retired after more than 25 years as Dean of the Faculty of Medicine. The *Queen's Review* at that time referred to him as a "Maker of Queen's" and referred to the medical faculty as "a fitting monument to his 26 years devotion to Queen's". The Board of Trustees of Queen's stated that "As a teacher and as administrator Dr. Connell has contributed more than any other man to the development and the excellence of the Medical Faculty . . . Queen's stands deep in his debt."
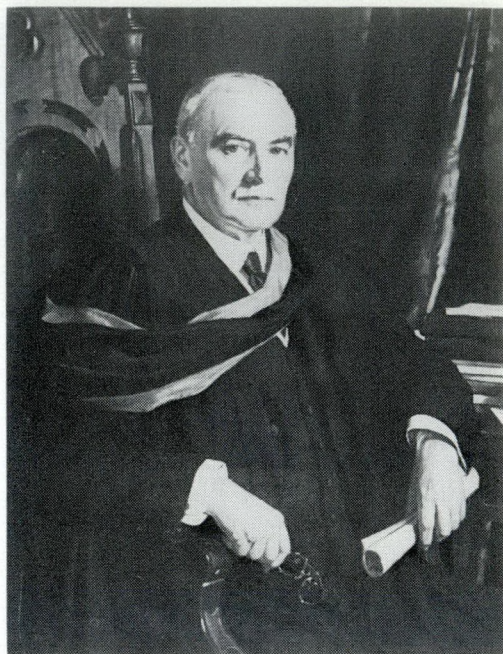
Fig. 3.—Portrait of Dr. Connell as Principal of Queen's University.

His retirement, so happily crowned by an LL.D. from a grateful alma mater, was short-lived because Dr. Connell was asked to act as Principal of Queen's University until a successor to Dr. Taylor could be appointed. He thus stepped into, from May until October 1930, a distinguished line of Queen's principals.

Dr. Connell was a man of many facets— a scholar, teacher and an administrator, pioneering a specialty in his profession and selflessly loyal to his university. In his "In Memoriam" address, Dr. W. E. McNeill said, "He was a man not only of vision, force and action, but also of rich humanity, kind and understanding, devoted to his family and loyal to his friends."

In 1889, Dr. Connell had married Agnes Hendry and their only son H. C. Connell also became an eye, ear, nose and throat specialist. Dr. Connell's home and lakeshore property, enhanced by his gardening skills, was the party scene for many graduating classes and returning alumni. In spite of his busy practice and administrative duties he made time to take a daily drive with Mrs. Connell. When Dr. John Austin arrived in Kingston as a guest in Dr. Con-

nell's home, he put his shoes outside of his bedroom door for the servants to shine. It was only years later that "Blimey" learned the true identity of his shoe-shine boy!

As a pioneer in the specialty of eye, ear, nose and throat surgery, Dr. Connell brought to Kingston new operative procedures and techniques. His skill as a surgeon was widely recognized but his kindliness and gentleness with children in particular was ever present on rounds. A gentle word and a coin in the hand of a fearful child won their confidence. When Dr. Connell's portrait (Fig. 3), painted by Kenneth Forbes, was unveiled at the October Convocation and presented to the University in 1930 by the medical graduates of Queen's University, Dr. E. Ryan (Meds. '89) said: "He was loved and respected by his students, by his confreres of the faculty and by his brothers in the profession."

There are few individuals who can look back on a life so rich in achievement. Dr. Connell had 10 years after his final retirement to enjoy life with his family and
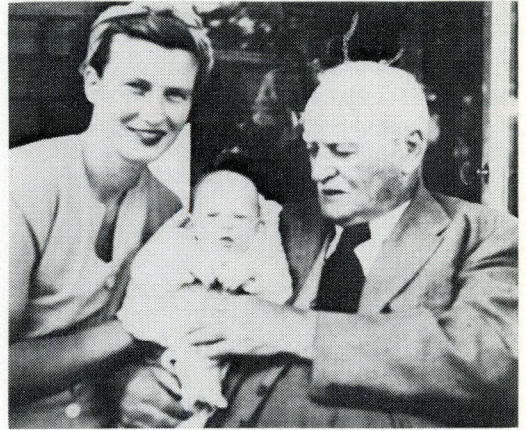
Fig. 4.—Happy retirement.

grandchildren (Fig. 4). No epitaph is more fitting than his own words taken from one of his own texts: "If one has lived well and full, known love and parentage, experienced success and failure, if he has done justly, loved mercy and worked humbly he can die with some measure of content and clear the stage for a newer and better play."

FAILURE AFTER VARICOSE VEIN OPERATION

This paper gives the late postoperative results in 170 patients after 242 operations for varicose veins done between 1959 and 1963. One hundred and one patients (59.4%) had no symptoms and no evidence of recurrence; the remaining 69 patients had a recurrence, either asymptomatic or associated with subjective complaints.

The author distinguishes three forms of recurrence, one of which is on the inner aspect of the thigh, in the area of the removed great saphenous vein. This is caused either by improper ligation of the great saphenous vein at a distance from the saphenofemoral junction, which leaves a long saphenous stump, or by mistaken ligation of another vein, leaving the great saphenous vein undisturbed. Sometimes the recurrence is due to an accessory great saphenous vein.

A second form occurs in the lesser saphenous vein area most often in patients in whom this vein did not show any evidence of varicosity on preoperative examination and was not removed. Postoperatively, under new hemody-namic conditions, varicosities of the lesser saphenous vein may develop quite rapidly.

Ubiquitous recurrence, the third form, arises from overlooked incompetent perforating veins. Knowledge of the anatomy of the perforating veins and thorough preoperative examination and marking helps prevent most of these recurrences.

The exact location of the incompetent perforators may often be recognized by characteristic, localized ectasia of the skin venules, by visible and palpable, easily compressible and promptly refilling vein blowouts, and sometimes by palpable defect in the underlying fascia.

Complications other than recurrent varicosities including damage to the saphenous and sural nerves during dissection and stripping, resulting in numbness of the corresponding skin, and complications resulting from coexistent impaired arterial blood supply to the operated extremity. The latter can be avoided by thorough evaluation of arterial circulation before surgical treatment.—Weber, F.: Misserfolge und ihre Ursachen nach Varicenoperationen, *Chirurg.*, **38**: 322, 1967.

ORIGINAL ARTICLES

ENDOLYMPHATIC INJECTION OF COLLOIDAL RADIOACTIVE GOLD AS AN ADJUNCT TO TUMOUR MANAGEMENT*

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THIS is an account of our clinical experience over the past $2\frac{1}{2}$ years with administration of colloidal radioactive gold by the endolymphatic route. Our objective was prophylactic irradiation of tumour cells and small tumour deposits in lymph nodes and along lymphatic pathways. We have attempted to evaluate the feasibility and proper place in tumour management of endolymphatic radiotherapy with colloidal gold.

Lymphangiography by cannulation of lymphatic vessels was introduced into clinical use by Kinmonth, Taylor and Harper in 1955.[1] Lymphography has subsequently been used widely as a diagnostic measure in diseases involving lymph vessels and lymph nodes. The step from diagnosis to therapy is a small one, and several investigators proposed that various agents might be injected through lymphatic channels to treat primary and secondary lymph node tumours. In the years since 1958, this proposal has been tested experimentally and clinically in many centres.[2-12] We are particularly indebted to Jantet and Kinmonth who pioneered the endolymphatic administration of colloidal radiogold.[2, 6]

RATIONALE

Agents used in intralymphatic tumour therapy must remain, to a greater or lesser degree, within a segment of the lymphatic

system if they are to exert destructive effects upon tumour situated within. A number of radioactive substances have been tried by various investigators and, after preliminary animal studies, we selected colloidal radioactive gold (^{198}Au) for our clinical trials.

The ability of lymph nodes to retain colloidal materials is well known. Gold-198 has a conveniently short half-life of 2.7 days. More than 90% of its biological effect is from beta radiation, and the beta particles have a maximum range of 3.8 mm. and an average range of 0.75 to 0.8 mm. The radiation effect is exerted chiefly upon lymph nodes, lymphatic channels and on *small* tumour deposits within these structures. The overlying skin is largely protected by distance. By the same token, the short range of the beta radiation precludes the possibility of treating large tumour masses. Gold-198 also emits gamma radiation, which enables us to monitor patients and study the distribution and concentration of the material.

If the radiogold can be injected into a lymph vessel near the primary site, the same lymphatic pathways, which may conduct tumour cells, will be swept by the radioactive material, a variable amount of which adheres to the vessel wall. A fraction of the colloidal material will be filtered and retained by the regional nodes, where it can irradiate clumps of tumour cells and small metastases. Because large radiation doses can be achieved in lymph nodes, so-called "radioresistant" tumours may be ideal for this type of treatment, since radioresistance is relative and no tumour can withstand necrotizing doses.

At the present time we regard endolymphatic administration of radiogold chiefly as an adjunct to regional node dissection for three reasons: (1) Nodes that are blocked by tumour are inaccessible to the radioactive material. (2) Tumour deposits

TABLE I.—Patients Treated by Endolymphatic Colloidal Radiogold

A—Patients With Malignant Melanoma of an Extremity

Patient	Age (years)	Sex	Primary site	Nodal metastases*		Injection site	Dose (mc.)	Volume (c.c.)
G.H.	39	F	Leg		0	Foot	4	5.0
G.H.	37	F	Forearm		0	Hand	10	0.8
A.B.	66	F	Toe		+	Foot	12	1.9
H.R.	73	F	Leg		?	Calf	14	8.6
G.S.	51	F	Leg		+	Foot	20	3.2
M.R.	47	F	Leg		0	Calf	25	3.5
I.W.	32	F	Forearm		0	Forearm	20	2.5
H.B.	38	M	Shoulder		0	Arm	6.4	2.0
I.S.	38	F	Leg (recurrence in thigh)		0†	Leg	24	3.0
E.R.	48	F	Leg		0	Ankle	29	3.0

B—Patients With Genital Epidermoid Carcinoma

Patient	Age (years)	Sex	Primary site	Nodal metastases*		Injection site	Dose (mc.)	Volume (c.c.)
G.M.	63	M	Penis		+	Foot	19	3.0
A.W.	72	M	Prepuce	L	+	Left Foot	18	3.0
				R	0	Right calf	18	3.5
M.H.	55	F	Vagina	R	?	Right calf	23	6.0
				L	+	Left thigh	18	3.9
R.M.	57	M	Penis	L	0	Left calf	24	3.0
				R	0	Right calf	24	3.0

*0=negative; +=positive.
†External iliac nodes negative. Inguinal nodes removed at previous (1960) operation.

over 1 to 2 mm. in diameter may be too large to be treated effectively at their centres by bombardment by short range beta particles from the periphery. (3) With our present methods, there is still much uncertainty about our ability to achieve adequate radiation doses in the regional nodes.

Patients

Fourteen patients (Table I) received endolymphatic injections of colloidal radiogold, three of them bilaterally. All patients had histologically proved malignancy of a type likely to spread by the lymphatics. The series consisted of 10 patients with malignant melanoma of the extremities (Table IA) and 4 patients with epidermoid carcinoma of the genitalia (Table IB). In all these patients the primary lesion was adequately controlled and, at the time of treatment, none had evidence of metastases beyond the regional lymph nodes.

Regional lymph node dissections were carried out in 12 patients after the endolymphatic injections. In one (M.H., who had carcinoma of the vagina) the second lymphogram showed extensive involvement of the external iliac nodes and she was

considered to be beyond cure. The other (H.R., a patient with melanoma of the leg) was elderly and not thought fit for operation. Following injection of radiogold, we waited 13 to 18 days before doing node dissection—a period during which 96 to 99% of the radiant energy was expended by physical decay.

Technique

Lymphatic cannulation was done using the same technique employed in diagnostic lymphography: alphazurine 2G is used as a delineating dye to colour the lymphatics, and a lymph vessel in the subcutaneous tissue is exposed under local anesthesia. Whereas for diagnostic procedures, the incision is usually made in the dorsum of the hand or foot, a lymphatic vessel in a more proximal part of the limb is often more suitable in this procedure. The lymphatic is dissected out, elevated on a mask wire, and cannulated with a No. 27 or No. 30 needle attached to plastic catheter. The needle is secured with several fine cotton ligatures, and saline is injected to demonstrate that there is no leak. One or two millilitres of ethiodized oil (Lipiodol Ultrafluid) is then injected and monitoring radiographs are taken. If it can be shown

that lymphatics and lymph nodes are being filled without leak or extravasation, colloidal radiogold is injected.

The radioactive gold used is the same material available commercially for several diagnostic and therapeutic purposes. We prefer to use concentrations in the order of 8 mc./ml., if available, in order to keep the volume below 3.5 ml. in the lower extremities and 2.5 ml. in the upper. Injection time for the gold is approximately 45 to 90 minutes. One or two millilitres of the Lipiodol Ultrafluid is then injected in order to clear the catheter, needle, and lymphatic of radioactive material. After the cannula is removed, the wound is washed with saline and the washings are monitored for radioactivity.

Patients are allowed to "cool off" for about two weeks, the time during which the radiation effect is being exerted, before the lymph nodes are dissected out. Diagnostic radiographs of the lymph nodes are deferred until the latter part of the "cooling off" period to minimize exposure of personnel.

CLINICAL OBSERVATIONS

No attempt was made to evaluate the effect of endolymphatic therapy with radiogold on the malignant tumour because our patients are few and recent, and our methods and doses are still evolving. We will confine our critique to the feasibility of the procedure, early effects on the patient, and the question of whether the doses achieved are, on theoretical grounds, adequate for tumour treatment.

Local Effects

While the technique is tedious and time consuming, we have not had a major problem with a leak of radioactive material at the injection site. While the injection in one patient (H.B.) was discontinued early because of a minor leak, wound washings did not show appreciable radioactive contamination after any of the 17 injections, nor has wound healing at the injection site been delayed.

On one occasion we planned to administer endolymphatic gold but could not

Fig. 1.—Monitoring film showing a leak of radio-opaque oil from lymphatics of the groin into the site of an inguinal node biopsy. Endolymphatic radiotherapy cannot be given in this situation.

because we could not cannulate a lymphatic in an obese woman. This patient, who had a melanoma on the lateral aspect of the arm, had had operations in the axilla for hiradenitis suppurativa. Cannulation was attempted on the medial aspect of the arm, where only a single lymphatic vessel was found at a very great depth. In retrospect, this cannulation should have been attempted in the antecubital fossa. In a second patient we had to abandon radiogold administration because of a leak of Lipiodol Ultrafluid from groin lymphatics, where an inguinal lymph node had been excised (Fig. 1).

In one patient, H.R., the first lymphatic cannulation was on the dorsum of the foot. Monitoring films showed that Lipiodol Ultrafluid was leaking out in the lower leg at the site of excisional biopsy of a malignant melanoma (Fig. 2). We then had to go above the biopsy site and cannulate a lymphatic channel in the upper third of
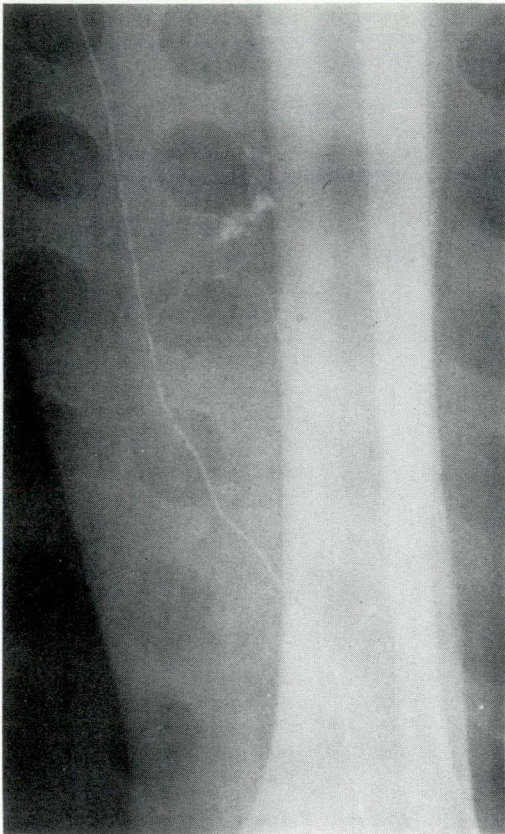
Fig. 2.—Monitoring film showing a leak of radio-opaque oil from lymphatics of the leg into the site of an excisional biopsy of a melanoma. It was necessary to cannulate a lymphatic above the biopsy site in order to inject colloidal radiogold.

Fig. 3.—Radiation changes in the skin of the mid-leg overlying an area of dermal reflux. This complication could have been avoided by injecting the colloidal radiogold into a lymphatic channel in the upper one-half of the leg.

the leg in order to give this patient endo-lymphatic radiogold.

We learned from experience how to avoid leaving large amounts of radioactive material in the limb below the regional nodes, where it may produce radiation changes in the skin, and where it may have no useful effect on lymph nodes:

(1) G.M., who had carcinoma of the penis and clinically involved lymph nodes, was given radiogold via a lymphatic channel in his foot. He had an area of lymphatic dermal reflux in the lower half of his leg. A significant fraction of the colloidal gold was trapped in the skin of his leg, he nearly developed an area of radiation necrosis over his shin (Fig. 3), and the regional lymph nodes received very little radioactivity. Now, in such a patient, we would inject the gold in the upper third

of the leg or in the lower thigh. Further-more, under no circumstances would we inject radiogold below an obvious area of dermal reflux.

(2) Gold was also injected into the left foot of another patient (A.W.), whose primary and regional nodal involvement were similar to those of G.M. He did not have dermal reflux, but sufficient gold was left along the course of the lymphatics in his leg to produce brown pigmentation of the overlying skin (Fig. 4). While such irradiation of lymphatic pathways might prove useful in preventing "in transit" metastases from malignant melanoma, the radioactivity was wasted in this patient. Subsequently, the right side was treated using an injection site in the calf and, fol-lowing radical groin dissection, a larger fraction of the administered radiogold was recovered from the nodes on the right than on the left.

(3) From experience with other pa-tients, we have concluded that the amount

Fig. 4.—Radiation changes in the skin on the medial side of the leg after colloidal radiogold was injected into a lymphatic on the dorsum of the foot. Large amounts of the gold have adhered to the walls of the lymphatic vessels, which in this patient lie just beneath the brown streak on the skin. In this particular case, the radioactivity expended in the limb was wasted because therapy was directed at inguinal and iliac nodes draining a carcinoma of the penis.

of radio-opaque oil injected before the gold, used for monitoring purposes, should be limited to 1 or 2 ml. to keep to a minimum the amount of colloidal material lost in the limb.

Undesirable local effects have been few and confined to our early cases but we emphasize them to help others avoid similar experiences.

Following regional node dissections, most patients have had prolonged accumulations of lymph beneath the skin flaps. These collections of lymph had to be aspirated or drained for as long as four or even six weeks after dissection. This complication may be due to the Lipiodol Ultrafluid rather than to the radiogold.

Four patients had some delay in wound healing following lymph node dissection. In only one (I.W.) could delayed wound healing reasonably be attributed to the radiogold.

One patient (G.H.) had considerable but not disabling lymphedema of the lower extremity following biopsy of a melanoma of the leg, excision and grafting of the biopsy site, injection of radiogold, and radical groin dissection. Since she received the smallest amount of radioactivity of any patient in our series, it does not seem reasonable to attribute her complication to radiation.

Systemic Effects

In the two weeks after endolymphatic injection of radiogold, most of our patients had good appetites; those who were anorexic had either respiratory infections or emotional problems. No patient had nausea or vomiting after injection.

Half of the patients who received more than 10 mc. of radiogold by the endolymphatic route had a transient lymphopenia. The total white cell count and hemoglobin did not appear to be affected.

STUDIES ON RADIATION DOSES RECEIVED BY REGIONAL LYMPH NODES

Methods

Immediately after lymph node dissection, the surgical specimen was put in its correct anatomical orientation and radiographed. Individual nodes were dissected out, identified, and again radiographed. Lymph nodes were weighed while still

fresh, sliced, and every second slice was kept for analysis. The alternate slices were sent to the pathologist.

Specimens for analysis were again weighed and put in flasks with an approximately equal weight of potassium hydroxide pellets. These flasks were heated for about two hours in boiling water. Distilled water was then added to give a total volume between 25 and 100 ml./g. of tissue, and the flasks were heated for another two to four hours. If the specimens were still not homogeneous, a small amount of detergent was added and the specimens were homogenized at 4500 r.p.m. Aliquots of the resulting solutions or homogeneous suspensions were counted against a standard, either in an automatic well-type scintillation counter, or in a Tobor twin-crystal scintillation counter. The concentration of ^{198}Au was then calculated in μc./g. of lymph node.

Radiation Dosimetry

Although ^{198}Au emits both beta and gamma radiation,* the deeply penetrating gamma radiation contributes little (less than 10%) to the dose absorbed within the relatively small dimensions of the lymph nodes. It has been shown[13] that if one assumes a uniform concentration of radioactive material within a given volume of matter, the beta dose to infinity may be calculated from the following equation:

$$\text{DOSE}\beta \; (\infty) \; = 73.8 \times C_i \times \overline{E}\beta \times T_{eff} \text{ rads}$$

Where:

$\text{Dose}\beta \; (\infty)$ = Dose in beta rads at the centre of a node, calculated to infinite time.
C_i = The initial concentration of radiogold in μc./g. of tissue.
$\overline{E}\beta$ = Mean energy of the beta particle (one-third the peak energy or 0.32 Mev. for ^{198}Au).
T_{eff} = The effective half-life of ^{198}Au within the lymph node (2.6 days).

The validity of this formula, when applied to dosimetry of lymph nodes, depends upon the following considerations:

(1) Calculations assume uniform concentration of gold within the node, where-

as concentrations are not strictly uniform. If there are areas of fibrosis, fatty replacement or tumour infiltration, calculated doses will underestimate the true dose within the lymphoid tissue. Even within normal lymphoid tissue, the concentration of colloidal material is not uniform. The extent and magnitude of this non-uniformity are now being investigated.[14]

(2) The effective half-life of colloidal gold in lymph nodes is not known precisely, but in the whole body it is generally accepted as being just over 2.6 days.[15] Daily monitoring of lymph nodes, using an external scintillation counter, suggests that the effective half-life is in the order of 2.6 days, and this is the value we have taken for our calculations.

(3) The initial concentration of radiogold is obtained from the concentration found by analysis following lymph node dissection. Correction is made for the slow loss of the colloidal material between the day of endolymphatic injection and the day of node dissection as follows:

$$C_i = C_a \times e^{\dfrac{.693\,t}{70}}$$

Where:

C_i = Initial concentration of radiogold (μc./g.).
C_a = Concentration of radiogold from analysis, following node dissection.
t = Time, in days, between endolymphatic administration and node dissection.

The value, 70, is the biological half-life in days, derived from the known physical half-life and the assumed effective half-life.

(4) On the basis of uniform concentration of the ^{198}Au within the node, the peripheral dose would be about 50% of the calculated dose at the centre. Animal studies indicate, however, that fat immediately surrounding a lymph node (this includes afferent and efferent lymphatic vessels) contains 10 to 20% of the concentration of radioactivity found in the node itself.[16] Thus, the dose at the periphery of a node would be somewhat greater than 50% of that at the centre.

Results

Following eight groin dissections an at-

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*Principle beta = 0.957 Mev.; principle gamma = 0.412 Mev.
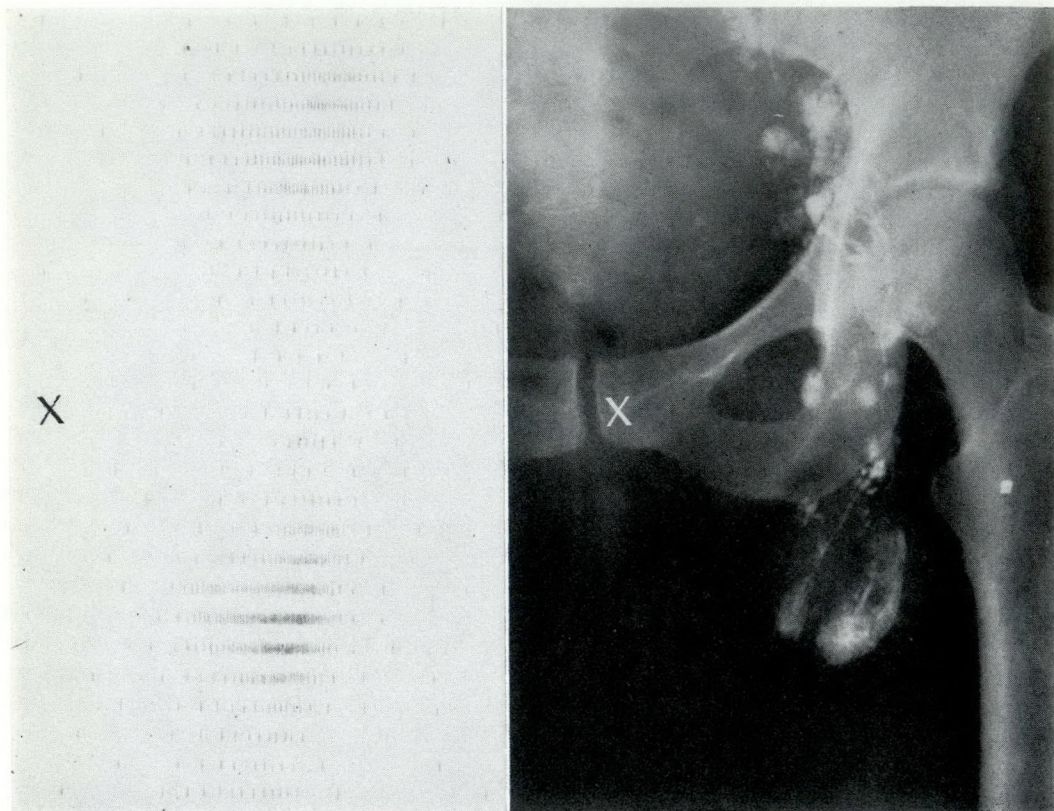
Fig. 5.—Scintiscan and lymphogram of the left groin area (G.H.), 11 days after injection of radio-opaque oil and 4.0 mc. of radiogold. Most of the radioactivity is in the lymph nodes and there is more activity in the lower nodes than in the upper. The X indicates the position of the pubic tubercle.

tempt was made to account quantitatively for radiogold in the lymph nodes. Between 6.3 and 31.5% of the injected dose was recovered in the dissected nodes, and the fraction recovered did not appear to be a function of the weight of the lymph nodes. Another 4 to 17% of the administered dose could be accounted for in the fatty and areolar tissue of the surgical specimen, chiefly in association with afferent lymphatic channels to nodes containing the highest concentrations of radiogold. In addition, appreciable quantities of radiogold were left in the injected limb along the course of lymphatic pathways between the injection site and the regional nodes; we estimate this amount to be about 10 to 20% of the administered dose.

