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L. E. DESJARDINS (1837-1919): UN DES PIONNIERS DE L'OPHTALMOLOGIE AU CANADA FRANCAIS

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L'OPHTALMOLOGIE a été la première spécialité chirurgicale reconnue au Canada. Son histoire a fait l'objet de nombreux écrits, dont les principaux sont ceux de C. M. Boissonnault,[1] Robert Craik,[2] J. J. Heagerty, [3, 4] Maude E. Abbott,[5] H. E. MacDermot,[6, 7] J. A. MacMillan[8] et L. Daniel Mignault.[9]

Les renseignements puisés à ces sources, sont parfois incomplets ou variables quant aux dates; dans le passé, le souci de l'exactitude mathématique était secondaire et les normes de la méthodologie historique n'étaient pas toujours respectées.

Au Canada français, les premiers oculistes furent le doyen Louis-Joseph-Alfred Simard de Québec et Louis Edouard Desjardins (Fig. 1) de l'Hôtel-Dieu et de l'Institut Ophtalmique de Montréal. Le docteur Simard a été le premier médecin à montrer une prédilection pour l'oculistique. Ancien élève de Laval, il suivit des cours à Louvain et à Paris; de retour à Québec en 1864, il fut chargé d'une multitude de cours: anatomie, pathologie générale, physiologie, histoire de la médecine, déontologie médicale et ophtalmologie.

Il est facile d'imaginer que les fonctions que le docteur Simard occupait à la Faculté, dont il fut doyen de 1899 à 1905, le retenaient fréquemment hors de sa clinique de la rue Saint-Louis et l'empêchaient de consacrer à sa spécialité la totalité de ses activités.

Le docteur L. E. Desjardins fut le premier médecin de Montréal qui décida dès 1869 de s'orienter exclusivement en spécialité. Médecin de l'Hôtel-Dieu, il y eut la charge de consultant en ophtalmologie dès 1871. Desjardins fut deux fois titulaire de la chaire d'ophtalmologie, à la Succursale de Laval et à l'Ecole Victoria; il fonda en plus un dispensaire pour les maladies oculaires à l'Institut Nazareth.[10] Il traita

Fig. 1.—L. Edouard Desjardins, professeur d'ophtalmologie à l'Ecole de Médecine et de Chirurgie de Montréal.

798 malades à cette clinique du 19 avril 1873 au 1 mai 1874.

On lit même dans un journal belge la note suivante: "Les Instituts ophtalmiques s'élèvent dans toutes les parties du monde. Il vient de s'en créer un à Montréal (Canada) sous la direction du docteur Desjardins que l'on a vu naguère au Congrès de Londres où il s'était rendu au prix d'un voyage long et coûteux pour s'y retremper aux sources de l'ophtalmologie européenne.[11]

Le docteur Frank Buller du Montreal General Hospital fut le premier oculiste anglophone de Montréal. Chargé de cours à McGill en 1878 il fut nommé titulaire en 1883. Frank Buller avait fait ses études médicales au Collège Victoria de Toronto; il se rendit ensuite en Europe, où il servit

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Fig. 2.—Photographie prise à Rome en novembre 1869 d'un groupe de membres du clergé se rendant au Concile I du Vatican et du docteur L. E. Desjardins, venu à Rome pour suivre les cliniques ophtalmologiques. La traversée de l'Atlantique se fit à l'aller de Québec à Londres, à bord du S.S. *Nestorian;* le départ eut lieu le 23 octobre 1869. Ledocteur Desjardins porte le no. 6 sur la photo.

dans le corps médical allemand pendant la guerre franco-prussienne de 1870. A la cessation des hostilités, il occupa à Londres le poste de "house surgeon" au Royal London Ophthalmic Hospital, dit Moorfield's Eye Hospital; sur les instances du docteur Shepherd, il accepta la direction du service ophtalmique du Montreal General Hospital. Les noms de Buller et de William Osler, ont toujours été associés, car Buller, pendant plusieurs années, donna asile à Osler dans sa maison de la rue Sainte-Catherine. Frank Buller n'eut pas la réputation littéraire d'Osler, mais il savait à l'occasion exprimer ses pensées crûment. A preuve, ces mots: "Let me tell you first of all that the student who leaves college and immediately goes abroad to study some specialty for six months or a year during which time he merely attends the clinics and then returns home and starts as a specialist, is nothing short of an imposter, a superficial narrow-minded, ill-trained egoist, too ignorant to understand his own incapacity."[12]