Fig. 5 shows a "scintiscan" and a radiograph of the same groin following endolymphatic administration of colloidal radiogold. The tendency of the radioactivity to concentrate along lymphatic channels and over lymph node areas is apparent. This patient (G.H.), the first in our series, received only 4.0 mc. of radiogold. Even so, 11 days after injection, when only 6% of the initial activity remained in the lymph nodes, the sensitive scanning instrument had to be covered with lead and attenuated electronically to carry out the scan. It was not practical, then, to scan our patients as a routine procedure.

Fig. 6 is a radiograph of the left ilioinguinal nodes of R.M. following endolymphatic injection of Lipiodol Ultrafluid and radiogold. The doses in beta rads to infinity calculated for many of the nodes are superimposed on the film. A more detailed summary of our study on this groin dissection is shown in Table II.

Photomicrographs of representative lymph nodes from the left ilioinguinal dissection of R.M. are shown in Fig. 7. These
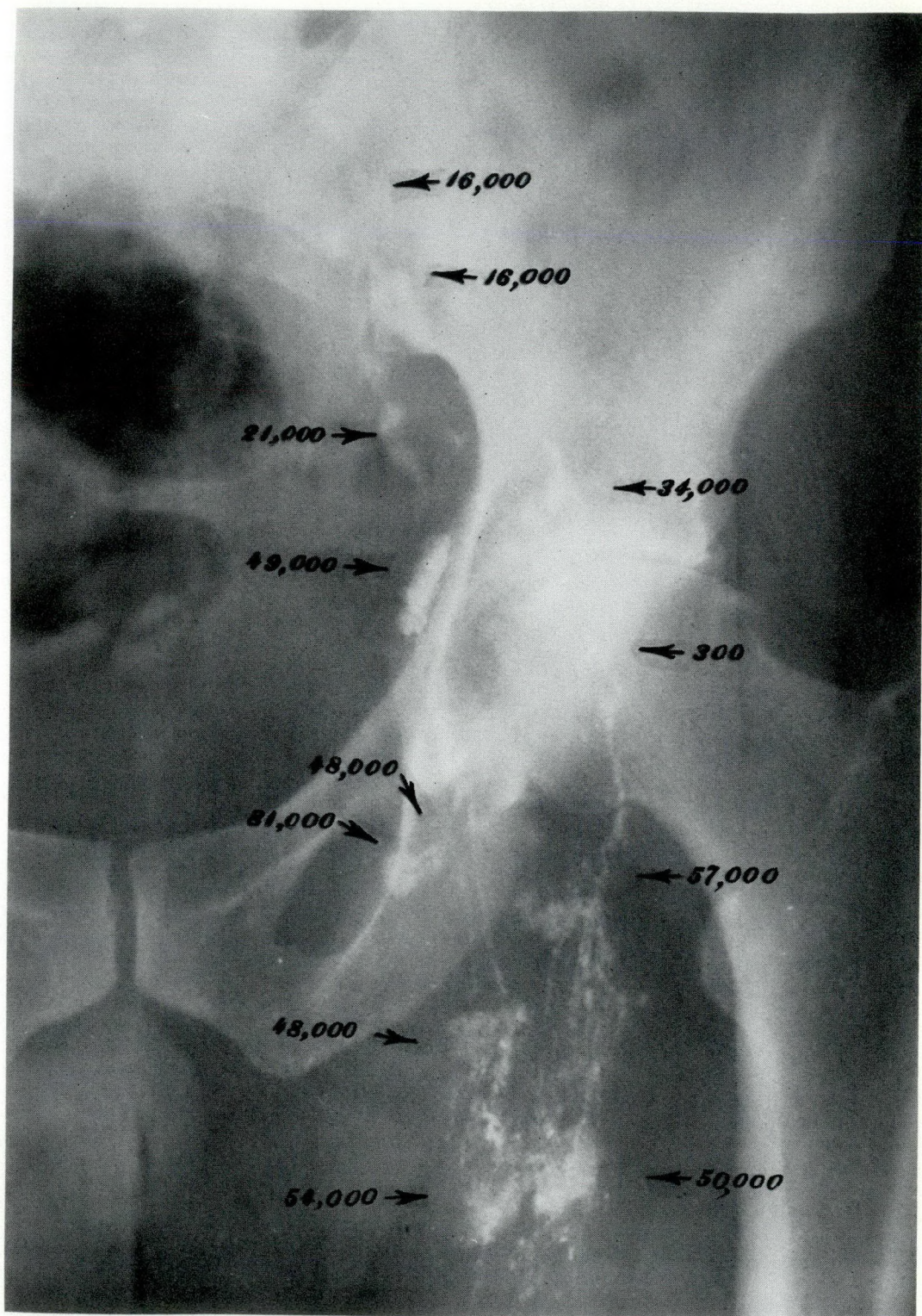
Fig. 6.—Lymphogram of the left ilioinguinal region (R.M.) after endolymphatic administration of 24 mc. of colloidal radiogold. The calculated doses in beta rads received by many of the nodes are indicated.

TABLE II.—R.M.—Nodes of the Left Groin

External iliac nodes	Percentage of administered dose	Activity (μc.)	Weight (g.)	Specific concentration (μc./g.)	Dose (beta rads to infinity)
Lateral chain					
A	0.261	62.69	0.243	258.0	15,800
B	0.768	184.4	0.686	268.8	16,500
Middle chain					
C	0.181	43.37	0.120	361.4	22,200
D	0.402	96.6	0.288	335.4	20,600
E	3.86	926.5	1.821	508.8	31,200
Medial chain					
F	2.37	568.1	0.710	800.2	49,100
G	0.231	55.53	0.105	528.9	32,500
H	0.233	55.93	0.071	787.7	48,400
I	1.173	281.6	0.213	1322.0	81,200
Inguinal nodes					
Horizontal chain					
J	0.013	3.07	0.702	4.4	270
K	0.048	11.61	0.183	63.44	3900
Lowest nodes					
L	6.054	1453.0	1.790	811.8	49,800
M	6.03	1477.0	1.665	886.9	54,500
Remaining nodes					
N	1.892	454.0	0.586	774.8	47,600
O	1.535	368.4	0.326	1130.0	69,400
P	5.90	1416.0	1.516	933.8	57,300
Q	0.597	143.2	0.387	370.0	22,700

show a range of radiation changes, correlating with the calculated radiation doses, from minimal lymphoid atrophy at the one extreme, to radiation necrosis at the other.
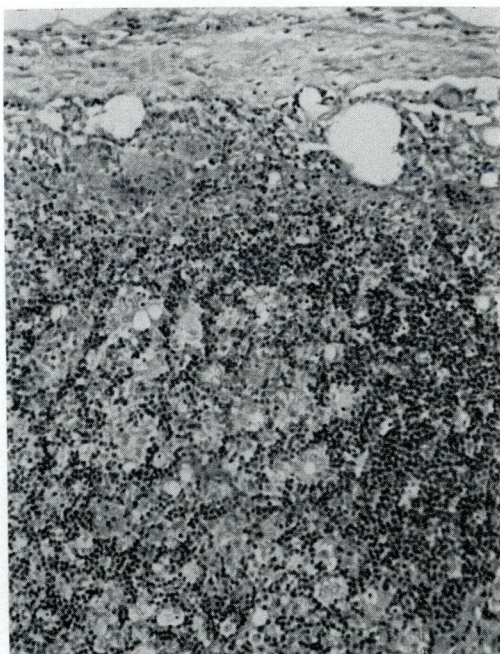
a   b

Fig. 7.—Sections of lymph nodes from a left ilioinguinal node dissection (R.M.). (a) Section from a node that received 3900 beta rads. Only minimal radiation changes are seen. (b) Section from a node that received 15,800 rads. More marked radiation changes are seen, especially lymphoid atrophy.
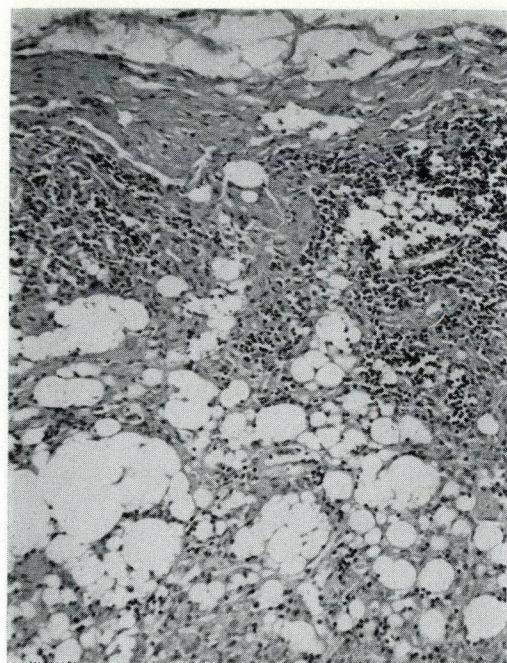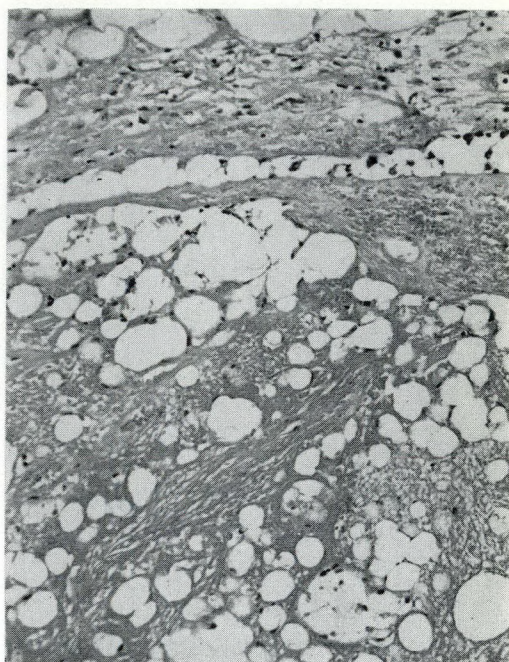
Fig. 7.—(c) Section from a node that received 31,200 beta rads. The changes described above are more advanced. (d) Section from a node that received 81,000 beta rads. The node is necrotic, even the supporting cells (x 120).

Fig. 7d shows a section from a node that received 81,000 beta rads; radionecrosis of even the supporting tissues is apparent, and it is reasonable to suppose that similar effects would extend to small deposits of tumour, even of types ordinarily considered to be relatively radioresistant.

Tables III and IV show the radiation dose in beta rads in various groups of nodes following eight groin dissections. Doses are

TABLE III.—Radiation Dose in the External Iliac Nodes After Eight Groin Dissections

Patient	Lateral chain				Middle chain				Medial chain*			
	Number of nodes	Beta rads × 10³			Number of nodes	Beta rads × 10³			Number of nodes	Beta rads × 10³		
		Max.	Min.	Median		Max.	Min.	Median		Max.	Min.	Median
A.W.—left........	4	90.4	17.0	38.8	0	—	—	—	6	9.7	0.3	4.0
A.W.—right......	5	183.0	14.7	40.6	4	24.7	6.5	11.7	8	15.0	2.8	8.8
R.M.—left........	2	16.5	15.8	16.0	3	31.0	20.6	22.2	4	81.2	32.5	48.7
R.M.—right......	4	31.7	10.7	19.5	2	8.3	4.4	6.3	5	22.2	6.4	17.1
G.S..............	5	3.5	1.3	2.7	5	8.4	0.6	2.1	3	8.3	1.2	4.9
M.R.............	3	10.4	2.8	8.2	6	4.8	1.5	4.0	7	18.2	2.6	6.1
I.S..............	8	121.2	19.6	44.9	4	106.7	12.3	67.7	4	26.4	6.4	11.2
E.R.............	—	—	—	—	—	—	—	—	2	3.7	3.7	3.7

*Includes deep inguinal (femoral) nodes.

TABLE IV.—Radiation Dose in the Inguinal Nodes After Eight Groin Dissections

Patient	Horizontal chain (superficial inguinal nodes)				Lowest superficial subinguinal node(s)				Remaining inguinal nodes			
	Number of nodes	Beta rads × 10³			Number of nodes	Beta rads × 10³			Number of nodes	Beta rads × 10³		
		Max.	Min.	Median		Max.	Min.	Median		Max.	Min.	Median
A.W.—left........	2	0.7	0.2	0.4	1	76.8	—	76.8	1	7.6	7.6	7.6
A.W.—right......	1	0.5	—	0.5	1	35.6	—	35.6	9	20.2	11.2	17.0
R.M.—left........	2	3.9	0.3	2.0	2	54.5	49.8	52.0	4	69.4	22.0	52.4
R.M.—right......	4	2.8	1.9	2.6	2	34.2	21.2	27.7	11	44.0	10.3	14.2
G.S..............	5	0.9	0.1	0.4	1	16.0	—	16.0	8	14.5	1.4	6.4
M.R.............	4	2.6	0.2	0.7	1	19.2	—	19.2	11	27.1	2.3	15.7
I.S..............	—	—	—	—	—	—	—	—	—	—	—	—
E.R.............	3	0.2	0.2	0.2	1	23.6	—	23.6	8	16.9	1.3	9.4

TABLE V.—I.W.—NODES OF THE RIGHT AXILLA

Node	Percentage of administered dose	Activity ($\mu c.$)	Weight ($g.$)	Specific concentration ($\mu c./g.$)	(Dose beta rads to infinity)
Upper axilla					
A	0.2475	48.50	0.448	110.6	6800
B	0.0028	0.55	0.143	3.8	200
C	0.40	79.90	0.105	761.8	46,800
D			0.210		
Mid-axilla					
E	4.125	825.0	1.306	631.9	38,800
F	0.26	52.05	0.078	670.7	41,200
G	0.136	27.16	0.356	76.25	4700
H	0.182	36.49	0.239	152.8	9400
I	4.56	911.8	1.391	655.3	40,200
Lower axilla					
J	0.822	164.3	0.156	1051.0	64,500
K	1.807	361.4	1.012	357.1	21,900
L	1.825	365.0	0.411	888.2	54,500
M	0.098	19.55	0.156	124.9	7700
N	0.253	50.53	0.416	121.5	7500

calculated at the centres of nodes and to infinite time. Because there is much disparity between maximum and minimum doses, the median dose received by a node in each group is shown as an index of central tendency.

In general, lower nodes tended to receive higher doses of radiation than upper nodes, although there were conspicuous exceptions. The lowest of the subinguinal nodes, which would be expected to receive the highest radiation doses, are the nodes most likely to be affected by fibrosis and fatty infiltration. It seems likely that if only lym-
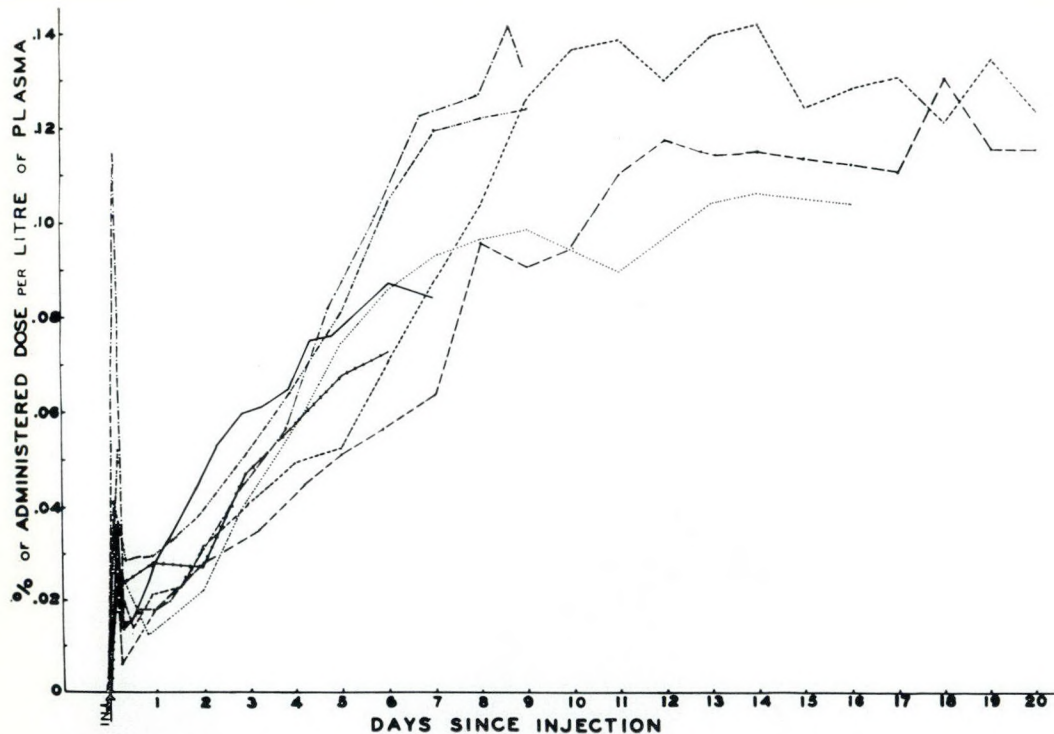
Fig. 8.—Serial plasma levels of colloidal radiogold in seven treated patients. At no time does the concentration exceed 0.15% of the administered dose per litre of plasma.
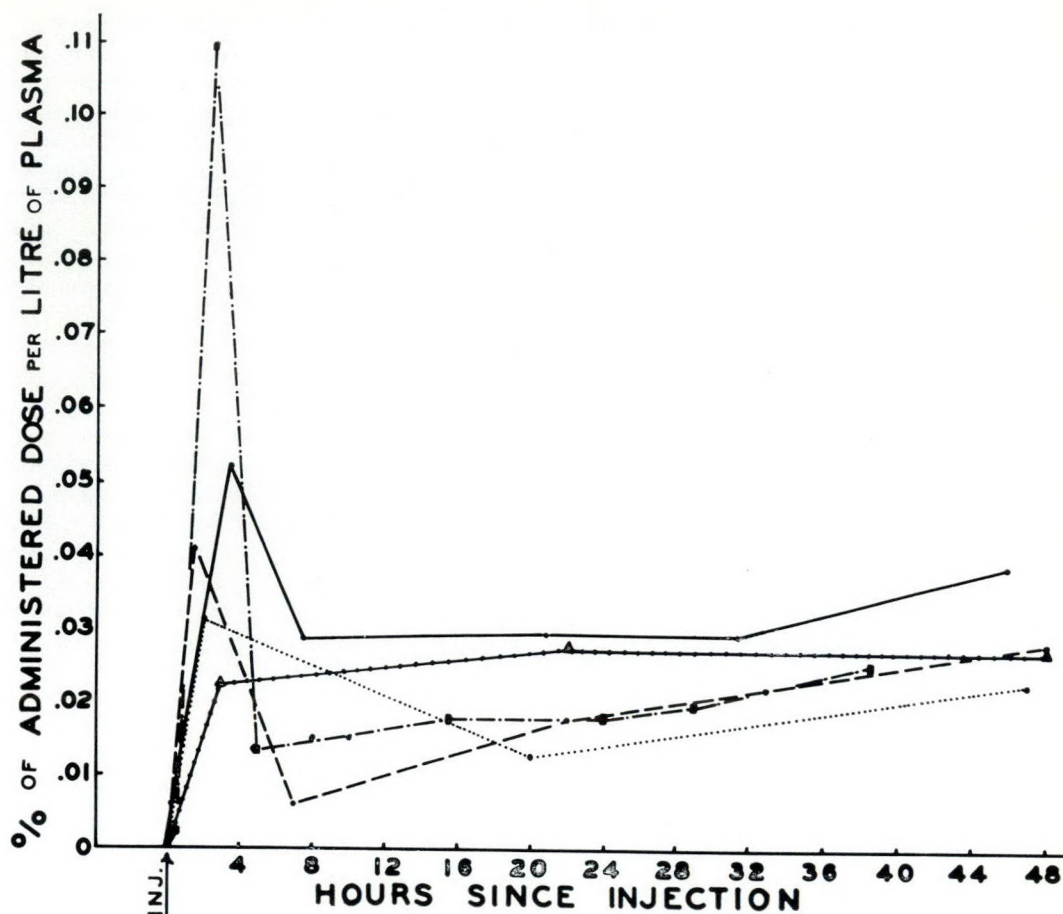
Fig. 9.—Detail from Fig. 8 showing changes in the plasma levels of colloidal radiogold in five patients during the first 48 hours after injection.

phoid tissue were analyzed, radiation doses in the subinguinal nodes would be greater.

The very low doses in the horizontal chain of superficial inguinal nodes, draining abdominal wall, genitalia, and buttocks may be explained on anatomical grounds. Colloidal material injected into a lymphatic channel in the foot or leg should not be expected to fill these nodes in any significant concentration, although appreciable doses may be achieved in the next echelon, the external iliac nodes.

Significant radioactivity was found in axillary nodes in only one patient, because the other two, who had upper limb injections, received only small amounts of radioactivity and an appreciable amount of activity was lost in the arm of one of these. A summary of the results of this study is shown in Table V (I.W.).

STUDIES ON CONCENTRATIONS OF RADIOGOLD IN BLOOD AND URINE

Methods

Venous blood specimens were obtained before and every few hours after endolymphatic administration of radiogold, then daily or every other day for as long as three weeks after injection. One millilitre aliquots of plasma and hemolyzed whole blood were counted against a standard in an automatic well-type scintillation counter. In recent patients, microhematocrit determinations were done on each specimen.

Twenty-four-hour urine specimens were collected for periods up to three weeks following injection of radiogold. One-hundred millilitre aliquots were counted against a standard in a Tobor twin-crystal scintillation counter. In recent patients,

urine collections were controlled by daily creatinine determinations. Because some 24-hour urine specimens were apparently not complete, volumes were calculated to conform with an assumed constant daily creatinine excretion.

Results

Plasma and whole blood levels.—The total amount of colloidal radiogold in the blood at any one time is exceedingly small. Fig. 8 is a composite graph showing serial plasma levels of colloidal gold in seven patients for periods up to 20 days after endolymphatic injection. A basic similarity in the pattern shown by these patients is apparent. Within the first six hours after injection is begun, there is a rapid rise to a variable peak and a rapid fall to a very low level. The early rise is presumably due to a small lymphaticovenous leak, and the rapid fall is thought to be due largely to clear-

ance of the blood by the reticuloendothelial system, in particular by Kupffer cells in the liver. A gradual secondary rise in the plasma concentration of the colloidal gold then takes place over 9 to 12 days to reach a plateau, where it remains for as long as measurement is feasible. It will be seen that the concentration of colloidal gold never exceeds 0.15% of the administered dose per litre of plasma, and maximum levels are reached only after 90 to 95% of the energy has been dissipated. The same events in the first 48 hours are shown in greater detail in Fig. 9.

Figs. 10 and 11 show serial plasma and whole blood levels of colloidal gold in two patients following endolymphatic injection. Analysis of similar data and hematocrits in several patients indicates that the greater part of the colloidal gold is found, as a rule, in the plasma. In the first few hours following endolymphatic injection and for
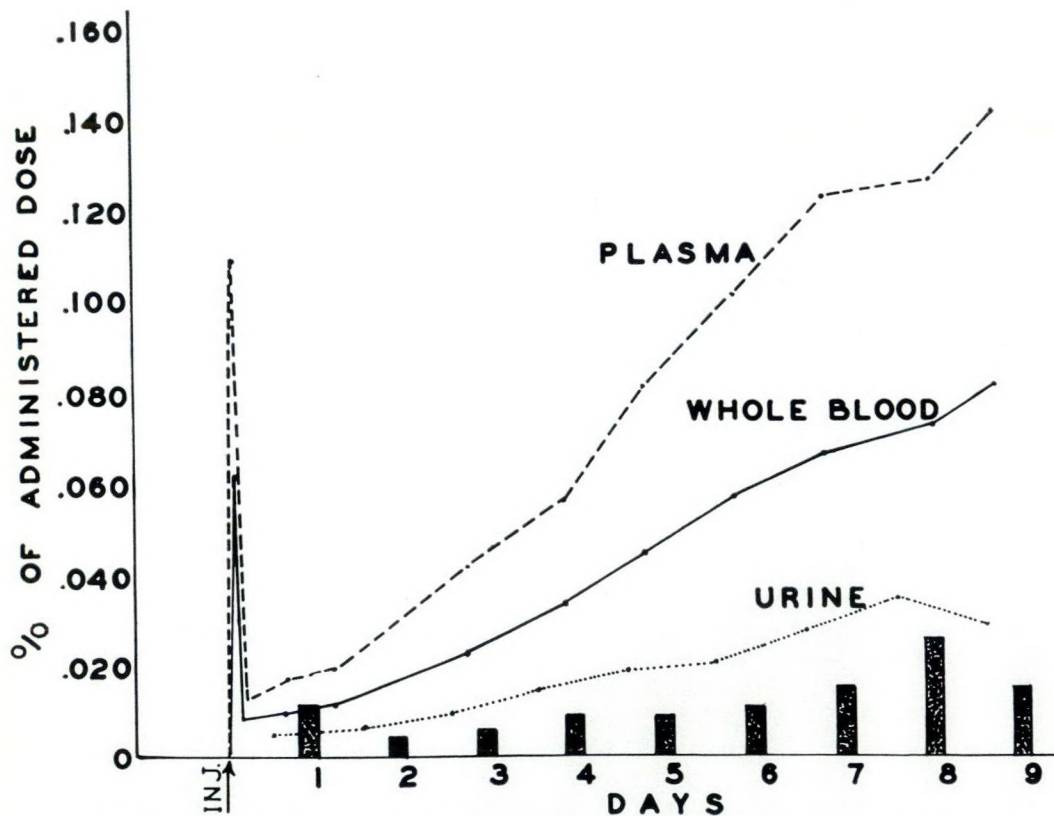
Fig. 10.—The three line graphs indicate concentrations of colloidal radiogold per litre of plasma, whole blood, and urine, respectively, during the first nine days after endolymphatic injection (R.M., left side). The bar graph indicates the total urinary excretion of colloidal gold per 24 hours. All values are a percentage of the administered dose.
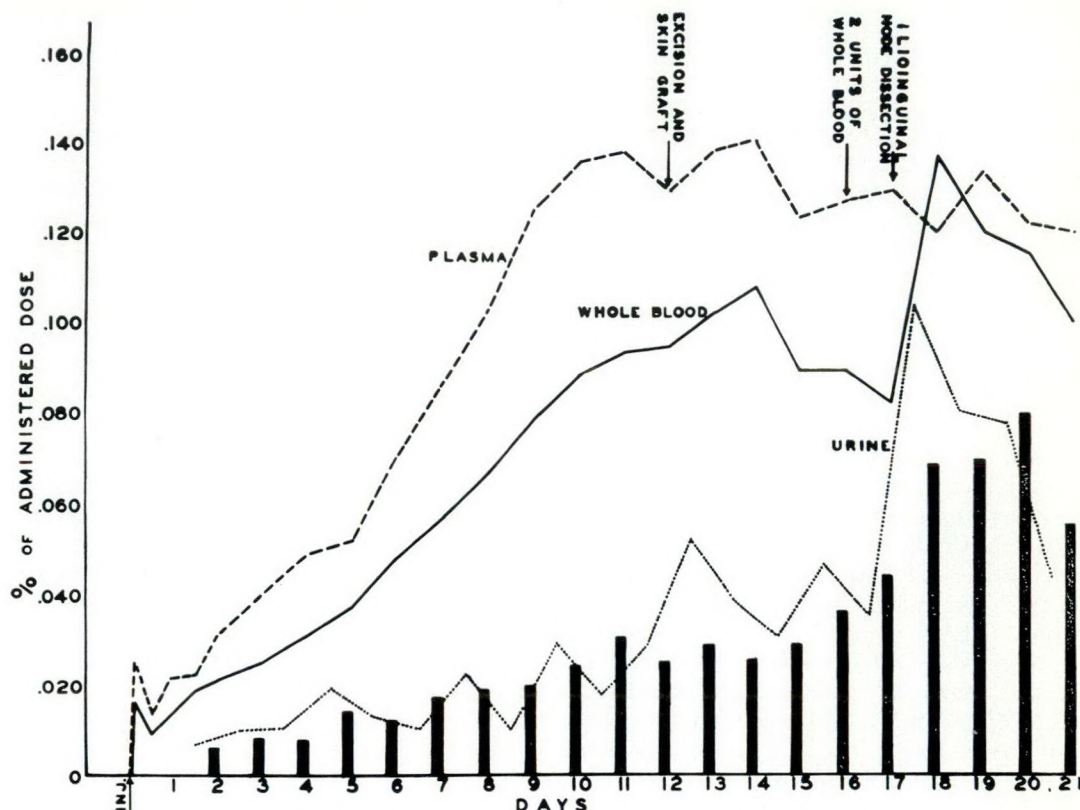
Fig. 11.—The three line graphs indicate concentrations of colloidal radiogold per litre of plasma, whole blood, and urine, respectively, during the first 21 days after endolymphatic injection (M.R.). The bar graph indicates the total urinary excretion of colloidal gold per 24 hours. All values are a percentage of the administered dose.

several days following node dissection, larger amounts of colloidal gold may be found in the cellular fraction of the blood.

Urinary excretion of colloidal radiogold.—The amount of colloidal radiogold excreted in 24 hours seems to vary with the plasma level. It is, however, well below the quantity found in one litre of plasma at any given time (Figs. 10 and 11). Fig. 11 also illustrates the increased concentration of the gold in the urine at times of postoperative antidiuresis. It should be emphasized that the greatest quantities of colloidal gold are found in the urine at a time when the energy is largely expended.

The total amount of colloidal radiogold excreted in a period of three weeks varied, in eight patients, from 0.5 to 3.8% of the administered dose.

In one patient, the quantity of colloidal radiogold excreted in the feces over a three-week period was similar to that excreted in the urine.

DISCUSSION

It is an intriguing concept—that of using, for the treatment of metastases, the very channels along which tumour cells disseminate and the very filters in which they are initially trapped. However, before extensive clinical trials with any particular form of endolymphatic radiotherapy would be justified, two prerequisites should be met: (1) it should be shown that radiation doses to the nodes are adequate for effective tumour treatment; and (2) that the therapy is reasonably safe.

The large radiation doses that have been achieved with colloidal radiogold in some nodes and in some patients have been gratifying, especially as these doses have been given with only minimal early local and systemic effects. While a tumouricidal dose is difficult to define precisely, it seems likely that doses in excess of 30,000 to 50,-000 beta rads will sterilize most small secondary deposits, depending somewhat

on the type of tumour, the size of the metastatic focus, and its position in the node.

It is apparent, however, that many nodes, which are theoretically accessible by lymphatic cannulation of the extremity, have not received therapeutic doses. This, then, is the challenge that must be met if this mode of endolymphatic radiotherapy is to become practical.

In some instances the low doses could be artifactual, produced by appreciable amounts of fatty and fibrous tissue within the nodes. If this is the case, we would expect the radiation doses received by the lymphoid tissue to be higher than we have calculated.

Assuming that the suboptimal radiation doses are real, and for the most part they probably are, there are several means by which we may hope to achieve higher doses. The first of these is to administer radiogold of a higher specific activity. This would enable us to deposit larger amounts of radioactivity in the regional nodes without injecting either a larger volume or a larger number of colloidal particles. This proposal does, however, raise the question of the probable maximum dose that may be safely administered to the patient.

It is also possible that higher radiation doses would be obtained if only a minimal quantity of radio-opaque oil were used for monitoring before injection of the radiogold. We have some evidence to suggest that the oil, when injected in advance of the colloidal material, may interfere with the node's ability to retain colloidal particles.

We have little knowledge of the effect (on the ability of lymph nodes to filter and retain colloidal material) of such variables as: (1) the number of colloidal particles presented to a given mass of lymphoid tissue; (2) the size of the colloidal particles; (3) the rate of administration; (4) the volume in which the radioactive material is given; and (5) pre-existing states of inflammation and hypersensitivity in the lymph nodes. Further investigation of these parameters may suggest modifications in our technique that might result in the higher radiation doses required and yield a more even distribution of the gold along a chain of nodes.

Is this therapeutic adjunct safe? Untoward local effects have not been common or serious and we believe that they can be kept within acceptable limits if one heeds the precautions we have outlined. Early systemic effects have been minimal or nonexistent with the doses we have administered. We are further reassured by our data on blood and urine concentrations; that is, the quantity of colloidal material in the circulation at any instant is certainly minute, as is the whole body dose from circulating radioactive material.

Possible long-term effects of colloidal radiogold, especially on the liver, suggest caution in increasing administered doses. Massive doses of colloidal radiogold by the intravenous route will produce cirrhosis and ascites in puppies,[17] but the dose that is nontoxic is not easy to establish. If data from Rygard's study[18] on liver damage following intraperitoneal colloidal radiogold in mice is extrapolated we could predict that the safe dose would be somewhat less than 1 mc./kg. body weight.

It is well established that colloidal gold given intravenously is rapidly cleared by the liver.[19] It seems likely then, that most of the radioactive material that escapes from the lymphatic system and into the blood will eventually lodge in that organ. There is no quantitative data in man, but Schwartz et al.[3] have shown that between 13 and 20% of colloidal gold given to dogs by the endolymphatic route is found in the liver. Our own studies on rabbits, however, indicate that the liver retains about twice this amount.[16] Even if one assumed that as much as 50% of the administered dose goes to the liver, the maximum absorbed dose to the liver in any of our patients would have been approximately 600 beta rads and 100 gamma rads. This is reasonably safe for a patient with malignant disease, and it is likely that doses twice as large would be acceptable, at least for older patients.

We note that Jantet et al.[6] administered doses of between 16 and 70 mc., and that Ariel, Resnick and Galey[9] gave doses between 20 and 50 mc. via the endolymphatic route.

In the light of present knowledge and practice, then, the doses administered to our patients appear to meet a reasonable standard of safety. Moreover, we could probably improve the therapeutic efficacy of endolymphatic radiogold without undue risk by increasing our administered doses in selected cases to something in the order of 35 to 60 mc. The small risk entailed would be far outweighed by the fact that the patient has a potentially lethal neoplasm of a type likely to spread by lymphatic pathways.

SUMMARY

Endolymphatic colloidal radiogold was given 17 times to 14 patients with malignant melanoma of the extremities or squamous carcinoma of the genitalia. The rationale and limitations of treating small metastases in regional nodes by this method are discussed. The technique is described with emphasis on avoidance of local complications.

Radiation doses were tabulated in 189 lymph nodes obtained in nine node dissections. Whole blood and plasma levels of colloidal gold and urinary excretion of colloidal gold were determined after therapeutic endolymphatic injection.

Endolymphatic radiotherapy with colloidal gold can be carried out with minimal local and systemic effects and without undue hazard to the patient. While radiation doses in excess of 30,000 beta rads have been achieved frequently with current methods, many lymph nodes receive less than therapeutic doses. Possible means of increasing radiation doses to regional nodes are discussed. For the present we believe that endolymphatic administration of colloidal radiogold should be used only as an adjunct to node dissection.

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RÉSUMÉ

Administré par voie lymphatique, l'or colloïdal radio-actif est filtré par les ganglions lymphatiques régionaux. A l'intérieur du ganglion, des particules bêta de faible portée exercent un effet destructeur sur le tissu lymphoïde et les petites tumeurs qui

y sont incluses. La peau est bien protégée parce qu'elle est très éloignée du ganglion.

Les auteurs exposent leur expérience personnelle des 17 injections intralymphatiques d'or radio-actif effectuées chez 14 malades souffrant de mélanome malin des extrémités ou d'épithélioma spino-cellulaire des organes génitaux. Ils exposent leur technique et donnent des conseils pour éviter les complications, tant locales que générales.

Ils classifient les résultats des doses de radiation appliquées sur 189 ganglions lymphatiques à partir de neuf dissections ganglionnaires. Les doses de radiation très supérieures à 30,000 rad bêta ont été fréquemment données, mais plusieurs ganglions n'ont reçu que des doses inférieures aux doses thérapeutiques.

L'injection intralymphatique d'or colloïdal radio-actif ne devra être considérée que comme un adjuvant de la dissection ganglionnaire. La substance colloïdale ne pénétrera pas dans les ganglions qui sont obstrués par des métastases et la portée des particules bêta est trop courte pour pouvoir traiter efficacement de gros dépôts secon-daires.

SPONTANEOUS RUPTURE OF ESOPHAGUS

In nearly all of the described cases of spon-taneous rupture of the esophagus, vomiting for one reason or another was the exciting cause of the rupture. Fundamentally, the esophagus is suddenly filled by gastric content expelled in an upward direction through the relaxed cardia by forcible contraction of the diaphragm and the muscles of the abdominal wall. It would appear that gastric retroperistalsis plays a minor part, but Moynihan points out that there is contraction of the pyloric end of the stomach. Mackler suggests that, in cases in which there is no evidence of stricture in the upper part of the esophagus, spasm may play an important part in causing a build-up of intraesophageal pressure to the critical point at which rupture takes place.

In the previous report on the patient de-scribed in this paper, it was suggested that spasm of the cricopharyngeus was an important factor. It is noteworthy that no local abnor-mality was demonstrated after her previous perforation when a barium swallow was given some two months later. On this occasion, how-ever, when this examination was repeated, the appearances suggested the presence of a post-cricoid web, but this could not be demon-strated by the later esophagoscopy.

It is of great interest that in a few cases of proven rupture, vomiting was not a feature. In Davidson's case of rupture of the hypo-pharynx, a bolus of soft, steamed fruit pudding seemed to stick in the throat with the sudden onset of intense pain and almost instant aware-ness of a swelling in the left side of the neck, followed by the rapid appearance of surgical emphysema. At operation the patient was found to have a 3-cm. long split in the left posterolateral wall of the pharynx extending upwards from the cricopharyngeus. There was some dark-coloured fluid and pudding in the edematous tissues around the pharynx. Nanson and Walker's patient had pain while swallow-ing a piece of soft Swiss roll and did not vomit until several hours later, when she brought up some food and a small quantity of blood.

In three of four of Anderson's cases perfora-tion occurred without vomiting. In his first case a bolus seemed to stick while swallowing. Another of his patients was quite remarkable as he apparently noticed swelling of the eye-lids with generalized puffiness of the face due to surgical emphysema when eating in a café. This was followed a short time later by severe cramping upper abdominal pain. Vomiting was not a feature. At subsequent thoracotomy he was found to have a rent in the esophagus 1 cm. above the diaphragm.

This patient gave an antecedent history of some difficulty in eating and swallowing during the preceding three weeks and Anderson's other two patients had had some previous digestive disturbance. He stressed that in true spontaneous rupture of the esophagus no dis-ease process, either gross or microscopic, is demonstrable at the site of the tear. He stated that changes which are seen are often thought to be the result of the rupture, but it may well be that more often than not they are its cause. In his cases it would certainly appear that some element of esophagitis might have been present. Numerous other authors have stressed the importance of the association of spontaneous perforation of the esophagus with peptic ulceration in the stomach or duo-denum and have even labelled the condition "acid peptic perforation".—Russell, J. Y. W. and Macdonald, N.: Spontaneous rupture of the oesophagus, *Brit. J. Surg.*, **55**: 311, 1968.

PANCREATIC ANNULUS: A SIGN OR A CAUSE OF DUODENAL OBSTRUCTION?*

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THIS communication will describe five consecutive instances of duodenal obstruction in infants, associated with pancreatic annulus. In four, it was demonstrated that the obstruction was not due to the annulus. This observation is in keeping with clinical findings in accessory or heterotopic pancreatic tissue in adults, which characteristically does not produce obstruction or symptoms. We propose that an annulus is the physical sign of partial failure of growth in a duodenal segment, and that the annulus itself probably does not cause obstruction.

Fig. 2.—Case 1. The supine film shows the extent of gastric dilatation, and the flat acetabular angles of Down's syndrome.

CASE REPORTS

Case 1.—The patient, a male newborn weighing 6 lb. 2 oz., was the product of a pregnancy with hydramnios. At 3 days of age he had not passed meconium, and had vomited fluid that contained blood flecks but no bile. In appearance, he was obviously mongoloid.

An abdominal radiograph showed a single bubble due to a distended stomach and acetabula typical of Down's syndrome (Figs. 1 and 2). At laparotomy the stomach was grossly distended and the first part of the duodenum was found to diminish to the size of cotton thread. There was malrotation of the midgut. The cecum was in the right upper quadrant and the small-bowel mesentery was not fixed. When the cecum was separated from the thread-like atretic duodenum (Ladd's procedure), an annular pancreas was found that only partly occupied this area. The jejunum contained bile stained meconium. By anastomosing the jejunum to the first part of duodenum, the atresia was bypassed. The child is well six years after operation.
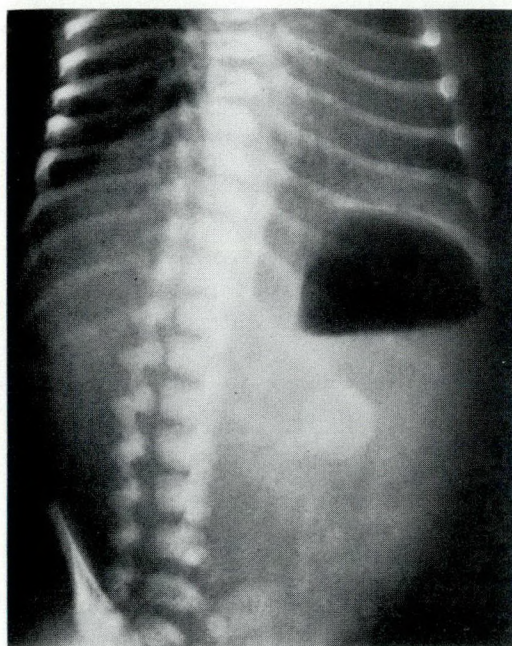

Fig. 1.—Case 1. An upright film shows dilatation of the stomach, which was due to complete atresia of the first part of the duodenum. Annular pancreas surrounded the second part of the duodenum. Umbilical cord stump shows as a round opacity.
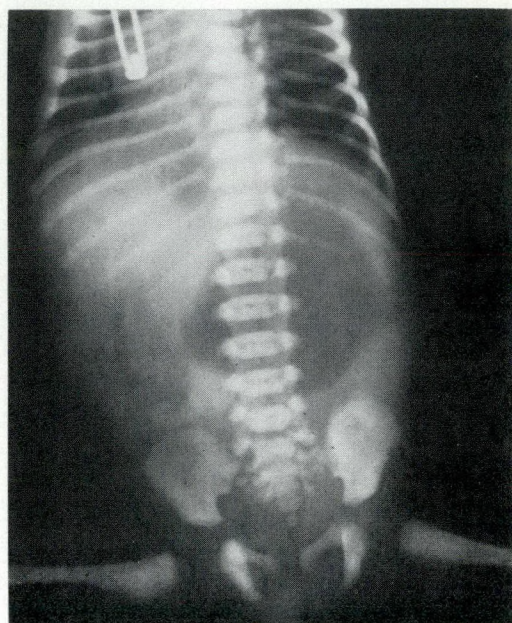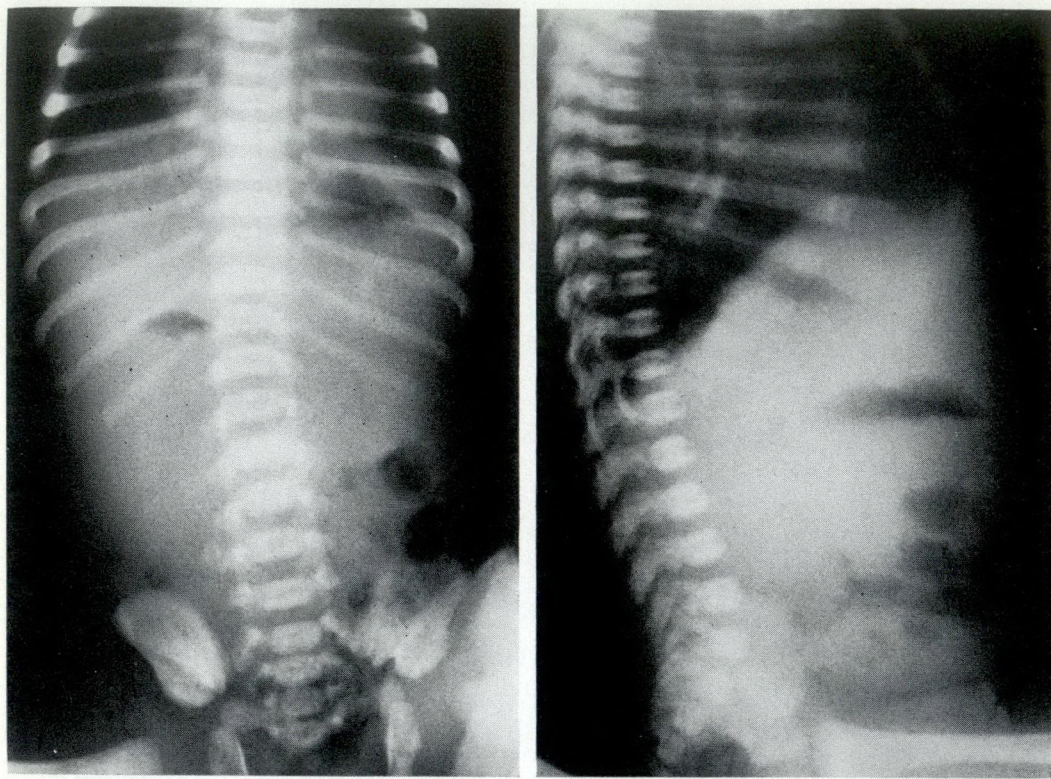
Fig. 3.–Case 2. Upright films show the characteristic "double-bubble" appearance of gastric and duodenal distension. Some gas is present in the lower intestine. Pre and post-ampullary obstruction was due to two internal diaphragms. The intervening patent unobstructed duodenum was encircled by an annular pancreas.

In this child, the cause of the duodenal obstruction was complete atresia of the duodenum. The annular pancreas was a coincidental finding.

Case 2.—A 5-day-old male infant, birth weight 6 lb. 13 oz., was the product of a normal pregnancy and delivery. He had persistently vomited bile stained fluid. On examination he was dehydrated and had upper abdominal distension. Radiographs demonstrated the "double bubble" of a dilated stomach and megaduodenum. Some air noted distally in the intestine indicated that the obstruction was incomplete (Fig. 3).

At laparotomy the stomach, pylorus and first part of the duodenum were grossly distended. A large stomach tube was inserted and met a diaphragmatic obstruction at the beginning of the second part of the duodenum; at this point there was a cone-like gradual narrowing. Malrotation of the intestine was also present. When the cecal attachment was divided and brushed aside, the duodenum

was seen proceeding inferiorly in the right abdomen. A catheter passed upwards through the narrow third part of the duodenum met a second diaphragm. The area between the two diaphragms was smaller but of relatively normal size. This segment was covered by an "annular pancreas". Duodenojejunostomy was done to bypass the obstruction. This child is alive and well 3½ years after operation.

The child had both an annular pancreas and malrotation with bands. However, the intestinal obstruction was due to two internal diaphragms.

Case 3.—A 7-day-old female infant was admitted with vomiting and diarrhea which subsided in 48 hours. Vomiting recurred, but was not bile stained. When it became projectile over the ensuing week, it was presumed to be due to pyloric stenosis.

At laparotomy, the pylorus was not dilated. There was, however, a volvulus of the midgut and failure of fixation of the small-bowel mesentery. The ileocecal junction was just

Fig. 4.—Case 4. A 1½-year-old infant with Down's syndrome showing the cardiac outline of congenital heart disease and double bubble of duodenal stenosis. Clearly evident on the upright film is a large megaduodenum. The annular pancreas only partly filled the indentation of a duodenal stenosis.

Fig. 5.—Case 4. The supine film corresponding to Fig. 4 shows the gross gastric dilatation that accompanies megaduodenum.
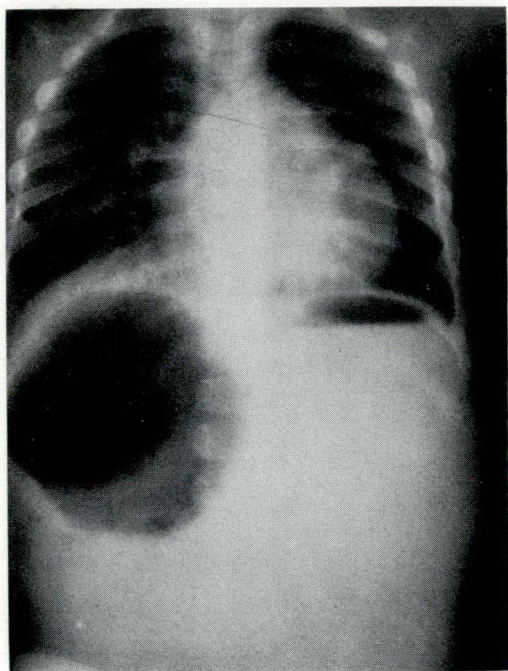
under the gallbladder and a broad sheet of peritoneal attachment, which passed from that area to the posterior abdominal wall, had obstructed the duodenum. When this sheet was divided, an annular pancreas was found surrounding the second part of the duodenum. The obstruction appeared to be completely relieved. A large stomach tube was passed through this segment to confirm that there was no intrinsic obstruction. The child is alive and well six years after operation. No duodenal obstruction is evident clinically now.

In this child, the annular pancreas was an incidental finding. The obstruction was due to peritoneal bands associated with malrotation of the midgut.

Case 4.—This female infant, the only child of a 42-year-old primigravida, was known to be mongoloid when she was admitted at 1½ years of age. She had the classical facies, hands, dermatoglyphics, sacral pigment spot, atrial septal defect and the trisomy-21 of Down's syndrome. The child had not been able to tolerate solid foods because each time such food was tried it was vomited.

The child had gross abdominal distension, was dehydrated, and in collapse. Rehydration, replacement of electrolyte losses and gastric suction resuscitated the youngster. Radiological examination revealed a typical double bubble of duodenal obstruction (Figs. 4 and 5).

At laparotomy, there was malrotation of the midgut; the cecum was in the right upper quadrant. The small intestine was not fixed and, when the sheet of mesentery that attached the cecum to the right upper posterior abdominal wall was divided, a duodenal stenosis approximately 1 cm. in width was discovered. An annular pancreas only partly occupied this indentation. A duodenoduodenostomy was done to bypass the obstruction. After operation the wound reopened, a complication that we attributed to the infant's hypoproteinemia. She has gained weight and is well two years after operation.