L'Union Médicale du Canada a, dès 1872, donné à Desjardins l'hospitalité de ses pages; il en profita pour publier ses

observations personnelles sur les progrès accomplis en ophtalmologie chez L. de Wecker, Sichel et Galezowski. C'était la confirmation officielle de l'entrée de Desjardins dans le domaine particulier de l'ophtalmologie. Jusqu'à hier, tout médecin, qui se jugeait qualifié, pouvait traiter les maladies oculaires et opérer les cataractes ou le glaucome.[13]

Desjardins, l'aîné d'une famille nombreuse, naquit à Terrebonne, en 1837, l'année sombre. Le climat, qui prévalait à l'époque sur les coquets villages qui longeaient la Rivière des Mille-Iles, n'a sûrement pas entravé la destinée des Prévost, des Masson, de L. O. Taillon, d'Adolphe Chapleau, d'Alphonse Desjardins et de ses frères Edouard et Guillaume-Henri. Le soulèvement des gens de Terrebonne fut minime à côté de la rébellion des habitants de Saint-Denis, de Saint-Charles, de Saint-Benoît et de Saint-Eustache. Il se termina par la signature d'une entente entre l'autorité et les patriotes que L. O. David a rappelée sur un mode sarcastique: "Cette convention eut pour effet de rétablir la paix et l'ordre dans le comté de Terrebonne, mais n'empêche que deux mois après, Bouc, Rochon, Leclaire, Gravelle, Roussin et St-Louis étaient arrêtés, subissaient leur procès et étaient condamnés à être pendus."[14]

Desjardins commença ses études secondaires au Collège Masson de Terrebonne et il les termina au Séminaire de Nicolet. Il s'inscrivit en 1860 à l'Ecole de Médecine et de Chirurgie de Montréal; il eut la bonne fortune de faire sa cléricature chez le docteur Jean-Gaspard Bibaud, auprès de qui il demeura comme collaborateur, après 1864, date de sa licence. Il ouvrit alors son cabinet dans le faubourg Saint-Antoine de Montréal, à l'ombre de la nouvelle Cathédrale. Dévoué et sympathique, il se créa tôt une clientèle fidèle, mais mis en présence quotidiennement d'affections oculaires fréquentes chez ses malades ouvriers, il se sentit attiré dans une voie jusqu'alors inexplorée: la spécialisation oculaire. Passionné par les problèmes de l'oculistique, il décida d'aller en Europe acquérir la science qui lui faisait défaut. Il s'embarqua pour la France en 1869; il fut bien accueilli par les maîtres dont il réclama le privilège de suivre les cliniques. Auprès d'eux, il enrichit ses connaissances d'ophtalmologie et il connut les meilleures techniques chirurgicales (Fig. 2).

Desjardins fut admis à l'Hôtel-Dieu en 1870 et au retour de son troisième voyage outre-mer, celui de 1873, l'hôpital lui conféra le titre de chef du service d'ophtalmologie. Les registres de la salle d'opération mentionnent que, fin octobre et début de novembre 1875, il pratiqua une ablation de cataracte, une énucléation oculaire et trois iridectomies (Fig. 3). Peu auparavant l'Institut Nazareth avait été fondé par les Sœurs Grises pour venir en aide aux aveugles. Le docteur Desjardins y fut admis comme consultant médical; il examinait les patients à l'Institut et les opérait à l'Hôtel-Dieu.