In this child the annular pancreas occupied only part of the indentation that produced the duodenal stenosis.

Case 5.—The patient, a first child, was a male who weighed 6 lb. 12 oz. at birth. When first discharged from hospital, he vomited occasionally. The vomiting increased gradually during the following month, until all feedings
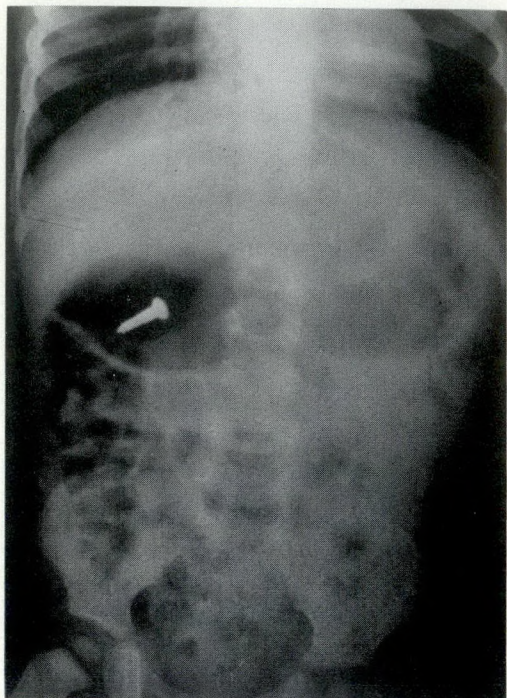
Fig. 6.—Case 5. A gas-containing megabulbus showing a wood screw in its lumen. Gas is present in the stomach and normally distributed in the intestine.

Fig. 7.—Case 5. The barium-filled duodenal bulb shows a rounded distal margin. The pylorus is also dilated and barium has passed into the small bowel.

were forcefully regurgitated. On examination he was dehydrated, had a depressed fontanelle and was emaciated. His abdomen was distended and epigastric peristalsis could be seen. After drinking a sugar solution he vomited forcefully, and the examiner thought he could palpate a pyloric mass. The clinical diagnosis of congenital hypertrophic pyloric stenosis was made. At laparotomy no pyloric thickening was found. The small bowel was explored but no abnormalities were found. A Ramstedt operation was done and up to the age of 7 months he appeared to improve.

He then began to burp foul gas, and occasionally vomited large amounts of coffee-ground material. Medical treatment did not benefit him. These symptoms continued for a year without retardation of growth. An upper gastrointestinal radiographic series at 20 months of age showed marked dilatation of his stomach and first part of the duodenum in a "bubble", which in appearance resembled that described in annular pancreas. The inferior margin of the distended duodenum was smooth and rounded. Gas distribution was normal through the remainder of the bowel and barium passed through the megabulbus. A 1½-inch wood screw was also seen in various positions in the stomach and on radiographic

examination one week later it had not passed through (Figs. 6 and 7).

At laparotomy the stomach and first part of the duodenum were markedly dilated. A band of pancreatic tissue was felt overlying the anterior aspect of the second part of the duodenum in the region of the ampulla of Vater. The screw was removed through a gastrotomy. The duodenal lumen was not probed from below so the exact cause of the obstruction was not confirmed. A small hiatus was then made through the transverse mesocolon, and through this the jejunum was anastomosed to the dilated duodenum. Such anastomosis brings improvement because it relieves both intrinsic and extrinsic duodenal obstruction. Convalescence was uneventful, and this patient has remained well for 3½ years.

RADIOLOGIC FEATURES

When complete atresia of duodenum is present, the intestinal lumen sometimes ends at the level of a mass of pancreatic tissue. This variety of anomaly is sometimes confused with annular pancreas. However, duodenal atresia also occurs most often near the level of the ampulla of Vater.

As a consequence, bile duct anomalies are often associated with such atresia. Radiologic changes described in patients with the incomplete atresias of the duodenum are indistinguishable from those described for annular pancreas. We doubt that the latter is a clinically meaningful condition and consider that the duodenal lesion is primary.

The criteria common to these radiologic descriptions are: (1) Megabulbus or megaduodenum with a smooth, rounded, inferior margin. (The finding of megaduodenum is seldom given the clinical significance it deserves, partly because we lack suitable norms for reference. In adults, a duodenal bulb of more than 4 cm. in diameter is definitely abnormal but, in infancy, enlargement has to be evaluated by its relative size as compared with the lumbar vertebrae.) (2) A simultaneous dilatation of the stomach producing a characteristic double bubble. The duodenum often appears much the larger chamber in the upright view but not in supine films (Figs. 4 and 5). (3) Extensive gastroduodenal peptic ulceration may be present. (4) A "writhing duodenum" may be seen at fluoroscopy when peristalsis is provoked.

Most individuals with an annular pancreas are completely without symptoms. We now wish to show that when obstruction occurs it is seldom due to the presence of a pancreatic annulus, but to a ring-like duodenal stenosis. In these patients the ventral pancreas remains in its initial (fetal) position and indicates that duodenal development has been incomplete.

Developmental Anatomy

"Rotation of the ventral pancreas" is the process that leads to fusion of the two components that form the pancreas. It is worth reconsidering the type of failure in sequential duodenal development that leaves the primitive ventral lobe in its initial position. In humans, pancreatic tissue arises from several different segmental primordia, at different levels scattered over the pyloroileal intestine. All except two remain minute. The larger of the two lies in the dorsal primitive mesentery of the duodenum. Eventually it forms the bulk of the gland, and is arranged around the duct of Santorini. The other pancreatic bud is smaller and grows into the ventral mesentery of the duodenum. The duct of this bud is Wirsung's duct. This smaller component is the most inferiorly placed of a contiguous cluster of three buds. These form the hepatic duct, gallbladder and fetal ventral pancreatic lobe respectively. Because of this contiguous origin they share an exit at the ampulla of Vater.

During the growth in circumference of the duodenum, the ampullae for the ducts of Santorini and Wirsung separate relatively little on the right side during the growth of the duodeum. Even in adult life, the average distance between these ampullae is only 2 cm. Meanwhile, as the circumference of the duodenum increases, the whole duodenum elongates forming a ventral loop. This falls upon its right side to fuse with the posterior abdominal wall.

By selective expansion or growth of the left side of the duodenal circumference, the duct of the former ventral pancreatic component comes to lie more to the right, and posteriorly. Finally it lies behind the terminal part of the duct of Santorini (Fig. 8). The two ducts now unite at right angles between the tail of the smaller duct of Wirsung, where it crosses the larger duct of Santorini. In this manner, the smaller duct of Wirsung eventually becomes the terminal outflow portion for the entire pancreas. The pancreas is now formed by a fusion of the large dorsal pancreas with a smaller component which originally lay ventrally.

The duct of Santorini disconnects at its ampullary (duodenal) end in 50% of the population. This segment becomes the narrowest part of the duodenum in adults. Even when it remains patent, the duct of Santorini can only substitute functionally for Wirsung's duct in 20% of the population. In 8%, the ampulla alone becomes solid. Flow in this portion of the duct is then retrograde into Wirsung's duct. Radiologically, the progressive narrowing of this duct, as it approaches the duodenum, can be demonstrated.

Semantically, this complex process is called "a rotation of ventral pancreas". It is a passive displacement due to selective
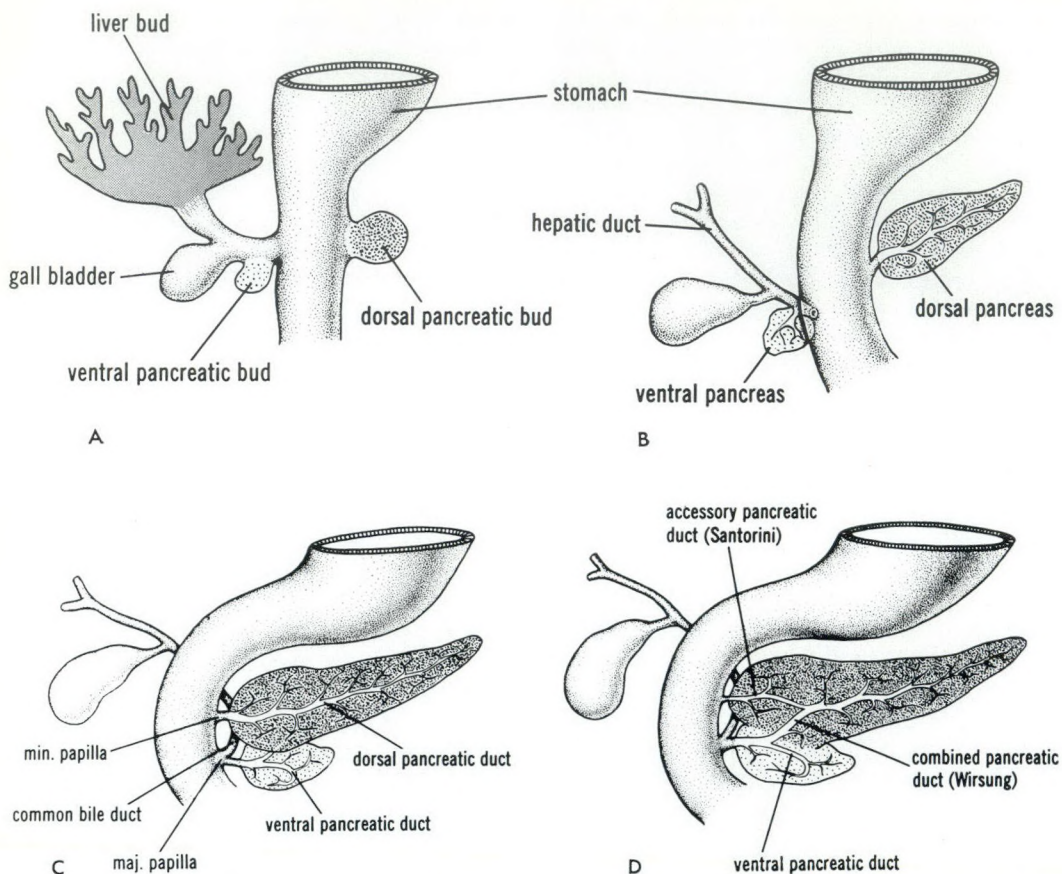
Fig. 8.—Line drawings showing the sequence of events in the developmental anatomy of the pancreas. A smaller and more distal pancreas forms in the ventral mesentery and "rotates". It fuses with the dorsal pancreas and provides the terminal part of the outflow duct. Drawn to scale, the duodenum in "A" would be much smaller than in "D". (Reproduced by permission from Langman, J.: Medical embryology; human development—normal and abnormal, The Williams & Wilkins Company, Baltimore, 1963.)

expansions of the duodenum altering the position of the pancreatic body.

In a careful retrograde cholangiographic investigation of 200 pancreatic ducts, Millbourn[1] found two instances of annular pancreas. In both, the duct supply of the annulus belonged entirely to the Wirsung system. In the ventral pancreas, the ring lay on the anterior and right aspects of the duodenum. No variations from this distribution have been recorded. Such a pancreatic "annulus" is therefore a sign that selective overgrowth of the left side of the fetal duodenum has not occurred at the levels of the two ampullae. In other words, it is a sign of hypoplasia of the duodenum.

DISCUSSION

We now wish to review the evidence

put forward to suggest that annular pancreas may be obstructive by bulk or position. It is well recognized clinically that pancreatic heterotopias and accessories are characteristically asymptomatic.[2] Those symptoms ascribed to them in earlier reviews[3] belonged to concurrent conditions. Only very rarely, when they form a large polyp or duct retention cyst, do they appear to obstruct.[4]

Difficulty in mobilizing the normal duodenum from the head of the pancreas is also a common surgical experience. This fixation is quite normal. It is the reason why carcinoma of the pancreas is often impossible to detect by palpation. As shown by Kirk[5] in 1944, the fixation is due to the presence of nodules of pancreatic tissue sandwiched between the muscle coats of

Fig. 9.—Section through the normal duodenal attachment of the head of the pancreas, from an adult without any evidence of obstruction. Gland lobules and ducts are interspersed throughout the muscle wall.

the duodenum, but continuous with the main gland. In addition, there is further normal fixation and union. Tiny blind tributaries of the extraduodenal portion of the bile duct sometimes penetrate through the muscle wall of the duodenum from without. They pass into the duodenal mucosa surrounded by coats of circul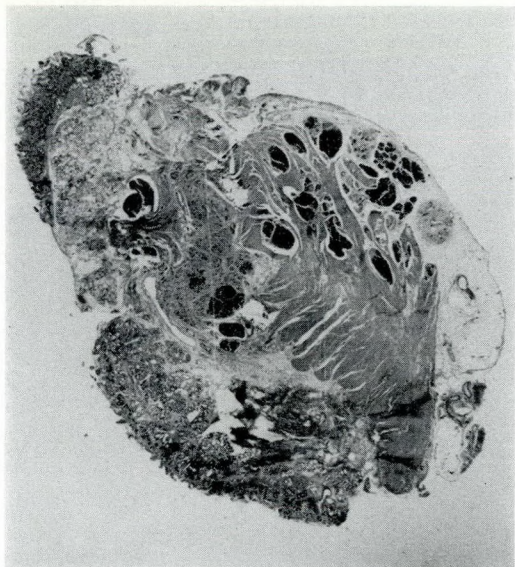ar muscle. There is no evidence that any of these firm anatomical attachments, which are seldom described in standard textbooks of surgical anatomy, are obstructive clinically (Fig. 9).

It is also well recognized that, in the majority of patients, annular pancreas is asymptomatic. It may appear to become obstructive when the patient is well through the growth period, but sometimes not even until adult life.[6, 7] The zone of narrowing is often described as smooth in outline, short and almost "membranous". Should obstruction be sufficiently far proximal to prevent reflux of alkaline pancreatic secretions, peptic ulceration in the megabulbus commonly develops.[8, 9] This complication would seem a more likely cause of delayed symptoms than delayed "congenital obstruction". In some infants the ring narrowing incorporates the ampulla of Vater producing obstructive jaundice. According to

Stowens,[10] when the condition causes duodenal obstruction, an inflammatory reaction is present in the annular tissues. This is the only reference to annular pancreatitis which we could find.

Experience with surgical treatment is interesting. Ramstedt's procedure commonly fails in pancreatic annulus. As Hunter[11] noted, "in addition, division of the pancreatic ring may not relieve the obstruction because of the possible intermingling of the duodenal musculature and pancreatic tissue". We have already seen that such intermingling is normal in the concavity of the duodenal loop. Surgical opinion now opposes division of the annulus, preferring duodenojejunostomy. Gastroenterostomy may not reduce the duodenal size and carries with it the risk of stomal ulceration.

Some 70% of reported cases of pancreatic annulus have had associated anomalies such as tracheoesophageal fistula, malrotation of intestine, congenital heart disease and imperforate anus. The association with mongolism is also recognized.[12] Of our five cases, four had malrotation, two had mongolism and one had multiple defects. The proportion of these patients who have chromosomal abnormalities is unknown. In our Case 3, the obstruction to the second part of the duodenum was removed by dividing a high cecal attachment to the posterior abdominal wall of the region. Two ring atresias of the duodenum were demonstrated in Case 2. The intervening duodenum—the portion covered by the pancreatic annulus—was of relatively normal size.

Gross[13, 14] pointed out that some infants have an atresia of the duodenum at the level of the annular pancreas. In analysis of combined intrinsic duodenal obstruction and malrotation, Knutrud and Eek[15] noted that "these facts seem to suggest that the obstruction due to annular pancreas is caused by a true atresia or stenosis". From the Mayo Clinic, Longo and Lynn[16] reported 29 patients with congenital duodenal obstruction. Seven had annular pancreas. Of these only three had vomiting as a prominent symptom; the others had symptoms referable to associated anomalies.

The accumulated evidence—embryological, clinical, and that from our own sur-

gical studies—strongly suggests that annular pancreas should seldom be thought of as the obstructing lesion. The basic condition is a failure of duodenal development and narrow segmental hypoplasia. Persistence of the pancreas in its fetal position is only its sign.

SUMMARY

In four out of five consecutive instances of duodenal obstruction in infants with pancreatic annulus, it was demonstrated that the obstruction was not due to the annulus *per se*. The true causes of obstruction included complete atresia of the duodenum, internal diaphragm and peritoneal bands associated with malrotation. Pancreatic annulus was only an incidental finding.

This fits well with the clinical experience of accessory and heterotopic pancreatic tissue in adults, which is characteristically non-obstructing and asymptomatic. Pertinent embryological, surgical and radiological evidence has been reviewed, suggesting strongly that annular pancreas should seldom be thought of as the obstructing lesion. It is of interest that the radiologic changes described for incomplete atresia of the duodenum are indistinguishable from those described for pancreatic annulus. Pancreatic annulus persisting in its fetal position is more reasonably regarded as a sign of failure of segmental duodenal development.

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RÉSUMÉ

Il est improbable que le pancréas annulaire puisse, en soi, entraîner l'occlusion intestinale. Celle-ci ne survient que dans les cas où coexistent de l'atrésie ou de la sténose duodénales ou un vice de rotation. Dans quatre des cinq cas d'occlusion intestinale que nous avons eu l'occasion d'observer chez le nourrisson souffrant de pancréas annulaire, l'occlusion n'était pas causée par l'anneau lui-même. Parmi les causes profondes d'occlusion, figuraient une atrésie complète du duodénum, la présence de diaphragmes internes et de pont fibreux du péritoine coïncidant avec un vice de rotation. La présence d'un anneau pancréatique n'était qu'une découverte fortuite.

Cette conclusion corrobore parfaitement l'expérience clinique qu'on possède sur le tissu pancréatique accessoire et hétéro-topique chez l'adulte: toutes les structures sont typiquement asymptomatiques et ne provoquent pas l'occlusion. Au point de vue radiologique, les images d'atrésie incomplète du duodénum ne peuvent être distinguées de celles qu'on décrit pour le pancréas annulaire. Il est plus raisonnable de considérer l'anneau pancréatique, persistance anormale de la position fétale, comme un signe d'arrêt de développement d'un segment duodénal.

EFFECT OF SMALL-BOWEL BYPASS ON GASTRIC SECRETION IN OBESE PATIENTS*

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IN the postoperative period, patients who have had extensive small-bowel resection often have gastric hypersecretion.[1-4] The hypersecretion is marked, ranging from 2.5 to 9.0 l./day, and contains up to 100 mEq./l. of hydrochloric acid. Moreover, in patients who have had gastrectomy previously, or who have it subsequently, the hypersecretion abates and they are easier to manage, particularly with regard to water and electrolyte balance.[5] Many experimental studies have been done which support these observations.[1-6] In dogs with Heidenhain pouches that subsequently have approximately 75 to 80% of the small intestine resected, the gastric acid output increases by two to six times the control values. In dogs with a small-bowel bypass, gastric secretion is doubled; in these animals, 75 to 80% of the bowel is excluded and the defunctioned bowel is either drained back into the colon[5] or excluded as a Thiry-Vella fistula.[6] According to some workers,[4] gastric hypersecretion increases directly with the amount of bowel resected.

The surgeon who is considering small-bowel bypass as a treatment for massive obesity will find these data of great interest. If such patients had gastric hypersecretion to the same degree as those who underwent intestinal resection, and if laboratory experience with Heidenhain pouch dogs is applicable to the human subject, bypass operations might well be followed by a high incidence of gastrointestinal complications. The duodenal ulcer diathesis might be provoked, and peptic esophagitis, duodenal and gastric ulcers develop.

In order to ascertain whether gastric hypersecretion developed in patients after small-bowel bypass, we did studies of gastric secretion in nine women, before and after operation.

EXPERIMENTAL METHODS

Nine obese women between the ages of 22 and 55 (mean 42 years) and weighing from 250 to 390 lb. were chosen for this study. Gastric secretion studies were performed to determine whether they were candidates for the bypass operation. Approximately three hours after a light, clear-fluid evening meal, a nine-hole gastric tube was passed through the nose and into the stomach and its position checked on the radiograph. The tube was fastened firmly in place and the patient was returned to bed. During a 12-hour period (overnight), the stomach was aspirated continuously and the gastric juice collected. In the morning, after the 12-hour collection was complete, a Histalog (3-beta-aminoethylpyrazole dihydrochloride; Eli Lilly and Company, Indianapolis, Ind.) gastric analysis was done. A 30-minute control specimen representing basal secretion was first collected, and then 100 mg. of Histalog was injected intramuscularly. Subsequently, the stomach was aspirated at 15-minute intervals for 90 minutes. In all specimens the following measurements were done: volume, pH, acid concentration (mEq./l.) and the amount of acid (mEq. per specimen). The highest hourly acid output and concentration were calculated and recorded.

The small-bowel bypass was constructed by joining the proximal 10 inches of jejunum distal to the ligament of Treitz end-to-end to 20 inches of distal ileum just proximal to the ileocecal valve. To provide drainage, the bypassed bowel was closed proximally and joined end-to-side to the colon.

In the postoperative period the gastric analyses were repeated. This paper presents the results of analyses done from four to six months after operation. Serum
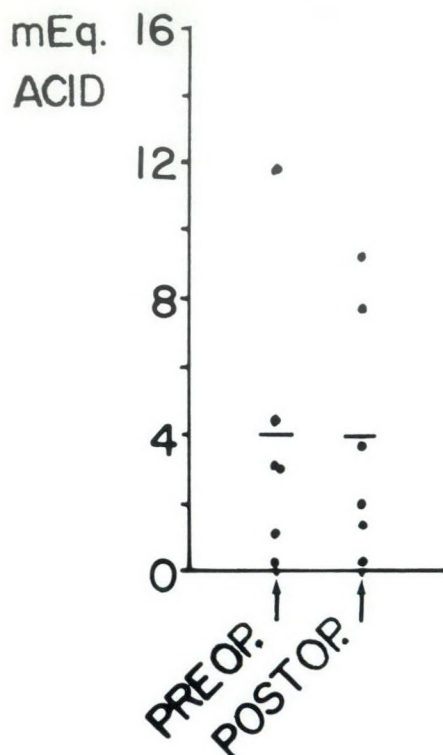
Fig. 1.—The results of 12-hour collections of un-stimulated gastric juice, before and after opera-tion. Gastric hydrochloric acid is measured in mEq. per collection.

electrolytes, proteins and hemoglobin levels were measured within a few days of pre-operative and postoperative gastric analy-sis. They were all within normal limits.

RESULTS

Fig. 1 shows the data from the 12-hour gastric aspirations. The means of the pre-operative and postoperative periods are similar; there was approximately 4 mEq. of acid in each collection. In the preopera-tive and postoperative values in individual patients, there were no major shifts in acid concentration.

Fig. 2 shows the results of gastric analy-sis after Histalog stimulation. Data from Breuer and Kirsner,[7] showing values in normal patients and in patients with duo-denal ulcer, are incorporated for compari-son. The means of data from the preopera-tive and postoperative periods are similar, about 16 mEq. of acid per hour. These values are only slightly greater than the
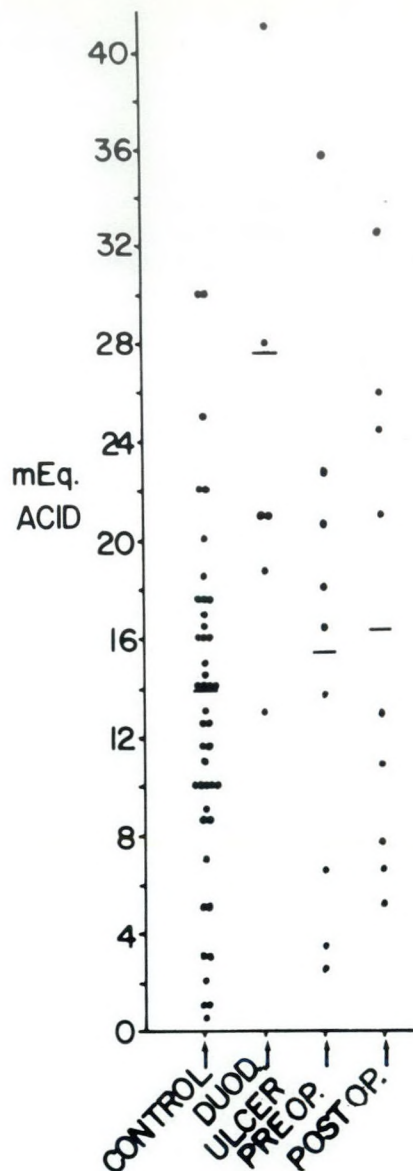
Fig. 2.—Mean and ranges of acid secretion in bypass patients, before and after operation, con-trol values in normal patients, and in patients with duodenal ulcer. The mean values in the experi-mental group and in the controls fall in the same range.

mean reported for control patients (14 mEq./hr.), and much below the mean value in patients with duodenal ulcer (28 mEq./hr.).