Les archives des Sœurs Grises contiennent deux lettres qui établissent la réputation solide de Desjardins: "Vous savez sans doute que le Dr Desjardins est revenu d'Europe avec une renommée des plus célèbres pour la guérison des maux d'yeux. Il en a fait des études toutes spéciales et depuis son arrivée, il soigne avec beaucoup de succès. Il a ouvert ou il doit ouvrir sous peu une espèce de dispensaire à Nazareth, où il soignera les pauvres gratis."*

Un mois plus tard, une autre lettre mentionnait que: "La petite Sœur Moffat de Lawrence est sous les soins de notre célèbre oculiste, le Dr Desjardins; il espère guérir ses yeux, mais il dit que ce sera long."†

Desjardins fut en 1879 nommé par l'Université Laval professeur titulaire d'ophtalmologie; pour une courte période cependant, car les événements qui suivirent l'installation de la Succursale de Laval à Montréal conduisirent à une mésentente complète. A la suite du président Pierre Munro et de ses collègues de l'Ecole Vic-

*Extrait d'une lettre écrite par Sœur Roy, secrétaire, le 23 avril 1873 et adressée aux Sœurs Grises de Toledo, Ohio. Copie de cette lettre est conservée aux Archives Générales des Sœurs Grises à Pierrefonds.

†Extrait d'une lettre écrite par Sœur Stubinger, secrétaire de la Supérieure Générale des Sœurs Grises de Montréal, en date du 28 mai 1873 et adressée aux Sœurs de Saint Boniface, Manitoba. Copie conservée aux Archives Générales des Sœurs Grises à Pierrefonds.

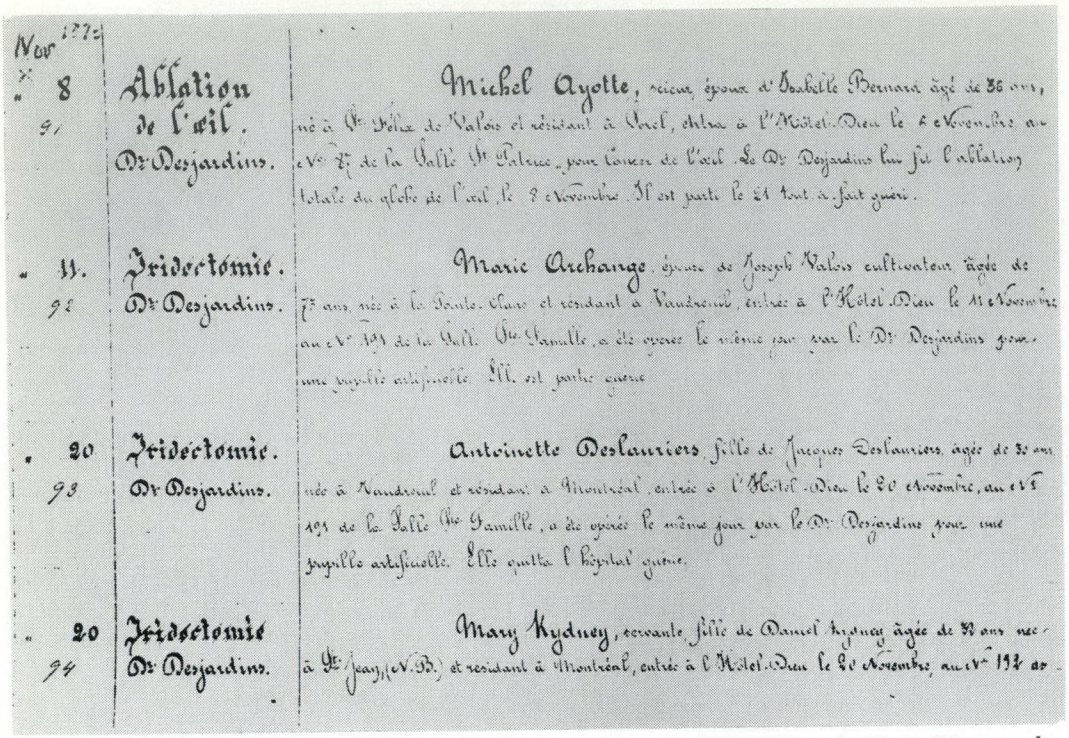

Fig. 3.—Photocopie du registre de la salle d'opération de l'Hôtel-Dieu 8, 11 et 20 novembre 1873.

toria (Fig. 4), Desjardins cessa d'appartenir au corps professoral de Laval pour continuer son allégeance à l'Ecole de Médecine et à l'Hôtel-Dieu.