The mean concentration of hydrochloric acid determined for basal secretions and Histalog-stimulated secretions in the pre-operative and postoperative periods is
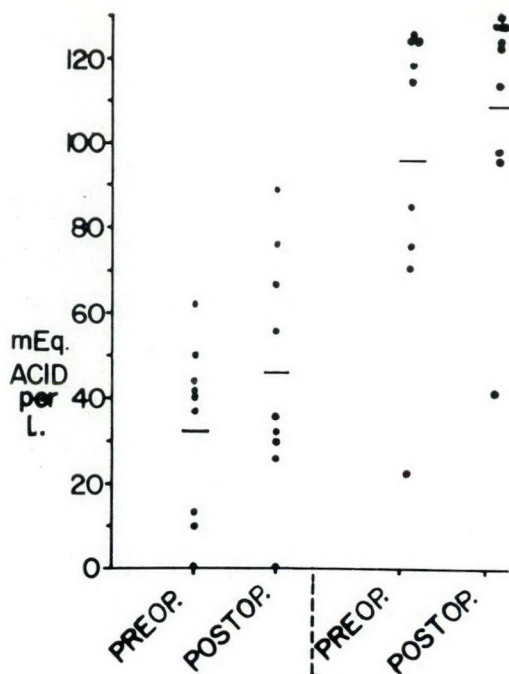
Fig. 3.—In the study illustrated in this figure, 30 minutes were allowed for the collection of basal secretions. Acid concentration in basal and Histalog-stimulated secretion are shown for the preoperative and postoperative periods. There is a slight increase in concentration after bypass.

shown in Fig. 3. The mean basal secretion increased from 32 to 47 mEq./l. Similarly, the mean acid concentration in the Histalog-stimulated specimens increased from 98 to 107 mEq./l. One patient who had no basal acid secretion before operation had none after operation. However, she was able to secrete acid under Histalog stimulation although her values were the lowest recorded in this group of patients.

DISCUSSION

Our data suggest that intestinal bypass in obese patients does not produce any detectable increase in total acid secretion, at least as measured by the tests used in this study. This conclusion was confirmed when these data were submitted to statistical analysis (t test). When preoperative and postoperative levels are compared, there is little variation between total acid in unstimulated secretions, basal secretions and under Histalog stimulation. Basal and Histalog-stimulated acid concentrations re-

sembled those obtained in normal controls. The acid concentrations throughout were well below those reported in patients with duodenal ulcer.

In line with these data, no changes were observed when the postoperative upper gastrointestinal radiographs in these women were compared with those made before operation. Furthermore, none of these patients had complaints suggestive of gastric hyperacidity.

The difference between these results and those reported in the literature in patients undergoing intestinal resection remains to be explained. In comparing the two situations, the method of resection of intestine may be of the greatest importance because, in patients undergoing bypass operations, the excluded bowel is retained. For this reason, the anatomic and physiologic conditions in these two groups of patients are not entirely comparable. Other factors may also be significant. Small-bowel resection is usually performed in patients who have sustained an embolus of the superior mesenteric artery, volvulus of the gut or some other serious insult. They have usually undergone an emergency operation and usually have had no preoperative measurement of gastric secretion. Furthermore, when the intestinal resection is done following vascular obstruction, residual arterial disease or obstruction may be producing ischemia of the remaining gastrointestinal organs.

Other factors may explain the differences noted between patients with resection and those with bypass. In the former, gastric secretory studies were not done after the sixteenth postoperative day because, by that time, all had died from sepsis, electrolyte imbalance and further intestinal infarction.[1-4]

It seems unlikely that gastric secretions collected during a complicated and stormy postoperative period would be representative, or that alterations observed can be attributed to intestinal gastric-stimulating or inhibiting factors. The gastric hypersecretion in these patients is probably due more to the trauma and stress of the postoperative period than the intestinal resection.

SUMMARY

Preoperative and postoperative gastric secretory studies on nine patients undergoing a small-bowel bypass operation are presented. Little change was observed in the mean total acid secreted in unstimulated, basal and Histalog-stimulated specimens. There was a slight increase in the mean acid concentration.

The mean total acid values in both preoperative and postoperative patients resembled those of normal control patients reported by other authors. The values were well below those reported for patients with duodenal ulcer.

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RÉSUMÉ

La littérature rapporte qu'une résection étendue du grêle est suivie, chez certains malades, d'une forte hypersécrétion gastrique. Par ailleurs, les expériences effectuées sur des animaux porteurs de fistule gastrique de Heidenhain indiquent une forte augmentation de la sécrétion dans le petit estomac après résection du grêle. Le mécanisme exact de cette hypersécrétion gastrique n'est pas encore aujourd'hui clairement compris. Les auteurs estiment que les patients qui ont subi une dérivation de 80 à 85% de l'intestin grêle entre le ligament de Treitz et la valve iléocécale peuvent présenter de l'hypersécrétion gastrique.

Des études de la sécrétion gastrique, avant et après dérivation, ont été faites chez neuf femmes obèses. Dans le suc gastrique, recueilli après la nuit, et après analyse gastrique avec l'Histalog, on a dosé le HCl total et la concentration de HCl.

Les résultats de l'analyse gastrique ont mis en lumière, après la dérivation, de légères augmentations des sécrétions gastriques, tant chez celles qui n'avaient pas été stimulées que chez celles stimulées par l'Histalog. Toutefois, ces données, soumises au t test, n'ont guère montré de différences, sur le plan statistique, entre les chiffres pré- et post-opératoires.

On peut donc supposer que le phénomène de l'hypersécrétion gastrique ne s'est pas produit parce que l'intestin grêle, privé de ses fonctions, a été conservé et n'a pas été réséqué. Il n'empêche que le mécanisme exact sous-jacent à cette réaction n'est pas encore élucidé.

COMPLICATED AORTODUODENAL FISTULA

Aortoduodenal fistula is an unusual complication of abdominal aortic aneurysm. Warning hemorrhage often precedes a terminal exsanguinating hemorrhage. Peptic ulceration and esophageal varices remain a more common cause of massive gastrointestinal hemorrhage, but when these causes have been excluded in the elderly patient, fistulous communication between the bowel and the abdominal aorta or iliac vessels should be considered. In the presence of an abdominal aortic aneurysm, urgent investigation and laparotomy must be undertaken.

In order to prevent contamination of the operative field at the primary operation, resection of the third part of the duodenum or the bowel and the fistula should be undertaken *en bloc*.

In the patient described in this communication, it is considered that infection brought about disruption of the proximal suture line which resulted in a false aneurysm and a further fistula into the duodenum.

The discovery of caseating tuberculous foci adjacent to the wall of the aneurysm suggests the possibility that the fistula may in some way have been related to this disease. However, there was ample evidence to show that the main etiological factor responsible for the aneurysm was atherosclerosis.—Eadie, G. A. and Pollock, D. J.: A complicated aortoduodenal fistula: a case report, *Brit. J. Surg.,* **55**: 314, 1968.

MECKEL'S DIVERTICULUM: A 10-YEAR REVIEW OF 218 CASES*

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WHEN the human embryo is 6 weeks old, obliteration of the omphalomesenteric or vitelline duct, which connects the yolk sac to the apex of the midgut loop, begins.[1] If this process is incomplete, part of the duct persists as a Meckel's diverticulum. This developmental anomaly is situated approximately 100 cm. proximal to the ileocecal valve on the antimesenteric border of the ileum. It is about 5 to 10 cm. in length and 1 to 10 cm. in diameter and may be attached to the posterior aspect of the umbilicus by a fibrous band. Found in approximately 1.5% of the population,[2, 3] the diverticulum is three times more common in males than in females.

Complications of this developmental anomaly, still a significant cause of morbidity in children, occur in 50% of children before they are 2 years of age.[2, 4]

At the Hospital for Sick Children, Toronto, we reviewed our experience with this condition over the 10-year period 1955 to 1964, and compared our results with those previously reported by Wansbrough, Thomson and Leckey.[5] Two recent cases not included in our review are discussed in some detail because they illustrate the constant diagnostic challenge that this anomaly presents.

RESULTS

Two hundred and eighteen children with Meckel's diverticulum were seen during the period 1955 to 1964 at the Hospital for Sick Children. The diverticula were found incidentally at operation or autopsy, or were found at operation performed for some complication of the diverticulum, for example bleeding or obstruction. These cases are compared with the previously reported series in Table I.

In the present series, 60 of the 81 children with complications associated with

TABLE I.—MECKEL'S DIVERTICULUM AT THE HOSPITAL FOR SICK CHILDREN, TORONTO, SEEN DURING THE PERIODS 1912 - 1954 AND 1955 - 1964

Meckel's diverticulum	1912 - 1954 (273 cases)	1955 - 1964 (218 cases)
Uncomplicated (incidental at unrelated operation)*...........	120	97
Incidental autopsy finding........	33	40
Complicated (found at operation for the complication)...........120	{94 male, 26 female} 81	{60 male, 21 female}
Ratio of uncomplicated : complicated cases.........................	153 : 120	97 : 81
Complications: Bleeding...........	25	32
Perforation.........	11	5
Inflammation.......	7	6
Patent vitelline duct	17	14
Obstruction:....................	30	15
Intussusception.....	30	8
Incarcerated Littre's hernia....	0	1

*The finding at autopsy of Meckel's diverticulum should logically be counted as "uncomplicated diverticulum". However, in order to compare our results with the previous review,[5] we have followed Wansbrough's lead and kept them separate. Hence, "uncomplicated diverticulum" in this table refers to an uncomplicated Meckel's diverticulum found at operation.

Meckel's diverticulum were males. This is in keeping with the 3:1 male-female ratio encountered in this anomaly. The diverticulum was examined histologically in 79 of the complicated cases. The types of mucosa found are listed in Table II.

TABLE II.—TYPES OF MUCOSA FOUND IN 79 CHILDREN WITH COMPLICATED MECKEL'S DIVERTICULUM

	Number of children
Gastric............................	51
Gastric and pancreatic................	3
Normal ileum......................	20
Colonic...........................	5
	79

In 45 of the children with complicated disease, the diverticulum was excised and, in 34, the small portion of the ileum to which the diverticulum was attached was resected. Ileal resection was done in the presence of a large diverticulum which, if excised, would produce narrowing of the ileal lumen. In two children the diverticulum was associated with the leading point of an intussusception. In one child, after reducing the intussusception, the abdomen was closed and the diverticulum removed at an elective operation three months later.

In the other, the diverticulum, although near the apex of the intussusception, had a broad base and was not considered to be the leading point. It was not removed.

Twelve of the 218 patients (6%) operated upon had complications after operation. Four eviscerated between the fifth and seventh postoperative days and in four the incisions became infected. Two had intestinal obstruction. The first patient had incomplete obstruction and was treated conservatively; the second had complete obstruction secondary to volvulus and died of pneumonia following laparotomy.

Three of the four patients who eviscerated had had an ileal resection with removal of the diverticulum. Subsequently, their wounds were closed by secondary suture and they had no further problems.

In the earlier series at this hospital, 28 major postoperative complications developed following 260 surgical procedures. Most of these infants developed intestinal obstruction or peritonitis, but pneumonia, meningitis, incarcerated hernia and evisceration were also seen. Table III gives the mortality for successive decades.

TABLE III.—PERCENTAGE MORTALITY IN 10-YEAR PERIODS FROM 1924 TO 1964

Period	Percentage
1924 - 1933	53
1934 - 1943	26
1944 - 1953	10
1955 - 1964	2

At the Hospital for Sick Children the frequency of postoperative complications has decreased in the past 10 years. There have been 12 such complications in 218 surgical procedures, a rate of 6%. These have been less serious than those encountered between 1912 and 1954, and accounted for only one of the two deaths in this decade.

A brief account of the two infants who died is as follows:

The first patient—a 4-week-old infant—developed small-bowel obstruction. At operation, remnants of the omphalomesenteric duct were freed, the Meckel's diverticulum and a part of the ileum were resected. On the fifth postoperative day, the infant developed abdominal distension and was transferred to this hospital for further management. Volvulus of a large portion of the small intestine was relieved but the bowel remained devitalized. The devitalized intestine was resected and anastomosis performed but the infant died 10 hours later. At autopsy there was generalized peritonitis and the proximal anastomosis had perforated.

The second patient who died was an 8-month-old girl with a three-day history of intermittent vomiting and abdominal distension. On admission she was *in extremis* and had severe electrolyte imbalance. At operation an intussusception was found and reduced. At the apex of the intussusception was an invaginated Meckel's diverticulum which was subsequently shown to contain ectopic pancreatic tissue. This, along with a segment of ileum, was excised. The child died in the immediate postoperative period but consent for autopsy was not obtained.

CLINICAL ASPECTS OF MECKEL'S DIVERTICULUM

In the present series, bleeding—often severe, recurrent and exsanguinating—was the most common complication of Meckel's diverticulum. Sixty per cent of the children who bled were under 2 years of age and 60% of these had a hemoglobin of less than 8 g./100 ml. on admission. Similar findings were reported by Rutherford and Akers.[4]

Heterotopic gastric mucosa, found in 64% of diverticula, leads to local peptic ulceration, with subsequent pain, hemorrhage and perforation. It may also produce obstruction of the ostium of the diverticulum and secondary diverticulitis.

Three patients in our series were admitted for the investigation of rectal bleeding. With conservative treatment, bed rest and blood transfusions, the rectal bleeding ceased. Sigmoidoscopic examination and barium enemas were negative and the patients were discharged with the final diagnosis of "bleeding Meckel's diverticulum". Subsequently, all three patients bled again from the rectum 1 month to 2½ years later. In each, the diverticulum was resected and pathological examination of the specimen revealed gastric mucosa and peptic ulceration.

Second in frequency of the complications

associated with Meckel's diverticulum in our series was intestinal obstruction, most commonly due to congenital bands and volvulus; intussusception was the next most common cause. Worthy of note in this respect are the mesodiverticular bands, fibrous bands of tissue joining the tip of the diverticulum to the mesentery and containing remnants of the left or right vitelline arteries (Fig. 1). Around this free
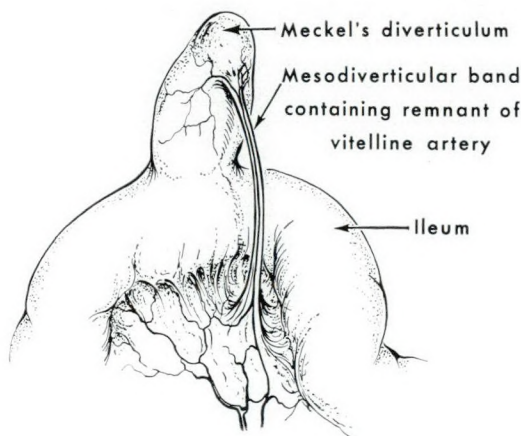

Fig. 1.—The mesodiverticular band.

band an internal hernia may develop and become incarcerated or strangulated. Volvulus may also occur around this fibrous cord.

One of our patients had a classical Littre's hernia in which the diverticulum alone had become incarcerated in an inguinal hernia.

If the Meckel's diverticulum was associated with obstruction, the mortality rate was higher and postoperative complications were more common than in children without a diverticulum. These patients required resection of the ileum more often and had a higher incidence of evisceration. In our series a diagnosis of intussusception, and not intestinal obstruction secondary to a Meckel's diverticulum, was frequently made preoperatively. No significant age distribution of these complications was noted.

Inflammation and perforation were the next most common complications, closely followed by patent vitelline duct. One-half of the diverticula that contained heterotopic gastric mucosa were inflamed

and perforated. The perforation may be secondary to peptic ulceration alone or secondary to obstruction of the ostium with resulting increase in intraluminal pressure, gangrene of the wall of the diverticulum and perforation. The latter lesion resembles acute perforated appendicitis and, in fact, that diagnosis was made in 63% of our cases.

DISCUSSION

The slightly higher ratio of uncomplicated to complicated cases in this present series is probably due to the fact that, when found incidentally at an operation undertaken for other reasons, more of these diverticula are resected now than previously. Intravenous therapy, intestinal intubation, improved anesthesia, prompt operative intervention and the administration of antibiotics for infective complications have reduced mortality. Once the diagnosis of bleeding Meckel's diverticulum has been made and the patient has been transfused and stabilized, operation should be carried out, because recurrent bleeding may be exsanguinating. Furthermore, patients in the age group in which such hemorrhage most often occurs do not tolerate hypovolemia well.

The lowered postoperative complication rate in our series (6%) as compared to 11% in that of Wansbrough, Thomson and Leckey from this hospital is no doubt a reflection of earlier diagnosis and better methods of supportive treatment. But it should be remembered that the commonest complication in their series[5] was intestinal obstruction and patients with this combination showed a higher incidence of postoperative complications. Some unusual complications of Meckel's diverticulum are recorded in the literature[2, 3, 6, 7] such as, perforation of the diverticulum with a fish bone and assorted other foreign bodies; Crohn's disease and carcinoid tumour, and enteroliths and gallstones in the diverticulum, have also been reported. In 52 years at the Hospital for Sick Children, Toronto, no such unusual complications were seen in 457 patients.

In both series at the Hospital for Sick Children (ours and that of Wansbrough, Thomson and Leckey[5]), 50% of diverticula

that produced symptoms were found in children under 2 years of age.

In the past year we encountered two infants with unusual Meckel's diverticulum that were not included in the present review.

CASE REPORTS

Case 1.—An 11-year-old boy presented with a 24-hour history of abdominal pain and vomiting. He had tenderness and rigidity in the right lower quadrant, a temperature of 100.8° F. and an elevated white blood cell count. The preoperative diagnosis was "perforated appendicitis". At operation he had an acutely inflamed Meckel's diverticulum, the ostium of which was blocked by a peanut. This was the first instance at our hospital of a foreign body in a Meckel's diverticulum.

Case 2.—This 8-month-old infant presented with abdominal distension, diarrhea, recurrent respiratory tract infections, malabsorption and failure to thrive. A barium enema showed a large cystic mass which filled with barium but did not empty. The preoperative diagnosis was "intestinal duplication" with incomplete small-bowel obstruction, malabsorption and diarrhea.

At operation, a large dilatation of the ileum 10 cm. in diameter was found 120 cm. proximal to the ileocecal valve. A "bound-down" mass of tissue (presumably a mesodiverticular band) ran from the mesentery to the apex of this dilatation (Fig. 2). On histological examination, this lesion, which contained heterotopic pancreatic and gastric mucosa, was a Meckel's diverticulum. The incomplete obstruction of the ileum was due to the bound-down mesodiverticular band. We postulated that the

obstruction caused by this band led to dilatation of both Meckel's diverticulum and proximal ileum producing a blind loop syndrome,[6] characterized by malabsorption, diarrhea, abdominal distension and failure to thrive. Resection of the diverticulum and dilated ileum was uneventful and the child has now regained normal height and weight.

SUMMARY

Two hundred and eighteen patients with Meckel's diverticulum were seen during a 10-year period (1955 to 1964) at the Hospital for Sick Children, Toronto. The histological findings are described.

The complications associated with the diverticulum, the method of operative treatment, postoperative complications and mortality are discussed.

There was a striking decrease in mortality over the last four decades which is a reflection of earlier diagnosis, energetic search for the lesion and improved methods of treatment. The higher ratio of uncomplicated to complicated diverticula is a reflection of the fact that the terminal ileum is now examined more frequently at operation.

Bleeding was the most common complication of Meckel's diverticulum in this series whereas in the previous review from the same hospital intestinal obstruction was the most common. The intestinal obstruction associated with Meckel's diverticulum produces more postoperative morbidity than bleeding.

Two infants seen recently with unusual manifestations of Meckel's diverticulum are described in detail.

Fig. 2.—The mesodiverticular band obstructing the ileum and Meckel's diverticulum.

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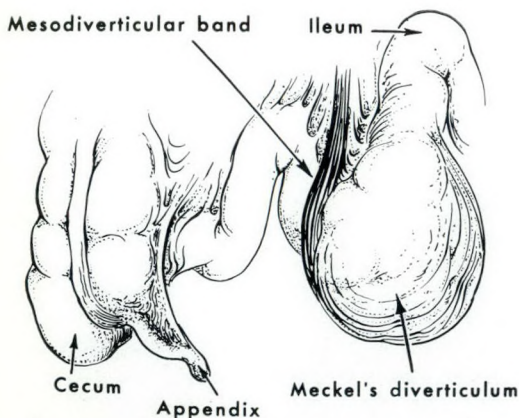4. RUTHERFORD, R. B. AND AKERS, D. R.: Meckel's

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Résumé

Au Hospital for Sick Children de Toronto, les auteurs ont eu l'occasion d'observer, au cours d'une période de 10 ans (1955-1964), 218 nourrissons présentant des diverticules de Meckel. Après avoir décrit les constatations histologiques, ils exposent le mode opératoire, les complications post-opératoires et étudient la mortalité.

Celle-ci a diminué de façon spectaculaire au cours des quatre dernières décennies, ce qui est une conséquence d'un diagnostic plus précoce, d'une recherche obstinée de la lésion et de l'amélioration des méthodes de traitement. La proportion plus élevée de diverticules sans complications par rapport aux cas compliqués provient du fait qu'on procède plus fréquemment qu'autrefois à l'examen peropératoire de l'iléum terminal.

Dans le présent groupe, l'hémorragie a été la complication la plus fréquente du diverticule de Meckel, alors qu'au cours de la revue antérieure émanant du même hôpital, c'était l'occlusion intestinale qui se rencontrait le plus souvent. Cette dernière complication est du reste frappée d'une morbidité post-opératoire plus forte que l'hémorragie.

Les auteurs présentent en détail les cas récents de deux nourrissons dont le diverticule de Meckel comportait des manifestations exceptionnelles.

ESOPHAGEAL HIATAL HERNIA

Herniation of the esophagogastric junction through an enlarged esophageal hiatus is the commonest hernia of the diaphragm. About 50% of all patients with hiatal hernia have symptoms of varying severity, while approximately 5% have sufficiently severe symptoms to require surgical repair. Coexistent symptoms in 30 to 47% of the patients are those of concurrent intra-abdominal disease, usually chronic cholecystitis or peptic ulcer.

Transabdominal repair was done in 26 patients with symptomatic esophageal hiatal hernia during a four-year period. Each patient had coexisting abdominal disease which was the major reason for selecting an abdominal approach. Seventeen patients had chronic cholecystitis, and the hiatal hernia was repaired in association with cholecystectomy and operative cholangiography. Fifteen patients had either a chronic duodenal or gastric ulcer, and herniorrhaphy was performed along with vagotomy and pyloroplasty, or vagotomy and gastrectomy of the distal portion of the stomach. Six patients had a combination of hiatal hernia, chronic cholecystitis, and peptic ulcer; herniorrhaphy, cholecystectomy, and vagotomy with pyloroplasty were all performed at the same procedure.

There were no deaths among these 26 patients. All have been contacted and their progress followed up by questionnaires to determine whether they have had complete or incomplete relief from preoperative symptoms. Of the 26 patients, 20 have had continued, complete relief from all epigastric or substernal distress. Symptoms in five of the patients have improved, but relief from all epigastric or abdominal distress has been incomplete. A recurrence of the hiatal hernia was noted in one patient, with a return of symptoms two years after hernial repair.

These early results are similar to those reported by other surgeons using different types of surgical repair of esophageal hiatal hernia. In other series, approximately 85% of the patients have been reported to be symptomatically benefited by hiatal hernia repair, whether repair was made by a transthoracic or a transabdominal approach. Hernias have recurred after all types of repair in 10 to 25% of patients who have undergone operation.

Esophageal hiatal hernia frequently coexists with other intra-abdominal disease, especially chronic cholecystitis and peptic ulcers. The chief merit of the abdominal approach to diaphragmatic hernial repair is that the coexisting pathologic lesions can also be treated. The main disadvantage of transabdominal repair is the difficulty of closure of the hernial defect in obese patients. A repair anterior to the esophagus is simple, appears to be equally as strong as that which is made posterior to the esophagus, and returns the esophagus to its normal posterior position against the spine, thus restoring the normally acute esophagogastric angle.—Hermann, R. E.: Transabdominal repair of esophageal hiatal hernia anterior to the esophagus, *Surg. Gynec. Obstet.*, **126:** 844, 1968.

COMPLICATIONS IN THE URETERAL STUMP AFTER NEPHRECTOMY°

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PATHOLOGICAL changes in the ureteral stump after nephrectomy are uncommon. Nevertheless, after the affected kidney has been removed, disease may develop or persist in the residual ureter. Such disease may present at varying intervals after operation and require resection of the ureteral stump. Three such patients came under my care within a nine-month period. It is the purpose of this paper to call attention to this complication and to the need for careful evaluation of the ureter before nephrectomy is done. If nephrectomy is performed for renal tuberculosis or tumour arising from the urothelium above the ureterovesical junction, the ureter should be removed completely. In certain other lesions—residual ureteral stone, dilated infected ureter, stricture or reflux with infection in the ureter—the ureter should be completely removed along with the kidney. This principle is illustrated in two patients (Cases 1 and 2) in whom a diseased ureter was left behind after nephrectomy, and empyema of the ureteral remnant developed. Ureterectomy was subsequently required.

CASE REPORTS

Case 1.—R.M., an 8-year-old white boy who was admitted to the Jewish General Hospital, Montreal, on September 15, 1953, had a six-month history of intermittent gross hematuria with recurrent severe epigastric pain. On investigation, there was a large staghorn calculus in the right kidney, no contrast medium was excreted from this kidney and there was a large (3 x 1 cm.) elliptical calculus in the right lower ureter. The urine was loaded with pus cells. The large stone in the lower right ureter was bypassed without difficulty and the urine obtained by catheterization grew a heavy growth of *Proteus vulgaris*. The kidney was removed with the upper one-half of the ureter, which was markedly thickened. The pathologist's report described a "staghorn calculus in
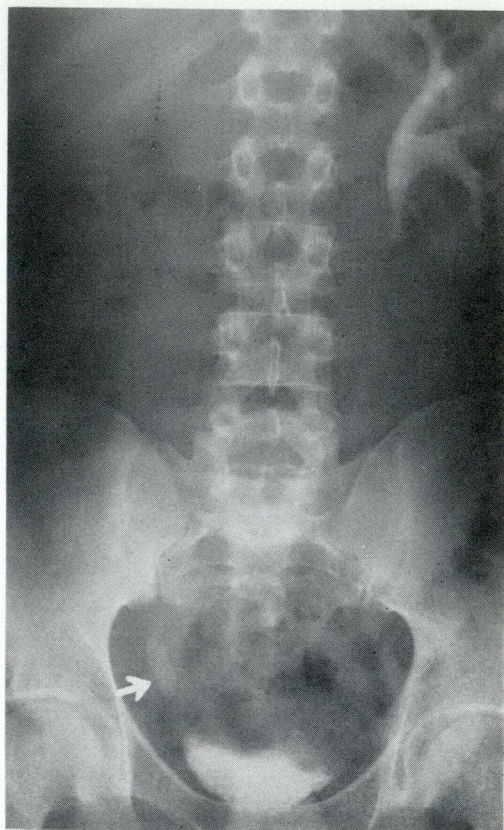
Fig. 1.—Intravenous urogram showing elongated calculus (arrow) in the stump of the right lower ureter, unchanged from 13 years before. There is compensatory hypertrophy of the left kidney.

the right kidney with chronic ulcerative pyelonephritis and ureteritis".