Le service d'ophtalmologie de ce dernier hôpital ne chômait guère; les statistiques opératoires démontrent que du 1er juillet 1879 par exemple au 1er juillet 1880, le docteur Desjardins a opéré 59 cataractes, fait 36 iridectomies et pratiqué 6 énucléations d'un œil.

Les activités d'Edouard Desjardins débordaient le champ de ses seuls actes professionnels; professeur d'ophtalmologie, il avait, depuis 1872, participé avec intérêt et dévouement à l'enseignement de sa spécialité. Titulaire et membre du Conseil de l'Ecole de Médecine, il prit dès le début de la querelle entre Laval et Victoria une part active aux négociations, aux missions diplomatiques à Rome, Québec et Montréal.

Dès 1876, Desjardins porta un nouveau fardeau; celui de délégué officiel de l'Ecole auprès des autorités chargées de résoudre le problème universitaire: Laval versus Victoria.

L'Institut ophtalmique fut fondé en 1883, par les Sœurs Grises; Louis Edouard Desjardins et son frère Guillaume-Henri en furent les spécialistes attitrés; malheureusement à l'été de 1883, survint la menace d'excommunication suspendue sur la tête des professeurs de l'Ecole, de leurs élèves et des Communautés religieuses qui les recevraient. Desjardins apprit un jour qu'il serait sous peu considéré comme rebelle, s'il ne se soumettait pas aux injonctions des autorités religieuses et que, dans ce cas, le dispensaire ophtalmique ne pourrait dorénavant plus l'accueillir.

Les relations étaient tendues entre les deux facultés francophones de médecine à Montréal; alors qu'en 1879, une entente paraissait conclue, survinrent des complications qui, au début semblaient solubles, mais qui à la longue s'avérèrent irréductibles. Le nœud du problème résidait dans le désir de l'Université Laval de Quebec d'absorber l'Ecole de Médecine Victoria; cette dernière avait toutefois des droits de priorité, puisqu'elle œuvrait à Montréal depuis 1843.

La rivalité entre Québec et Montréal, les conflits de personnalités, le durcisse-

Fig. 4.—Le dernier groupe des professeurs de l'Ecole de Médecine Victoria et des finissants avant la fusion avec la Succursale de Laval à Montréal.

ment bilatéral des positions conduisirent à une rupture. Il y eut menace de poursuites judiciaires contre Laval par l'Ecole de Médecine qui contestait la légalité de la Succursale de Montréal. Les appels à Londres, où Laval espérait voir confirmer ses droits à l'affiliation d'instituts provinciaux de haut savoir, le bill de Laval présenté et adopté par la Législature pour corriger les lacunes de la Charte Royale de Laval, les mémoires présentés à Rome tant par Québec que par Montréal, les décrets de 1876, 1881 et 1883 acceptés par l'Ecole sous caution, les délégations à Ottawa, à Londres et à Rome, la mission Conroy, les polémiques violentes entre ceux qui réclamaient au nom de la justice et de l'équité et ceux qui leur répondaient par: "Roma locuta est; causa finita est," tous ces démêlés avaient conduit à une attitude intransigeante. Devant la position inébranlable des membres de l'Ecole, il y eut à l'été de 1883 une menace d'exclusion de l'Eglise des membres de l'Ecole qui refusaient de se soumettre. La situation était grave. Desjardins offrit de se rendre à Rome plaider la cause de l'Ecole et de ses serviteurs accusés d'insubordination. Il quitta Montréal, muni des documents qui attestaient l'authenticité de sa mission. Il se rendit immédiatement à Rome, fort de l'appui de l'évêque de Trois-Rivières, Monseigneur Laflèche, du sénateur Landry, de l'abbé Luc Désilets et de conseillers en droit canon. Desjardins s'isola dans un bureau de l'Hôtel Aliberti entouré de ses consultants. Avec leur concours, il rédigea un mémoire des difficultés encourues qu'il présenta au Cardinal Simeoni. Non sans peine, il gagna la partie, puisque le 24 août 1883, arrivait au Canada le câblogramme historique qui se lisait, selon la traduction