This patient was seen in December 1966 (13 years later) complaining of burning and frequency of urination. His urine was loaded with pus cells, many in clumps. Over the next few months his urinary tract infection was treated with a number of drugs without success. On several cultures, heavy growths of *P. vulgaris* and *Escherichia coli* were isolated.

He was admitted to hospital on May 9, 1967. An intravenous urogram showed the previously noted elongated calculus in the right lower ureter (Fig. 1). At cytoscopy, he had extensive hemorrhagic cystitis and thick mucopurulent material was seen oozing from the right ureteral orifice, which was otherwise normal. The cystogram showed no evidence of ureterovesical reflux. A No. 5 French
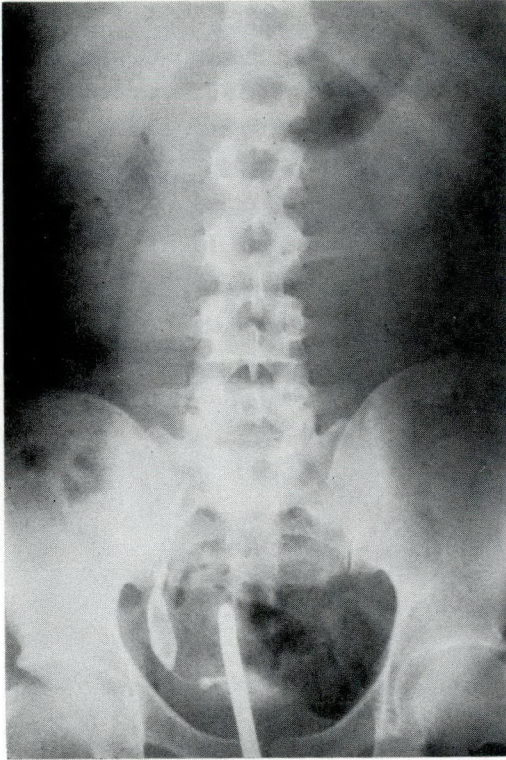
Fig. 2.—Right ureterogram showing a stone in the dilated ureteral stump.

Fig. 3.—Dilated stump of ureter containing an elongated stone.

catheter was passed 2 cm. up the right ureter where it stopped. A ureterogram showed a large stone in the ureteral stump—empyema around this stone was responsible for the persistent infection (Fig. 2). Urine cultures showed *Proteus mirabilis* more than 100,000/ml. The ureter, which was resected through a right Gibson incision on May 12, 1967, contained a calculus measuring 3 x 1.1 x 0.4 cm. (Fig. 3). The ureter was dilated and thickened, its wall was inflamed and ulcerated and the lumen was choked with inspissated pus. This patient recovered uneventfully and his urinalyses and urine cultures have since remained normal.

Case 2.—M.F., a 45-year-old white woman, was admitted to hospital on January 4, 1966, with a history of recurrent chills and fever of several years' duration. Recently she had had a severe episode accompanied by gross painless hematuria and persistent pyuria. An intravenous pyelogram showed a large irregular stone in the left kidney which excreted no contrast medium after 60 minutes. The right kidney appeared normal. Cystoscopy revealed extensive cystitis. A No. 6 French catheter was passed through a slightly gaping, left

ureteral orifice to the renal pelvis; no obstruction was encountered. Following this, there was a marked outpouring of turbid purulent fluid from the renal pelvis—approximately 300 c.c. of it was aspirated. A retrograde pyelogram showed marked hydronephrosis with extensive caliectasis and destruction of the renal parenchyma. There was a large stone in the renal pelvis (Fig. 4). Urine from the bladder and left kidney was negative on routine culture, acid-fast smears and guinea-pig culture. On January 10, 1966, a large flabby kidney was removed which contained a large staghorn calculus. The upper one-third of ureter was markedly dilated and thickened. The pathological diagnoses were: (1) renal calculus (staghorn), (2) chronic ulcerative pyelonephritis with marked hydronephrosis and hydroureter with marked renal atrophy, and (3) chronic ureteritis and periureteritis.

After an uneventful recovery, this woman was well until April 28, 1966, when she suddenly developed severe left lower abdominal pain associated with fever, nausea, vomiting and diarrhea. This attack was followed by marked dysuria and gross hematuria. She was acutely ill, had a temperature of 101.2° F., and a firm tender mass in the left lower quadrant. Her urine, which was cloudy red,
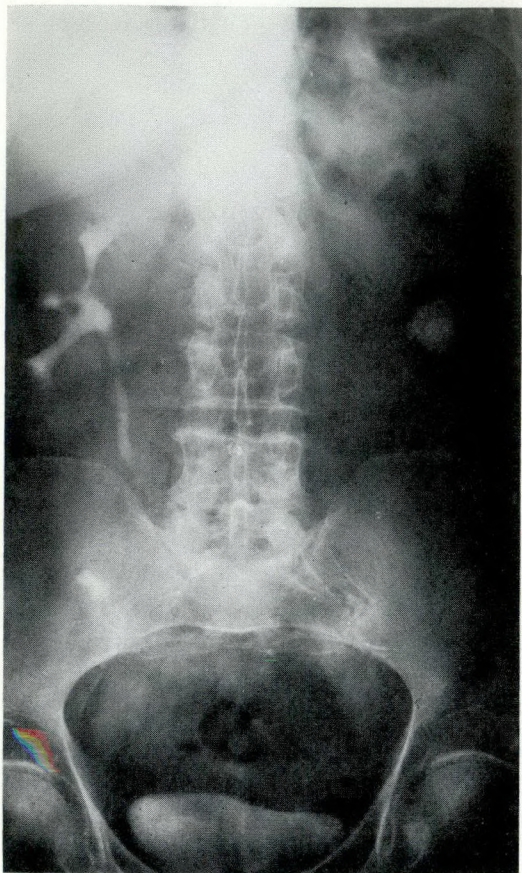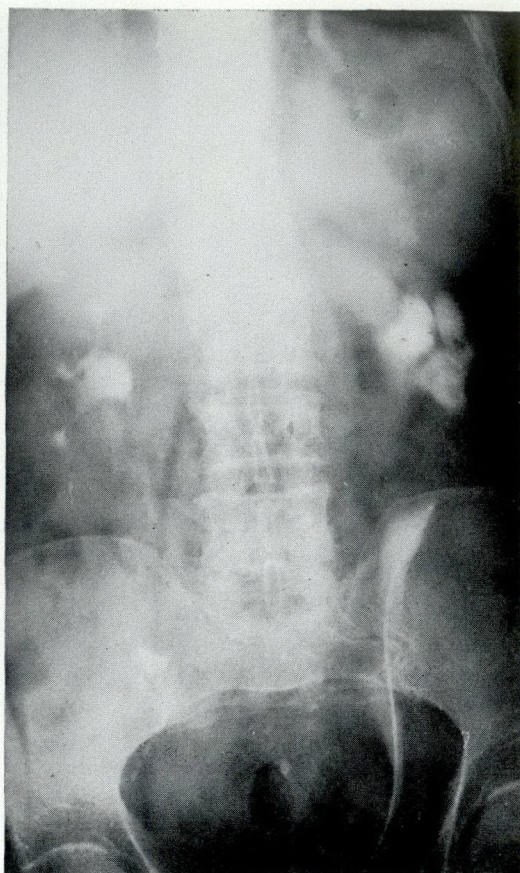
Fig. 4a   Fig. 4b

Fig. 4.—(a) Intravenous urogram and (b) retrograde pyelogram showing a large left renal calculus, with extensive pyelectasis, caliectasis, markedly dilated ureter and loss of renal parenchyma.

was loaded with red and white blood cells. On culture, it contai̇ d *E. coli,* more than 100,000/ml. Ampicillin was started and intravenous fluids were administered. An intravenous urogram showed a normal solitary right

Fig. 5.—Ureteral stump showing marked dilatation and thickening of the ureteral wall with coagulated blood clot and a periureteral adhesive mass.

kidney. At cystoscopy, the left ureteral orifice was edematous, reddened and somewhat gaping. When the left lower abdomen was compressed, thick reddish-yellow inspissated purulent material was expressed from the ureteral stump. The stump was obstructed by solid material within its lumen that was continuously extruded from the orifice on lower abdominal compression. The findings suggested empyema of the ureteral stump.

On May 4, 1966, left ureterectomy was performed through an extended Gibson incision. With considerable difficulty, the ureter was isolated at the brim of the pelvis, where it was greatly distended resembling small bowel. Dissection was further complicated by marked periureteritis. The intrapelvic ureter was adherent to the iliac vessels. After much tedious dissection, the entire ureter was freed and transected at the ureterovesical junction. The ureteral stump was filled with inspissated pus and clotted blood (Fig. 5). Microscopically,

Fig. 6.—Intravenous pyelogram showing duplication of the right collecting system with marked caliectasis of the lower pole.

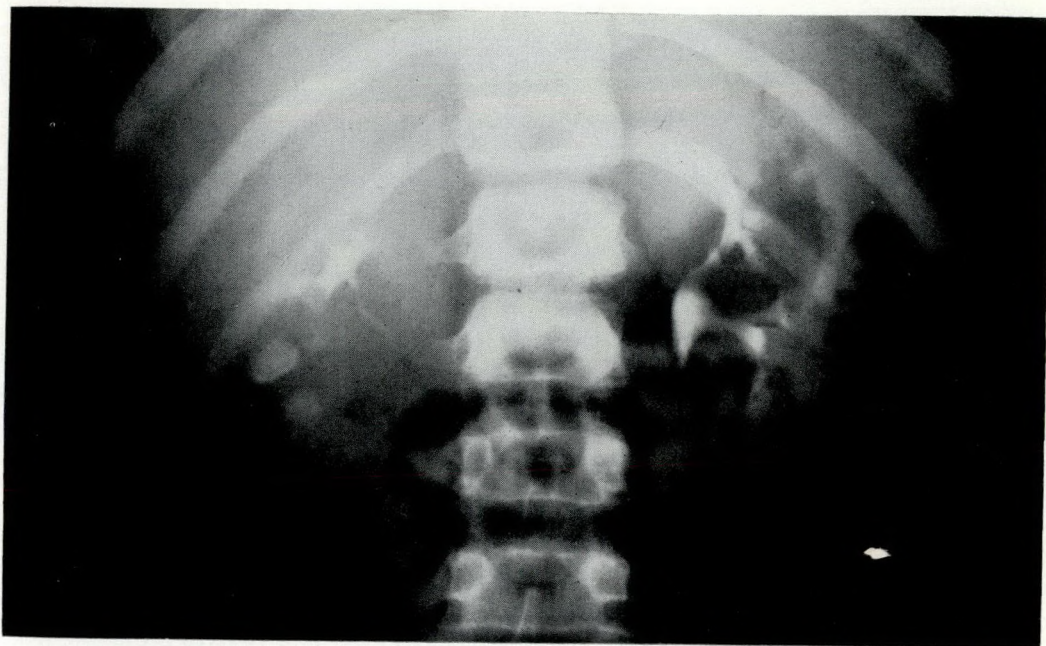there was subacute and chronic ulcerative inflammation of the ureteral stump, which was the seat of fibrosis and periureteral fibrosis.

The woman's postoperative course was uneventful. Her urinalysis and urine cultures have been negative since discharge from hospital.

Case 3.—J.S., a 9-year-old white boy, was admitted to the Jewish General Hospital on July 8, 1962 for investigation of asymptomatic pyuria. The intravenous pyelogram (Fig. 6) suggested a duplication of the right renal collecting system, a normal upper pole, and marked clubbing and hydronephrosis involving the middle and lower calyces, and poor filling of the renal pelvis. The left pyelogram was normal. At cystoscopy, the bladder was full of thick mucopurulent material which was washed from the cavity. Extensive cystitis cystica surrounded the trigone and vesical neck, and marked bullous edema made visualization of the ureteral orifices impossible. Culture of the urine gave a heavy growth of coagulase-positive *Staphylococcus aureus* sensitive to all antibiotics. The diagnosis was "infected hydronephrosis involving the lower half of the right kidney". Long-term penicillin therapy was begun at once. His urine cultures became sterile but he continued to have intermittent pyuria. A repeat intravenous pyelogram and cystoscopy six months later showed little change except that the contrast

medium in the lower clubbed calyces was more concentrated. One ureter filled completely on the right, suggesting complete duplication of the collecting system. At cystoscopy, the upper right ureteral orifice was seen to be surrounded by bullous edema but it could not be catheterized. The lower right orifice was of normal size and shape but entered well down on the posterior lip of the vesical neck. When antibiotic therapy was stopped, the urine grew coagulase-positive *Staph. aureus* repeatedly. For this reason nephrectomy was done on July 3, 1963. The lower one-half of the kidney was markedly atrophic and flabby; it was drained by a dilated thickened ureter. A small normal-appearing ureter arose from the upper pole. Both ureters were transected in their middle thirds. This boy had subacute and chronic pyelonephritis with infected hydronephrosis in the lower half of the kidney, and complete congenital reduplication of the collecting system (Fig. 7). His postoperative course was uneventful.

Over the next 18 months he was free of symptoms, but then developed pyuria, which could only be controlled intermittently with antibiotics. It was apparent that the ureteral stump was infected; this was confirmed on March 3, 1965, when a No. 5 French catheter was passed through the upper right ureteral orifice, which was obscured by extensive bullous edema, and thick pus was withdrawn.
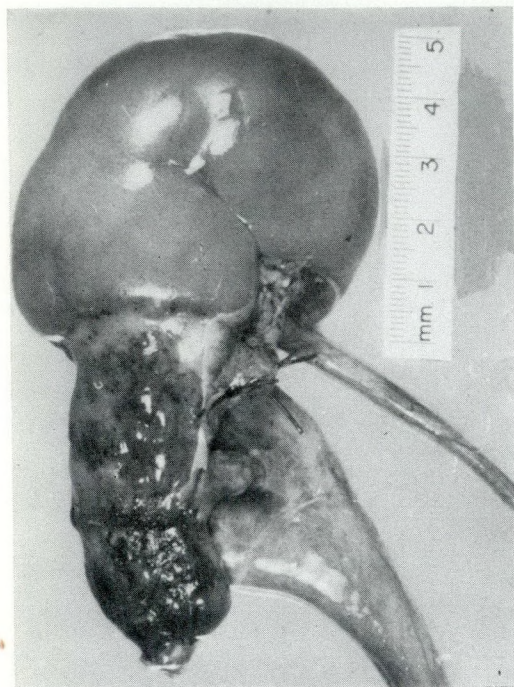
Fig. 7.—Marked atrophy of renal parenchyma in the lower pole with hydronephrosis, caliectasis and dilated ureter.
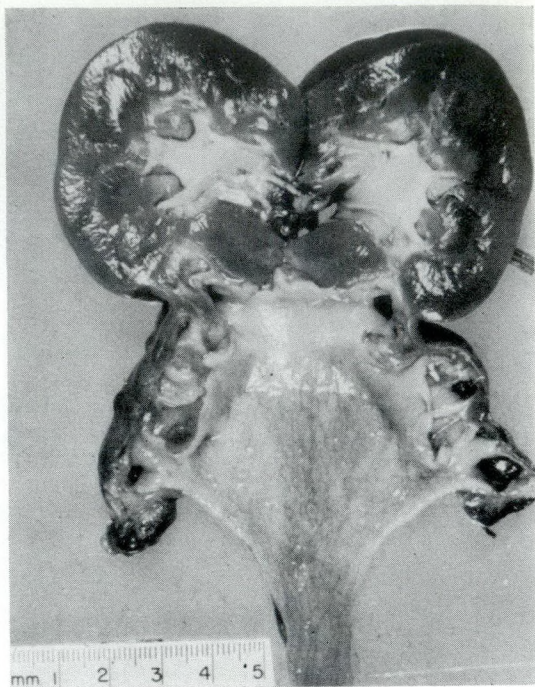
Ureterography then demonstrated a dilated tortuous ureteral stump (Fig. 8). Resection of

Fig. 8.—Ureterogram showing dilatation and irregularity of the right ureteral stump.

the infected ureter was recommended but was delayed because of parental reluctance. Antibiotic therapy was continued until August 1966. When he was admitted again, cystoscopy showed a wide, gaping right upper ureteral orifice on the right side with reflux. The extensive cystitis cystica and bullous edema had subsided. The duplicated right ureters were resected on August 10, 1966. Both ureteral stumps—one of normal calibre, the other markedly dilated and thickened—were removed down to the vesical junction. The infected stump showed chronic ureteritis and hydroureter (Fig. 9).

Since discharge the patient has had negative urinalysis and culture.

DISCUSSION

The fate of the ureteral stump after nephrectomy has been thoroughly described in the urological literature.[1-19] The possible complications (illustrated in this paper) although infrequent must be borne in mind in patients who develop persistent infection, pyrexia or abdominal pain after nephrectomy. The latent period may be long as in Case 1 (13 years) and those reported by Bennetts et al. (27 years),[14] Moore (26 years),[15] and Senger et al. (23 years).[8]

Fig. 9.—Ureterectomy specimens showing a markedly dilated and thickened ureter draining the lower pole, compared to a normal ureter leading to the upper pole.

The incidence of disease in the ureteral stump following nephrectomy is difficult to determine. In 1950, Rieser[10] collected 37 cases of pyoureter that had been reported by 18 authors over a 25-year period; in these, residual calculi and ureteral strictures

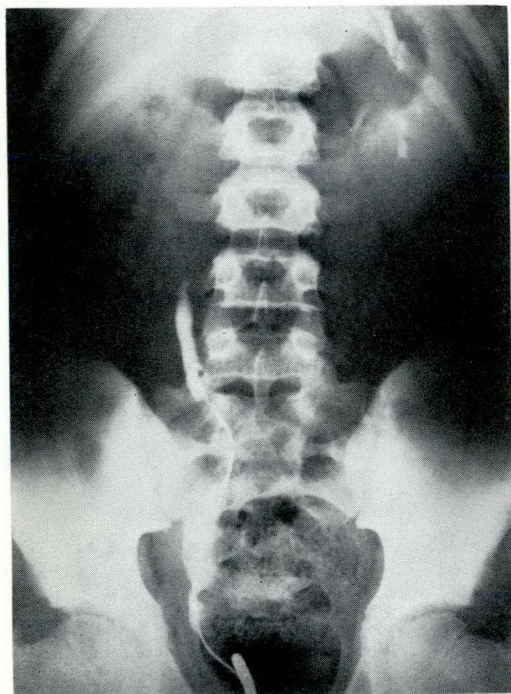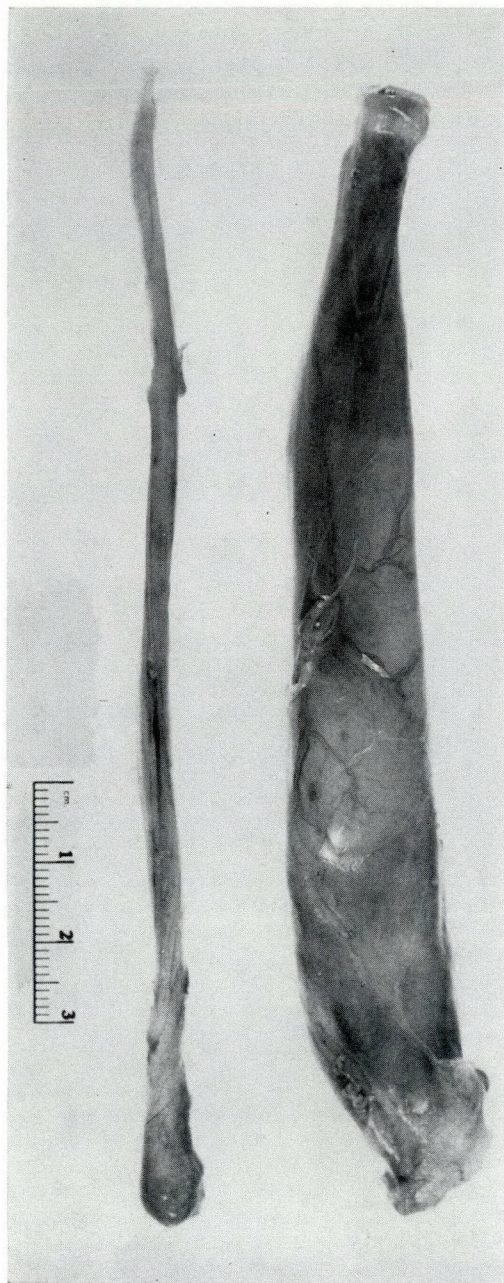were the most common complications. Since that time Amar,[17] and Bruce and Awad[18] have emphasized vesicoureteral reflux into the stump as a cause of abdominal pain and infection. Such cases and Case 2 in this paper demonstrate that this lesion may cause pain or be the site of infection after nephrectomy in patients from whom the complete ureter has not been removed.

Classical studies of the anatomical and physiological fate of the ureter after nephrectomy were reported by Latchem[1] in 1922. Ureteral electromyography by Baker and Huffer[13] in 1953 suggested that the ureteral stump has the same physiologic activity as the intact ureter. It is capable of peristalsis, hyperperistalsis and ureteral colic. When an infected ureter is obstructed, hypertrophy of the muscularis, persistent inflammatory changes and stricture may set the stage for local empyema. When drainage is poor, periureteritis and periureteral abscess may develop: the stump becomes a bag of pus and a fistula may form.[15] Congenital anomalies of ureteral insertion[6] or ureterocele[8] may contribute to persistent infection in a ureteral stump.

One of the symptoms of ureteral stump disease is pain in the lower abdomen, which may be mild to severe (as in Case 2), with associated tenderness due to periureteral involvement. There may be symptoms of cystitis—frequency, urgency, dysuria and nocturia—because of secondary bladder infection, or the patient may be relatively asymptomatic and have only pyuria.

Diagnosis is readily made and is based on the history of previous nephrectomy, cystoscopy with ureterography demonstrating the diseased stump, or voiding cysto-urethrography demonstrating reflux into the infected ureteral remnant.

Many advocate complete nephroureterectomy when there is obvious disease in the lower ureter, such as stone or stricture, or where reflux is demonstrated before operation. On the basis of these three cases, it is suggested that patients be selected for nephroureterectomy, bearing in mind that there are other positive indications for complete removal of the ureter besides tuberculosis and tumour. If the tumour arises in the urothelium of the renal pelvis or ureter, total ureterectomy is mandatory; this should

include a cuff of bladder at the distal end of the ureter, because this is a frequent site of tumour recurrence.

SUMMARY

Three patients with infected ureteral stumps are described in whom ureterectomy was necessary at varying intervals following nephrectomy. One had a stone in the ureter that produced persistent urinary infection over a 13-year period after nephrectomy; the second had reflux in a duplicated ureter with persistent infection; and the third had a pyoureter with hemorrhage and marked periureteral involvement.

Disease in the ureter should be recognized and evaluated before nephrectomy. When they have lesions other than tumour or tuberculosis, candidates for nephroureterectomy should be carefully selected to avoid subsequent development of empyema in the ureteral stump.

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RÉSUMÉ

Une pathologie qui survient dans le moignon d'uretère subsistant après néphrectomie peut, par la suite, rendre nécessaire une urétérectomie. L'auteur expose les cas de malades chez lesquels un calcul de l'uretère, un rétrécissement ou un reflux ont abouti à de l'empyème du moignon urétéral dans un délai allant jusqu'à 13 ans après la néphrectomie. Les symptômes présents étaient une pyurie rebelle, de la fièvre, une douleur abdominale bénigne ou sévère, des mictions fréquentes et de la dysurie. Le diagnostic a été basé sur une histoire clinique de néphrectomie et confirmé au moment de la cystoscopie avec urétérographie ou urétro-cystographie mictionnelle. L'auteur insiste sur l'importance de déceler la pathologie de l'uretère avant la néphrectomie et de procéder à la résection du rein et de l'uretère, du moins dans les cas différents d'une tumeur ou de la tuberculose, ceci afin de prévenir l'apparition d'empyème dans l'uretère résiduel. Etant donné que le moignon d'uretère conserve la même activité physiologique que l'uretère intact, un pyouretère peut se développer quand l'obstruction et l'infection persistent dans le restant d'uretère.

CASE REPORTS

END-TO-END ANASTOMOSIS IN ESOPHAGEAL ATRESIA WITHOUT TRACHEAL FISTULA: A CASE REPORT*

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ESOPHAGEAL atresia is divided by Gross into six types (Fig. 1): Type A, atresia without fistula, accounts for 5 to 7% of these abnormalities. In infants with atresia without fistula, the upper and lower esophageal segments are separated on the average by 5 to 6 cm. Until recently, end-to-end anastomosis was not considered feasible except in those infants with only minimal separation of the two segments. Mobilization of the stomach to allow primary anastomosis has not given good results because serious esophagitis nearly always results. In the past, the usual management has been to perform a gastrostomy to allow feeding and a cervical esophagotomy to allow drainage of the saliva; at some later date (1 or 2 years of age) the gap is bridged with a small-bowel or colon transplant. As a result of the cervical esophagotomy, a considerable length of the upper segment of the esophagus is ultimately lost and, for this reason, the transplant must be inserted at a high level. Bowel has not proved to be a satisfactory substitute for the esophagus in many of these infants because they develop regurgitation, dysphagia and obstruction due to stenosis.

It has been proposed that, instead of doing a cervical esophagotomy, the upper pouch be left in place and allowed to stretch under the stimulus of continued attempts to swallow saliva. Howard and Myers[1] and Johnston[2] recently described infants with simple atresia in which this appears to have occurred; they believe that the stretching may be assisted by gentle but firm bouginage of the upper pouch for 5 or 10 minutes twice daily. The infant must have constant nursing care because saliva has to be gently suctioned from the upper pouch every 15 or 20 minutes. Koop
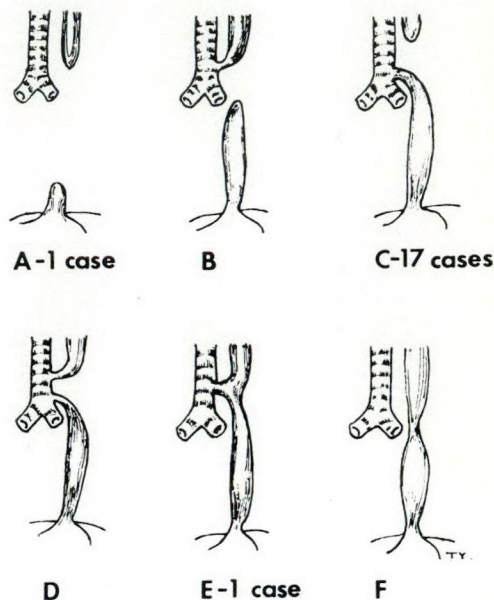
Fig. 1.—Classification of esophageal atresia (after Gross): Type A—simple atresia with blind upper and lower segments; Type B—upper segment with fistula to tracheobronchial tree, and lower blind segment; Type C—blind upper pouch, lower segment with fistula to tracheobronchial tree; Type D—upper and lower segments with fistula from each to tracheobronchial tree; Type E—"H" type; fistula only, no atresia; and Type F—stenosis.

and Hamilton[3] have also suggested that the lower segment may stretch upwards under the stimulus of gastroesophageal reflux during gastrostomy feedings. In simple atresia, Howard and Myers, and Johnston recommend that elongation of the upper and lower segments be encouraged as described above and, after a period of four to six weeks, if combined radio-opaque examination of the upper and lower segments shows that the gap is appreciably narrowed, an end-to-end anastomosis be attempted.

Reports of end-to-end anastomosis following stretching are still uncommon and, in most medical centres, colon or small-bowel interposition is probably still the accepted procedure. Howard's successful
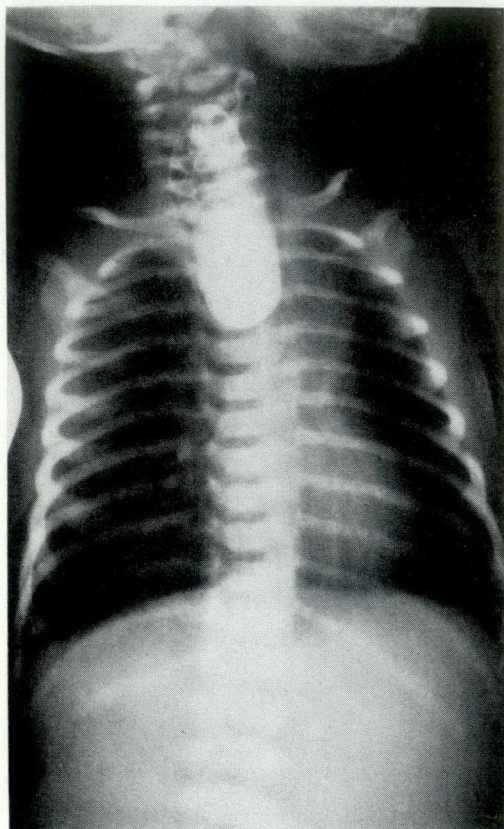
Fig. 2.—Dionosil swallow shows blind upper pouch and a gasless abdomen—simple atresia (Type A abnormality).

repair was in an infant who had the common Type C abnormality, but an unusually high pouch. Johnston used this method in an infant with fistula to the upper pouch and a widely separated lower pouch. Young[4] used this method in an infant with simple atresia. Hays, Woolley and Snyder[5] described three infants with simple atresia in whom end-to-end anastomosis was performed following a 4 to 10-week delay to encourage elongation.

This communication describes a successful end-to-end anastomosis, after a period of stretching, in an infant with simple atresia and wide separation of the segments.

CASE REPORT

H.A., a female infant, was born on May 30, 1967 at Prince Albert, Saskatchewan, weighing 8 lb. 13 oz. (4010 g.). Because of the mother's history of marked hydramnios, and excessive

frothy oral discharge from the baby since birth, Dr. G. Powles, the family physician, and Dr. W. H. Fry, consulting gynecologist, of Prince Albert, made the diagnosis of esophageal atresia. A pyridone-N-acetic acid (Dionosil) swallow (Fig. 2) showed a blind upper pouch with a gasless abdomen, which suggested simple esophageal atresia (Type A abnormality).

The infant was admitted to the University Hospital, Saskatoon, on June 1, 1967. The following day a feeding gastrostomy was created under local anesthesia. She was placed on constant nursing care with repeated aspiration of the upper blind pouch. Ten days later a barium gastrogram and mercury bouginage of the upper pouch (Fig. 3) demonstrated that
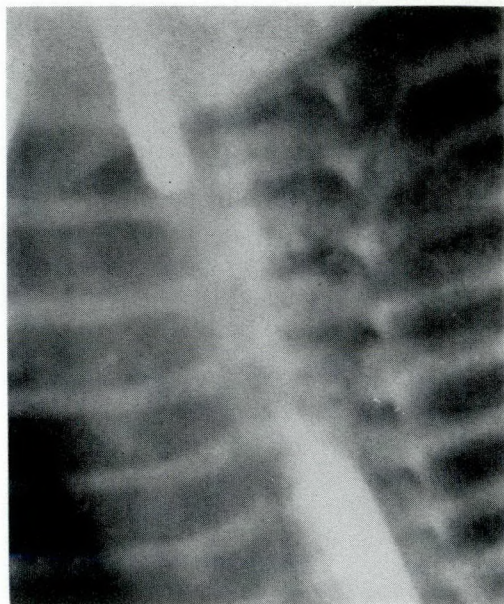
Fig. 3.—Initial barium gastrogram combined with mercury bouginage of the upper pouch shows a separation of 7 cm.

a lower segment of esophagus was separated from the upper segment by an estimated 7 cm.

She was continued for another six weeks on gastrostomy feedings, repeated aspiration of the upper pouch, and twice-daily stretching of the upper pouch with a No. 20 and later a No. 26 mercury bougie. Using 100 c.c. of barium in the stomach, and pushing the mercury bougie as far down in the upper pouch as it would readily go, repeat radiological studies were done (Fig. 4); these showed that the gap could be reduced to as little as 1.5 cm. It was decided, therefore, to continue the stretching for another two weeks and then operate.
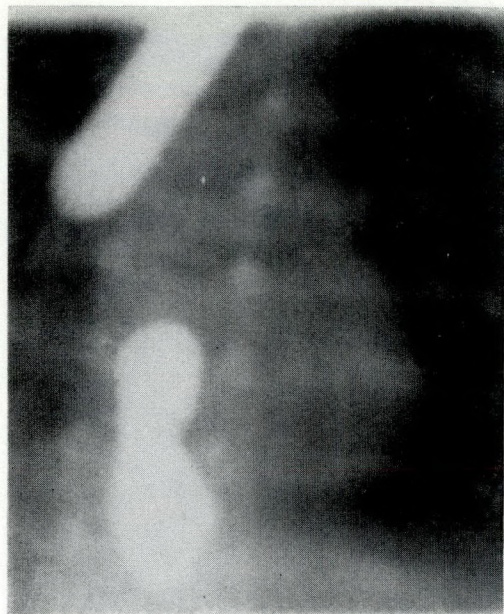
Fig. 4.—Repeat barium gastrogram and mercury bouginage of the upper pouch after six weeks of bouginage stretching of the upper pouch.

Fig. 5.—Dionosil swallow five days after operation.

On August 23, 1967, under endotracheal anesthesia, the right chest was opened transpleurally. There was a discouraging gap of 5 or 6 cm. between the two segments. The lower segment was approximately at the level of T8 and the upper segment about T3. However, the two segments stretched quite readily and, after mobilization, could be overlapped under tension; the upper segment was quite large and stretched readily to reach below the level of the azygos vein. Both segments were further mobilized to lessen the tension. The lower segment was mobilized down to the hiatus, but the stomach was not mobilized. Care was taken to identify and preserve both vagus nerves.

The two segments were then anastomosed with a one-layer technique using six to eight 4-0 Deknetal silk sutures, carefully placed to include both muscle and mucosa on each side of the anastomosis. Two posterior sutures were placed first, the knots being tied on the inside of the lumen. A tube was then passed to splint the anastomosis and to facilitate the insertion of four to six lateral and anterior sutures, which were tied outside the lumen.

Dionosil swallow (Fig. 5) on the fifth postoperative day showed that the anastomosis was intact. Small oral feedings, started that day, were gradually increased. Because she had never sucked in the early weeks of her life, about four days passed before she learned to

suck adequately. Once started, however, she was a greedy feeder, and our problem was to make sure that she was not fed too quickly until healing was more secure. She went on to full recovery and was discharged on September 18, 1967. Repeat Dionosil swallow before discharge showed no evidence of stricture. She was able to take full formula feedings quite readily and was also on cereals. Her weight on discharge was 15 lb. She was seen again on December 16, 1967 and has remained well.

DISCUSSION

This was the only infant with simple atresia in a personal series of 19 infants with esophageal atresia and/or fistula, seen over the past 11 years. Seventeen of these infants had Type C esophageal atresia—blind upper pouch and fistula from lower segment to trachea; this is in keeping with general experience that about 85 to 90% of reported cases are Type C. It is in this more common Type C atresia that the staged operation may find its widest application. To improve survival rates, Koop and Hamilton[3] suggested that planned, delayed, end-to-end anastomosis be done in prema-

ture and poor-risk infants with the common Type C abnormality. In the first stage, gastrostomy is performed, and, if the infant's condition allows, the fistula to the lower segment is tied off and anchored without tension to the posterior chest wall. Anastomosis is done as the second stage after a period of 4 to 10 weeks. If the upper pouch is unusually short, it is elongated by the technique described above. I have not used this procedure in premature infants, but it would seem to be an excellent method particularly in small prematures.

The single most important aspect of postoperative care is good tracheal toilet. In prematures, due to the small size of the trachea, this is very difficult, because they do not tolerate repeated endoscopic suction. If single or repeated aspirations are unsuccessful in relieving tracheal or major bronchial secretory obstruction, I do immediate tracheotomy under local anesthesia using a shortened 3-0 metal tube, or sometimes the shortened inner cannula of a 2-0 or 3-0 tube. The tube must be small enough to allow the infant to continue to breathe around it, so that normal respiratory function and glottis reflex will be maintained, yet large enough to accept a small plastic suction catheter. The cannula or tube can be removed in two or three days without difficulty; also, one is able to aspirate through the remaining tracheal stoma for another two or three days. Tracheotomy has been life-saving in three of our babies.

The staged procedure may not be indicated in lusty and larger prematures, if one is prepared to perform early tracheotomy when indicated. However, the staged procedure should be most useful in all small prematures and other poor-risk patients with the common Type C esophageal atresia. It would appear to be the best method of treating infants with widely separated segments in both Type A (simple atresia) and B (upper pouch fistula) abnormalities. If it is unsuccessful, a bowel transplant can still be done.

SUMMARY

The treatment of a female infant with simple atresia of the esophagus and wide separation of upper and lower esophageal segments is described. After initial gastrostomy and a six-week period of stretching of the upper segment, the upper and lower segments were successfully anastomosed. Staged operation with delayed anastomosis is recommended in premature and poor-risk infants with the common Type C abnormality.

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RÉSUMÉ

L'auteur présente le traitement adopté chez un nourrisson du sexe féminin souffrant d'une atrésie simple de l'ésophage et d'une large séparation des segments supérieur et inférieur de l'ésophage. Après une gastrostomie initiale et une période de six semaines d'étirement du segment supérieur, il a pu procéder à une anastomose bout-à-bout des deux segments. Il estime que l'opération en deux phases avec anastomose retardée est à conseiller chez le prématuré et chez les nourrissons qui présentent l'anomalie courante du type C, lesquels constituent de mauvais risques opératoires.

INTESTINAL OBSTRUCTION BY A FECALITH IN A
MECKEL'S DIVERTICULUM*

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THIS report describes a rare complication of a Meckel's diverticulum. A fecalith, formed in the diverticulum, was extruded into the lumen of the small intestine causing complete obstruction.

The anomaly known as Meckel's diverticulum was reported by Lavater[1] in 1672 and Ruysch[2] in 1701. Littré[3] was the first to report the presence of a Meckel's diverticulum in a hernial sac. The diverticulum bears Meckel's name because of his detailed description[4] in which he discussed the complications as well as the origin and pathology of this anomaly.

About 15 to 25% of Meckel's diverticula are associated with complications. Intestinal obstruction in a patient with a Meckel's diverticulum is usually due to adhesions or the persistence of an omphalomesenteric duct. Intestinal obstruction due to an extruded fecalith is rare. In our review of the literature since 1940, we found reports of only two other such complications: one by Danzis[5] in 1950 and the other by Field and Field[6] in 1959.

CASE REPORT

A 58-year-old man consulted his physician about one month before admission because of cramping abdominal pain and nausea. At this examination a walnut-sized mass, which was tender and freely movable, was palpable in the right lower quadrant. On barium enema the appendix was visualized but there was no evidence of a colon lesion. Intravenous pyelo-

Fig. 1.—Lateral view of the right lower quadrant mass showing compressed small-bowel mucosa and some irregular streaks of barium which were thought to represent narrowed small-bowel segments or, possibly, fistulous tracts.

graphy was negative. A small-bowel study suggested the possible presence of regional enteritis (Fig. 1). During the month before admission he had only intermittent cramping and was able to work free of discomfort for long periods. On the day of admission, a dull pain developed which, in crescendo fashion, became severe. This soon passed off and he was asymptomatic for two days. On the third hospital day, continuous, severe abdominal pain returned and was accompanied by prominent distension and projectile vomiting. The distension was relieved considerably after a Cantor tube was passed. Subsequent radiographs suggested that the distal small bowel was completely obstructed (Fig. 2).

At laparotomy, the duodenum, jejunum and ileum were dilated to a point about 25 cm. proximal to the ileocecal valve. At this point, the ileum was completely obstructed by a fecalith whose diameter was greater that the normal diameter of the ileum. A Meckel's diverticulum was located about 8 cm. proximal to the fecolith.

Because of the size of the diverticulum and fecalith, almost 35 cm. of ileum had to be
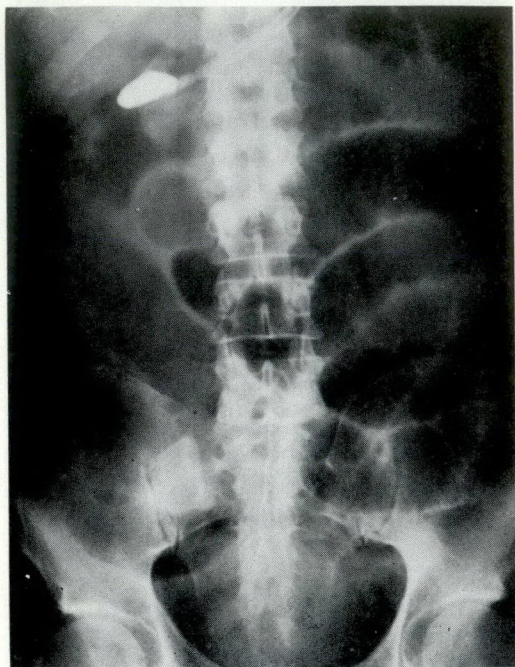
Fig. 2.—Supine film of the abdomen. The findings are highly suggestive of distal mechanical small-bowel obstruction.

resected. Intestinal continuity was restored by end-to-end anastomosis. The diverticulum

measured about 8 x 5 x 5 cm. and had an opening 3 x 3 cm. (Fig. 3). The fecalith, 5 x 5 x 4 cm., was firm and of the same configuration as the diverticulum. It had completely obstructed the ileum 8 cm. distal to the diverticulum. On microscopic examination of the ileum, areas of focal ulceration were seen surrounded by granulation tissue. The wall was edematous and the serosa thickened by connective tissue, fibrin and infiltrated neutrophils. The mucosa of the diverticulum also had some areas of ulceration and its serosa contained fibrin and neutrophils.

DISCUSSION

About 2% of the general population have a Meckel's diverticulum. Harkins[7] found the anomaly in 1.3% of 25,000 autopsies. According to Merritt and Rabe,[8] the incidence varies from 1 to 2.5%. The diagnosis of Meckel's diverticulum is usually made only after complications develop; of these, Greenblatt, Pund and Chaney[9] mentioned the following: inflammation, obstruction, peptic ulceration, and tumour, both benign and malignant. Oschner's classification[10] of the mechanisms of intestinal obstruction in Meckel's diverticulum is shown in Table I.
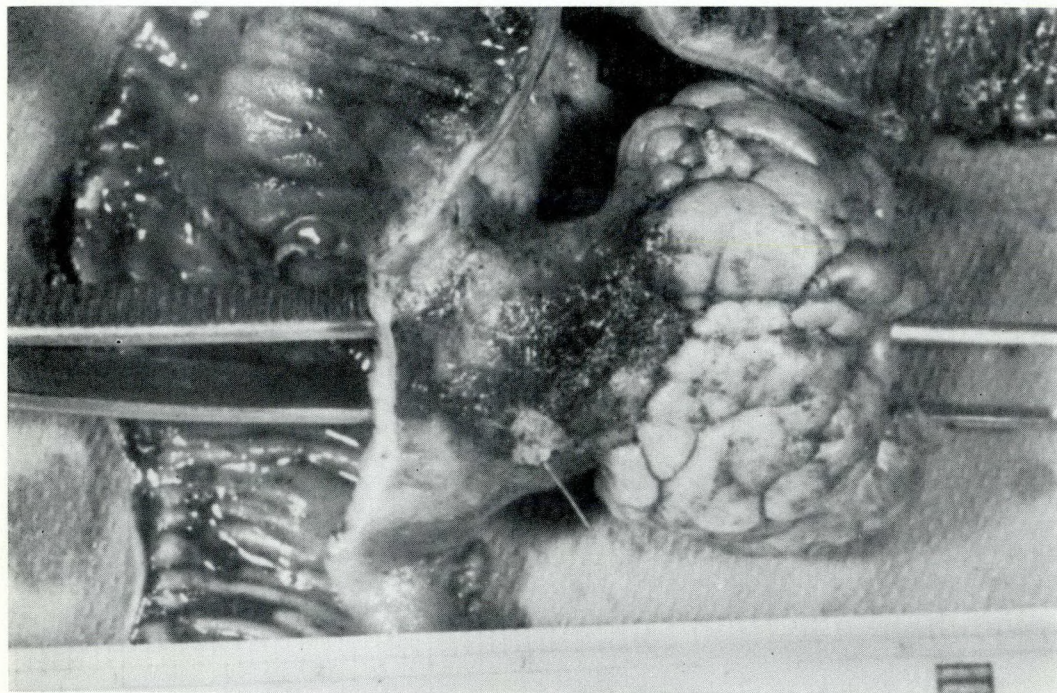
Fig. 3.—Giant Meckel's diverticulum in the middle of the resected ileum.

TABLE I.—Intestinal Obstruction Due to
Meckel's Diverticulum[10]

1. With free unattached diverticulum
   (a) Knot tied around the gut
   (b) Dragging and kinking of loop of intestine by distended or cystic diverticulum
   (c) Twisting of bowel at origin of diverticulum
   (d) Chronic inflammation of diverticulum and intestine with narrowing of ileum
   (e) Acute diverticulitis

2. Diverticulum attached to the abdominal wall or abdominal viscus
   (a) Band constricting or interfering with blood supply
   (b) Volvulus of loop of intestine passing under diverticulum and becoming twisted
   (c) Volvulus of intestine attached to diverticulum with point of attachment to fixed point of rotation
   (d) Acute diverticulitis
   (e) Prolapse of intestine through umbilical fistula

Taneja and Taneja[11] described 48 patients with Meckel's diverticulum of whom 23 had bowel obstruction. In their series, the mechanism of obstruction was volvulus, band, inflammation and adhesions. Haber[12] described 25 patients with Meckel's diverticulum with inflammation, ulceration, tumour and persistent bands; 7 of these had bowel obstruction. Bowel obstruction in these patients was due to the persistence of omphalomesenteric vessels, gangrene of a loop of small bowel and intussusception. When Meckel's diverticulum induces intussusception, a nodule of heterotopic gastric or pancreatic tissue usually forms the leading point.

Giant Meckel's diverticula have been described in the literature.[13] They may be as long as 15 cm. Generally, the mouth of the diverticulum is wide and active peristalsis usually keeps foreign material from forming or accumulating. Nevertheless, fecalith formation has been described.

Summary

We report a rare complication of a Meckel's diverticulum—intestinal obstruction by a fecalith. The fecalith arose in the diverticulum, was extruded into the lumen of the small intestine, passed to a point 8 cm. distal to the diverticulum, where it lodged causing obstruction.

In our review of the literature since 1940, we discovered only two other such cases.

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Résumé

Les auteurs présentent une rare complication d'un diverticule de Meckel. Un coprolithe sortant du diverticule provoquait une occlusion siégeant à 8 cm du diverticule jusqu'à 25 cm de la valve iléocécale. On réséqua un segment de 35 cm d'iléum. Cette résection portait sur le diverticule lui-même et sur la région sujette à l'occlusion. On procéda à une anastomose bout-à-bout. Dans la paroi du diverticule et dans l'iléum au point d'occlusion, existaient une ulcération fécale et de l'édème. La fréquence d'apparition du diverticule de Meckel varie entre 1 et 2% de la population. Le diagnostic n'est posé qu'après apparition des complications, par ex. inflammation, occlusion, ulcération peptique et tumeur. La pathogénie de l'occlusion, intestinale causée par le diverticule de Meckel est exposée par les auteurs. La fréquence de l'occlusion intestinale causée par un diverticule de Meckel varie, selon les groupes, de 25 à 50%. La littérature, passée en revue depuis 1940, n'a signalé que deux autres cas semblables.

FIBROUS POLYP OF THE VERUMONTANUM*

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IN the male, various abnormalities of the posterior urethra are associated with urinary tract obstruction; of these, hypertrophy and polyps of the verumontanum are uncommon causes of obstruction in children. Swinburne,[1] in 1910, called attention to significant enlargement of the verumontanum. Scattered reports of hypertrophy of the verumontanum, polyps of the verumontanum and polyps of the posterior urethra have appeared since that time. This report describes a fibrous polyp of the posterior urethra arising from the verumontanum.

CASE REPORT

A 2-year-old boy, with no significant past or family history, presented with a nine-month history of intermittent difficulty in voiding, manifested by straining and distress before voiding. Eight months before, a urethral meatal stenosis had been dilated without improving his symptoms. Four days before admission he developed a fever and his voiding difficulties increased. He had not passed urine for 12 hours.

On physical examination, the bladder was distended to a point midway between the pubis and umbilicus, and he was flushed and feverish. The external genitalia were normal and the prostate was normal on rectal examination. The blood pressure was 90/60 mm. Hg. The heart and lungs were normal.

The hemoglobin was 10.3 g./100 ml., leukocyte count was 14,000/c.mm. and blood urea nitrogen was 11 mg./100 ml. A catheter specimen of urine showed gross pyuria. Significant numbers of *Aerobacter aerogenes* were present on urine culture.

The upper urinary tracts were normal on intravenous pyelography. In late films, a smooth rounded filling defect was seen projecting from the floor of the bladder on the midline. The relationship of the filling defect to the vesical neck was more clearly demonstrated in the cystogram (Fig. 1).

At cystoscopic examination, a smooth pink pedunculated structure was seen in the pros-

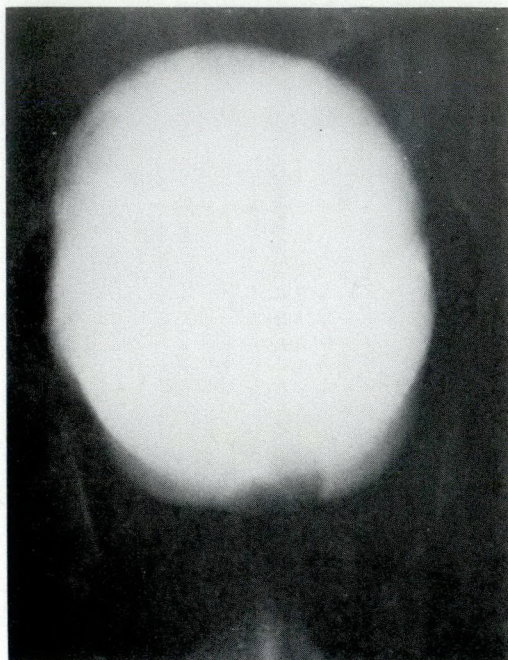Fig. 1.—The polyp at the vesical neck is seen in the retrograde cystogram as a smooth, rounded filling defect.

tatic urethra. Its base was in the area of the verumontanum and it extended just into the bladder through the vesical neck. The largest diameter was about 1 cm. The openings of

Fig. 2.—At operation, the bladder is open and the polyp can be seen in the depths of the wound, protruding through the vesical neck.

the ejaculatory ducts were seen at the base of the lesion.

A diagnosis of polyp or hypertrophy of the verumontanum was made.

At operation, a suprapubic approach was used. In Fig. 2, the proximal rounded end of the polyp can be seen through the operative incision. The polyp was 1 cm. in diameter at its widest point and 2.5 cm. in length. The stalk was attached to the dorsal wall of the posterior urethra at the verumontanum. The mass appeared to be a prolongation of the verumontanum. The stalk was ligated just above the opening of the ejaculatory ducts, divided and the polyp removed. Following suprapubic drainage, the child recovered without incident. He voided well at the time of discharge from hospital and has continued to do so.
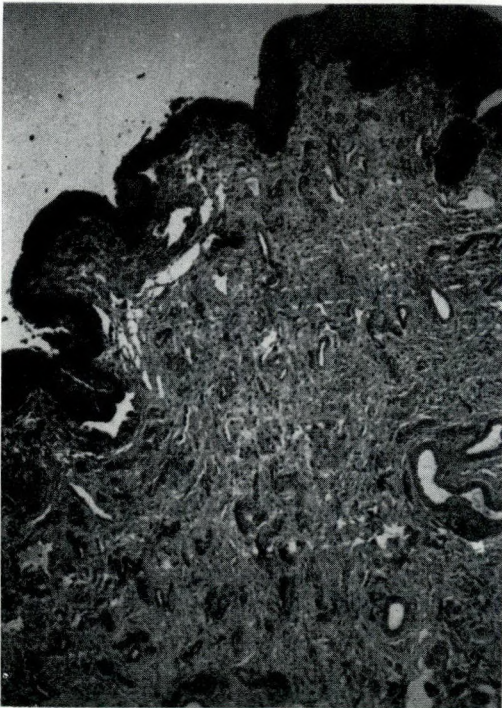

Fig. 3.—Microscopically the polyp consists of hyperplastic squamous epithelium without keratin, connective tissue core and numerous vessels. There are no glands or muscle fibres (original magnification x 75).

HISTOLOGY

The polyp was covered with hyperplastic squamous epithelium (Fig. 3). The epithelium was uniform. No keratinization or transitional cell epithelium was seen. The specimen was composed chiefly of loose fibrous tissue through which numerous vessels were scattered. A small amount of smooth muscle was present at the base. No glands or cystic structures were seen. Many small vascular and lymphatic spaces were also noted.

DISCUSSION

Similar lesions have been reported in recent urological literature and described as polyps of the posterior urethra. Williams and Abbassian[2] in 1966 described four patients with this lesion. Each had urinary infection and various degrees of obstruction of the upper urinary tract. On microscopic examination, the polyps had a core composed of loose connective tissue which contained many blood vessels but no glands. The external covering consisted of transitional cell epithelium which contained areas of squamous metaplasia and Brunn's nests. Muscle fibres were scarce. Flanagan, Kiefer and McDonald[3] reported a case with similar histology. In their case, Barrie and Simms[4] found considerable smooth muscle in the central portion of the polyp. The stroma contained glandular tissue but it was mucin secreting; the authors concluded, therefore, that these glands were not prostatic but probably ectopic Cowper's glands.

The fact that these polypoid structures arise from the verumontanum makes hypertrophy of the verumontanum a possible diagnosis. In his text, Campbell[5] says that, in congenital hypertrophy of the verumontanum, "the organ may appear as a large ball, but more often it is greatly elongated or enlarged and extends into the vesical outlet as a ball valve", wording that suggests a polypoid structure. Hypertrophy is generally defined as enlargement of an organ or tissue due to increase in size of the individual cells or components of the tissue. One expects to find histology similar to that of the original structure. Descriptions of the actual histology of the verumontanum are not easily found. We, therefore, made sections of seven prostates obtained at autopsy to obtain material for comparison. Six of these subjects were adults and one was a 12-year-old boy. In all seven, the verumontanum was composed of a core of smooth muscle and fibrous tissue in which multiple small

Fig. 4.—Section through a normal verumontanum in a 12-year-old boy. It is covered by transitional cell epithelium and there are numerous glands throughout the fibromuscular stroma (x 50).

glands were imbedded. The verumontanum was covered with the transitional cell epithelium of the prostatic urethra. The glands were lined with cuboidal or columnar epithelium (Fig. 4).

The term, "hypertrophy of the verumontanum", should be used to describe those enlargements of the structure which show histological increase in the various component tissues. The polypoid mass in this case had no glands and few muscle fibres. The bulk of the tumour was connective tissue. This is not the picture of hypertrophy of the verumontanum, and the polypoid shape and fibrous structure make it a typical "fibrous polyp".

The epithelial covering of the polyp consisted of well-differentiated but non-keratinized squamous epithelium. Other reported polyps have been covered with transitional cell epithelium although some have presented areas of squamous metaplasia. In 1940, Brody and Goldman[6] reported that they found squamous metaplasia in the epithelium of the prostatic glands and ducts, utricle and prostatic

urethra of male fetuses over 23 cm. in crown-heel length. This metaplasia disappears shortly after birth but may be seen as late as 2 months of age. They suggested that the metaplasia was due to the effect of maternal estrogens.

A polyp in the area of the verumontanum is probably formed following attempts at extrusion of the localized prominence by the muscular prostatic urethra. We propose that this polyp arose in the late fetal or neonatal period from the prominent metaplastic squamous epithelium of the verumontanum. The uniformity of the squamous epithelium supports this hypothesis.

SUMMARY

A fibrous polyp arising from the verumontanum in a 2-year-old boy has been described. It is suggested that this lesion can be differentiated from congenital hypertrophy of the verumontanum by its histological picture rather than its gross appearance. It may have been produced by a process analogous to squamous metaplasia of the transitional cell epithelium of the prostatic uretha seen in the newborn male child.

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RÉSUMÉ

Après un examen urologique courant comportant cystoscopie, pyélographie et cystographie, nous avons découvert chez un enfant de 2 ans, de sexe masculin, souffrant d'une occlusion urinaire basse et d'une infection urinaire concomi-

tante, une tumeur polypoïde provenant du veru montanum de l'urètre prostatique. Nous avons excisé ce polype, ce qui eut pour effet de lever l'occlusion. Depuis cette intervention, l'enfant se porte bien.

La structure de ce polypoïde se composait de tissu conjonctif couvert d'un épithélium à multiples squames. Des coupes histologiques faites sur le veru montanum chez six adultes et un garçonnet de 12 ans avaient montré que cette structure se compose de tissu fibreux, de muscle lisse et de multiples petites glandes recouvertes d'un épithé-lium de transition. Comme, dans le cas qui nous occupait, le polype ne contenait que du tissu conjonctif et des espaces vasculaires, nous en avons conclu qu'il s'agissait d'une polype fibreux plutôt que d'une "hypertrophie congénitale" du veru montanum.

Etant donné que ce polype était recouvert d'un épithélium squameux, il peut avoir été causé par une métaplasie squameuse qu'on rencontre parfois dans l'épithélium des glandes prostatiques et de l'urètre prostatique du nouveau-né de sexe masculin.

ANATOMY OF VAGAL SYSTEM

The findings of this study indicate that the fibres of each cervical nerve are distributed through the esophageal plexus to the entire stomach. The findings also suggest that the fibres of each cervical nerve are distributed equally to each wall of the stomach rather than the left cervical nerve favouring the anterior wall and the right nerve the posterior wall. However, it is noteworthy that when one surface secreted just before the other, the first surface to secrete corresponded to the conventional anatomic concept of the left anterior and the right posterior.

The results of stimulation of the esophageal plexus indicate that vagal innervation of the stomach becomes segmental at this level. However, the exact segment of stomach innervated by a specific branch of the plexus cannot be determined by this method because of the possibility that the artificial impulses may pass down one branch of the plexus and activate the other branch contributing to the trunk. This possibility is suggested by the similarity of the areas of secretion, both between each branch of the plexus and the trunk to which it contributes. On the other hand, the slightly different areas of secretion with stimulation of the branches of the plexus indicate that the fibres of each branch are not distributed to one entire surface of the stomach. In view of the fact that, proximally, at the cervical nerves, there is complete overlapping in ultimate distribution, whereas, distally, at the terminal gastric branches, there is but little overlapping in distribution, it seems reasonable to assume that at the esophageal plexus there is an intermediate degree of overlapping. From the esophageal plexus to the stomach, overlapping progressively decreases through the trunks and the gastric truncal divisions and the terminal gastric branches. At the stomach, therefore, innervation is quite segmental. Thus, the vagal system is not unlike innervation of the extremities by the spinal nerve roots, the brachial and lumbosacral plexuses, the peripheral nerves, and their terminal branches.

In all experiments in which a delayed response appeared, the cause of the delayed response was the vagal release of gastrin.

These results clearly disprove the thesis that any one vagal fibre may stimulate the entire gastric mucosa via connections with Meissner's submucosal plexus. The only fibres pertinent to this thesis are the right and left nerves above the esophageal plexus. However, by the vagal release of gastrin, the entire gastric mucosa may be stimulated by one branch of the esophageal plexus, or one trunk, or one gastric truncal division, or one of the more distal terminal branches; all of these fibres innervate the antrum. In contrast, a proximal terminal gastric branch to the fundus or corpus that does not innervate the antrum is not capable of stimulating the entire gastric mucosa but is capable of stimulating only its own small area of mucosa.

Segmental innervation of the stomach may well explain the variable adequacy of incomplete vagotomy associated with the variable secretory responses to insulin hypoglycemia as reported by Ross and Kay. A large and early response indicates an incomplete vagotomy with inadequate protection against recurrent ulcer, whereas a small and delayed response indicates an incomplete vagotomy with adequate protection. The basis for the difference between adequate and inadequate vagotomy is the difference in anatomic type of incomplete vagotomy.—Pritchard, G. R., Griffith, C. A. and Harkins, H. N.: A physiologic demonstration of the anatomic distribution of the vagal system to the stomach, *Surg. Gynec. Obstet.*, **126:** 791, 1968.

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BOOK REVIEWS

BOYD'S PATHOLOGY FOR THE SURGEON.
8th ed. William Anderson. 842 pp. Illust. W. B. Saunders Company, Philadelphia and London; McAinsh & Co. Limited, Toronto, 1967. $20.00.

It is difficult to make a modern assessment of this classic volume. The rewriting which characterizes the 8th Edition has done much to bring it up to date. Some of the more general chapters such as that concerned with inflammation and repair and the chapter on shock and burns suffer from the usual difficulty that an up-to-date statement on these topics is virtually impossible in a textbook.

The volume possesses much of the original charm of Dr. Boyd's vital use of language, and is as readable as is possible for technical material of a descriptive nature.

There need be no hesitation in recommending the book to students and practising surgeons alike. It is a pleasure to see the competent manner in which a volume, first published in 1925, has been rendered meaningful for the reader of 1968.

CLUB-FOOT. Alfred T. Fripp and Norman E. Shaw. 122 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1967. $6.25.

A periodical review of a subject such as club foot is necessary because so many theories have been advanced concerning its cause and so many methods of treatment have been recommended. In 114 pages, the present authors have succeeded in making sense out of a vast literature. The subject is introduced by a scholarly historical review and, in the chapters that follow, the pathology, radiological investigation and treatment are efficiently covered.

This book should be useful to students of orthopedics and practitioners caring for children with a club foot; nevertheless, some criticism is necessary.

The authors favour one theory of etiology and some forms of therapy over others, but do not support their choices in a convincing manner. For example, they believe "arrested development in embryonic life" is the most likely cause of club foot and discredit neurological causes because "failure of efforts to demonstrate neurological abnormalities makes a neurogenic theory untenable", even though much clinical evidence suggests that muscle imbalance plays an important role.

Because the severity of the deformity varies widely, evaluation of treatment of club foot is extremely difficult. These authors favour stretching and strapping rather than manipulation and immobilization in plaster, and their results are better than reported with the earlier method. However, it is not clear whether the less severe deformities were strapped and the more severe immobilized in plaster. Also, they dismiss tibialis posterior transfer without giving any evidence of their own experience with this procedure. They suggest that after a tibialis posterior transplant "the medial side of the foot *may* collapse" and "*may* result in cavus deformity". Other authors have not had this experience.

As a review of the field, this book is excellent, but it will hardly convince the experienced surgeon that the authors' choice of treatment is to be preferred above his own.

FATIGUE FRACTURES. A Clinical Study. James M. Morris and Loren D. Blickenstaff. 217 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1967. $18.50.

This is a clinical review of 700 patients with fatigue fractures of the lower extremity observed in infantry training centres. Additional stress fractures were collected from civilian practice, and some pertinent statistics have been culled from the available literature.

The authors discuss in detail fractures of the metatarsal bones and draw the reader's attention to the frequency of this type of injury in the os calcis. They give a classification of fatigue fractures of the tibia and femur.

Fatigue fractures of the neck of the femur, the authors think, should be treated by multiple-pin fixation. They point out that these fractures often take longer to heal than the ordinary traumatic lesion of the neck of the femur.

Towards the end of the book, chapters are devoted to fractures of the upper extremity commonly seen in Olympic contenders, javelin and discus throwers. There is also a chapter on cough fractures of the ribs. Finally, a portion of the book is devoted to exhaustion fractures of the vertebral arches of the back.

This 200-page book is well written, and has numerous and excellent illustrations. I would highly recommend this book. It could be read easily in an evening.

LOCAL ANALGESIA: BRACHIAL PLEXUS. 4th ed. Sir Robert MacIntosh and William W. Mushin. 62 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1967. $2.75.

This small book is one of a series of monographs on the use of local analgesia in various areas. It is restricted to the technique and methods of obtaining brachial plexus anesthesia.

Line drawings, photographs, and some diagrammatic sketches show the position and extent of the brachial plexus, and the surrounding soft tissues that may be injured in this technique. All in all, this small monograph

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is so well written and clearly illustrated that even an inexperienced person should be able to produce perfectly adequate analgesia and anesthesia using the methods described. This book is recommended to interns, general practitioners, and specialists who may be required to deal with injuries and other lesions of the upper limb.

MICRO-VASCULAR SURGERY. Report of First Conference, October 6-7, 1966, Mary Fletcher Hospital, Burlington, Vermont. Edited by R. M. Peardon Donaghy and M. Gazi Yasargil. 171 pp. Illust. Intercontinental Medical Book Corp., New York; Georg Thieme Verlag, Stuttgart, West Germany, The C. V. Mosby Company, St. Louis, 1967. DM 48,00. $12.50 (approx.). Paperbound.

This volume describes a Conference on Microvascular Surgery, held by the University of Vermont at the Mary Fletcher Hospital, Burlington, Vermont. The conference was held to discuss the problems encountered in operating upon small blood vessels 1 to 3 mm. in diameter.

Prefaced by an excellent introduction, the report includes 23 papers by many of the leading workers in microvascular procedures. The first 17 papers are largely devoted to principles and techniques, including the laser, electrocoaptation, the microstapler, adhesive substances and suturing techniques using the operating microscope. Other interesting papers describe microbipolar coagulation and the implications of the vasa vasorum in microsurgery. General discussions review the historical development of the subject. The final six articles describe clinical microvascular experience in neurosurgery.

The conference has not considered microsurgery as a separate field but as an accessory technique for dealing satisfactorily with small structures. The choice of subjects is good and the volume gives an effective review of the current work. At the moment the techniques have limited practical application in urology, vascular, plastic and neurosurgery but there are exciting possibilities, particularly in the latter specialty.

The papers are readable and well illustrated and include useful bibliographies. This volume should be of interest to any surgeon who has to anastomose small tubular organs.

MILESTONES IN MIDWIFERY. Walter Radcliffe. 110 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1967. $3.95. Paperbound.

This fascinating account of the development of obstetrics is given with grace and humour. Despite its unpretentious title, it gives a reasonably complete description of the evolution of obstetric practice within the compass of only 100 pages. Although attention is paid to the achievements of great men, Chamberlen, Smellie, Beaudelocque, Mauriceau and so on, the names of those less well known are also included. However, this is no mere catalogue, because the contribution of each to the advancement of the art is adequately described. Happily, the author has added, at the close of each chapter, generous but relevant quotations from the writings of the men whose work he has been considering. For example, we are given Guillemeau's description of internal podalic version for placenta previa, performed on the daughter of Ambroise Paré (himself the exponent of the maneuver), and Smellie's indications for the use of forceps, in a letter to one of his pupils. Modern advances are intentionally omitted, although Lovset's maneuver and Kielland's forceps are mentioned. Although published as a paperback, the book has a stiff cover which bears the reproduction of an old woodcut. The typography is pleasing and the book contains eight admirable plates from classic obstetric works, including one from William Hunter's famous atlas.

This is a book that every obstetric practitioner will own and read with delight. It should also be available to nurses on every maternity ward, to whom it will afford insight into the progress of midwifery to its present status.

PLASTICS IN SURGERY. Bernard Bloch and Garth W. Hastings. 187 pp. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1967. $13.00.

This book has been written by an orthopedic surgeon and by a polymer scientist. The surgeon was one of the pioneers in the use of plastic materials in orthopedic surgery, especially in the treatment of fractures. These attempts, which have largely passed into medical history, take up too much space in the present book. The short introduction into polymer chemistry entitled "Know Your Plastics" is excellent for the physician who needs to know some of the basic facts of polymer science. The individual polymers are reviewed in the subsequent chapter and the last two chapters are devoted to a description of the toxicity of plastics and of the sterilization of plastic materials.

The book gives a good description of the basic facts concerning the use of plastics in surgery. It can be recommended for the beginner in this field. Unfortunately, the book is not well written. The presentation is careless at times and there are many inaccuracies, three of which will be mentioned: on page 24, the name of the English physicist Hooke is mis-spelled; the statement on page 34, that synovial fluid "is less viscous at low rates of shear", is not universally accepted; on page 123, reference 56 is missing. As a Canadian, the reviewer regrets the almost complete absence of references to work done in Canada, especially that described in papers published in the *Canadian Medical Association Journal* and in the *Canadian Journal of Surgery*.

**THE SURGICAL RELIEF OF PAIN IN ARTH-
RITIC DISEASE. The Hip and Knee Joints.
Robert A. Herfort. 97 pp. Illust. Charles C
Thomas, Publisher, Springfield, Ill.; The Ryer-
son Press, Toronto, 1967. $9.00.**

This monograph describes a technique of
surgical denervation designed for the relief of
previously intractable pain in the arthritic hip
and knee. The lumbar sympathetic trunk is
resected beginning superiorly at the level of
the second lumbar vertebra and proceeding
as far distally as the superior margin of the
common iliac vessels. The accessory ganglia
on the rami communicantes are removed and
the decussating fibres in the prevertebral
plexus are divided by multiple cross-hatch
incisions over the anterolateral aspect of the
third and fourth lumbar vertebra, following
mobilization and retraction of the inferior
vena cava and the aorta. The author claims
that, of 69 patients with intractable arthritic
pain of the hip treated using this technique,
67 had complete or major relief of hip pain
on the first postoperative day. Because he
failed to relieve pain in all his patients, the
author modified the procedure. His current
operation for severe arthritic pain of the hip
consists of combined perifemoral adventitial
stripping and extended lumbar sympathectomy.
In 32 patients so operated upon and evaluated
subjectively up to 24 months later, all 32 pa-
tients are said to have achieved significant or
major relief of pain.

The author has employed the same operation
for the relief of severe arthritic knee joint pain.

The author is dealing with a very difficult
group of patients and appears to have achieved
a high measure of success. However, the results
of operation are only briefly described and
while numerous case reports are provided to
illustrate results, the overall results are not
adequately documented. This surgical tech-
nique may have a place in the management
of these patients, but this monograph is not
convincing.

This book should be in the reference library
but is recommended only to those with a
special interest in this particular branch of
surgery.

**SYNOVECTOMY AND ARTHROPLASTY IN
RHEUMATOID ARTHRITIS. 2nd International
Symposium January 27th-29th 1967 in Basle,
Switzerland. Edited by G. Chapchal. 131 pp.
Illust. Intercontinental Medical Corp., New
York; Georg Thieme Verlag, Stuttgart, West**

**Germany, 1967. DM 38,00. $10.35 (approx.).
Paperbound.**

This volume, which consists of the papers
given at the 2nd International Symposium on
rheumatology held in Basle in 1967, should
be in the hands of every rheumatologist and
orthopedic surgeon. A wealth of information
relating to current problems is set out in a
familiar fashion. Current theories of etiology
are described in a way that will be under-
stood by the surgeon. A comforting note about
the psychiatric attitudes of rheumatoid pa-
tients suggests they are on our side.

Some excellent reviews from large eastern
European centres and from the U.S.S.R. con-
firm the undoubted worth of early synovectomy
of the knee and confirm our own anxieties
about cup arthroplasty. The term "revision"
is used constantly in relation to this operation.
Total hip replacement may be better. (In
parts of Europe, there are no legal restrictions
on the use of self-curing cements for implant
fixation.)

As reported in this symposium, the results
of hemiarthroplasty and of arthroplasty of the
knee, using either the Canadian MacIntosh
or the British Platt prosthesis, leave something
to be desired. The reader would probably
want to hear more about total knee replace-
ment and of synovectomy combined with
osteotomy, particularly where early movement
had been facilitated by some type of internal
fixation. Certainly this approach appears to be
worth while in other joints, especially the hip.

The many fine papers in this volume that
describe surgery of the rheumatoid hand
should be considered a "must" for study. Sur-
gical enthusiasm is nicely modified by con-
tributions from the rheumatologists. The study
of Barbara Ansell, from Taplow, on arthro-
plasty of the metacarpal phalangeal joints is
a gem.

Throughout, the reviewer was worried by
the absence of any experimental work on
synovectomy and synovial regeneration, and
especially by the complete omission (with the
single exception of a paper by Savill of Edin-
burgh) of any primary studies of the natural
history of this disease. Surely operative treat-
ment should be more firmly based on knowl-
edge of the disease?

Despite these criticisms, this symposium can
still be viewed as a significant contribution
to the practice of rheumatology, and one
would hope that a meeting similar to the
Basle conference can soon be arranged in
North America.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

An Atlas of Advanced Surgical Techniques. Edward J. Beattie, Jr. and Steven G. Economou. 422 pp. Illust. W. B. Saunders Company Canada Limited, Toronto, 1968. $21.60.

An Atlas of Surgery of the Face, Mouth, and Neck. Robin M. Rankow. 292 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada, Limited, Toronto, 1968. $27.00.

Bedsores. B. N. Bailey. 130 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1967. $8.50.

The Blood Supply of the Lower Limb Bones in Man. Henry V. Crock. 104 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1967. $16.80.

Cancer Therapy by Integrated Radiation and Operation. Edited by Benjamin F. Rush, Jr. and Robert H. Greenlaw. 167 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $15.25.

Die Chirurgische Naht. P. F. Nockemann. 145 pp. Illust. Intercontinental Medical Book Corp., New York; Georg Thieme Verlag, Stuttgart, West Germany, 1968. DM 25,00. $6.80 (approx.). Paperbound.

Diseases of the Ear. 2nd ed. Stuart R. Mawson. 554 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1967. $16.00.

Exploration of the Abdomen. John W. Bassett. 109 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1967. $8.00.

Life After Fifty: The Prostatic Age. Henry M. Weyrauch. 175 pp. Illust. The Ward Ritchie Press, Los Angeles, 1967. $6.95.

Mécanique du Coeur et des Artères. L. Vadot. 254 pp. Illust. Expansion Scientifique Française, Paris, 1967. 78F. $17.20 (approx.).

Milestones in Midwifery. Walter Radcliffe. 110 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1967. $3.95. Paperbound.

Modern Trends in Ophthalmology. 4th series. Edited by Arnold Sorsby. 272 pp. Illust. Butterworth & Co. (Publishers) Ltd., London; Butterworth & Co. (Canada) Ltd., Toronto, 1967. $15.75.

An Outline of Orthopedic Surgery. A Concise Handbook of Basic Orthopedics, Fractures and Dislocations. Paul S. Derian. 122 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $9.00.

Physiotherapy in Obstetrics. 3rd ed. Maria Ebner. 158 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1967. $3.60.

A Practical Guide to the Care of the Injured. P. S. London. 777 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1967. $25.50.

A Practical Manual for the Treatment of Burns. 2nd ed. Eli Rush Crews. 234 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1967. $15.00.

Principes et Pratique de Médecine Hyperbare. J. H. Baixe. 187 pp. Illust. Expansion Scientifique Française, Paris, 1967. Price not stated. Paperbound.

Principles and Practice of Podiatry. Frank Weinstein. 508 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1968. $24.75.

Psychological Aspects of Surgery. International Psychiatry Clinics, vol. 4, no. 2. Edited by Harry S. Abram. 208 pp. Little, Brown and Company, Boston; J. B. Lippincott Company of Canada Ltd., Toronto, 1967. $9.25.

Radium Dosage. The Manchester System. 2nd ed. Edited by W. J. Meredith. 134 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1967. $6.75.

Rehabilitation in Extremity Fractures. Eugene Moskowitz. 150 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $10.25.

Retaining Ligaments of the Digits of the Hand. Gross and Microscopic Anatomic Study. Lee W. Milford, Jr. 60 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $14.05.

Surgery in Infancy and Childhood. 2nd ed. W. M. Dennison. 597 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1967. $11.25.

Surgery of the Aged and Debilitated Patient. Edited by John H. Powers. 611 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $20.55.

The Surgical Management of Rheumatoid Arthritis. Robert L. Preston. 579 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $25.40.

Torticollis in Infancy and Childhood. Sternomastoid Fibrosis and the Sternomastoid "Tumour". Peter G. Jones. 138 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $15.00.

The Treatment of Carcinoma of the Breast. Proceedings of a Symposium held at Gonville & Caius College, Cambridge, 9th September 1967. Edited by Antony S. Jarrett. 64 pp. Illust. Published for Syntex Pharmaceuticals Ltd., Maidenhead, England by Excerpta Medica Foundation; H. K. Lewis & Co. Ltd., London, 1968. 14s. $1.82 (approx.). Paperbound.