française: "L'Ecole a proposé au Pape une réconciliation — Suspendez tout — Que l'Ecole continue l'année qui vient – J'écrirai — Simeoni." A toutes fins pratiques, la menace d'excommunication était relevée par Rome. Modeste, Desjardins attendit en Europe que le calme revienne au pays natal avant de retourner à son domicile de la rue Bleury. Arrivé à l'automne de 1883, son retour passa inaperçu, car il coïncidait avec l'arrivée du Commissaire dom Smeulders. Le rôle de Desjardins n'était pas terminé, car il fut choisi avec le docteur d'Orsonnens pour présenter au délégué les revendications de l'Ecole; ils eurent aussi mission de contresigner l'entente qui mit enfin d'accord, Laval et Victoria.

Ophtalmologiste renommé, professeur compétent, Desjardins fut invité à présider la première séance du Congrès de la Société française d'Ophtalmologie qui se tint à Paris du 2 au 5 mai 1904. Il y présenta deux travaux, l'un sur l'étendue de la vision chez les sourds-muets et l'autre sur un cas exceptionnel de corps étranger (fer) enfoncé dans la région du corps ciliaire depuis 20 ans.[15, 16]

Tempérament ardent, Desjardins fut toujours d'attaque; ceci lui valut des ripostes parfois violentes de ses adversaires. Malgré la vivacité de ses réactions, il sut conserver le respect et la sympathie de ses adversaires. Il combattit avec loyauté et "la délicatesse de ses procédés laissa intacte l'amitié qui l'unissait à ses opposants . . . Franc comme l'épée du roi, il supposait la même franchise chez les autres".[17] Il crut toute sa vie à l'honnêteté, à la droiture, au respect de l'équité et de la justice. Sa science, son sens pédagogique, son dévouement à la cause de l'éducation en firent un chef d'école. Ses élèves furent nombreux; A. A. Foucher, fondateur du Service d'Ophtalmologie à Notre-Dame, lui dédia son magnifique Traité d'ophtalmo-oto-rhino-laryngologie, Jules Jehin-Prume qui exerça à New-York, lui fit le même honneur pour une de ses plaquettes. Chrétien-Zaugg, Albert Lasalle et J. N. Roy lui furent également redevables de leur formation.

Pionnier de l'ophtalmologie à Montréal, professeur écouté, diplomate averti et heureux, Desjardins avait un tempérament d'artiste, il adorait la musique et il occupait ses loisirs à l'harmonisation de vieilles chansons canadiennes. Sous le pseudonyme de "Bon Vieux Temps", il publia 14 séries de ces chansons réunies en pots-pourris.[18] Il fut aussi très attiré par la musique religieuse. A l'occasion du centenaire du Séminaire de Nicolet, il écrivit un messe de minuit sur de vieux airs de Noël.[19] Chantée à l'Eglise Saint-Jean-Baptiste d'Ottawa, elle se mérita les remarques suivantes. "If midnight mass had not its seal of piety one would have been under the same melancholy impression while listening to the old Christmas airs of which Dr. Desjardins' mass is composed; all would have, like the French poet, 'bowed their heads on finding them so old'. Some had completely escaped the memory, while for others, it was a joy to seek and discover through the variety of harmonies, the waving of which always disguises, a little, the natural air, while enriching it. One remarked especially the end of the Gloria which contains a phrase of a very pretty style on the theme, 'Il est né le divin enfant,' and the 'Incarnatus Est,' set with admirable art to the air of 'Adeste Fideles'. One of Dr. Desjardins' principal points of merit consists in his taste, and happy choice of the Christmas airs which by their tone and movement are best adapted to the thought expressed."[20]

Plus tôt, Desjardins monta une opérette: "l'Amour Médecin", qu'il fit jouer dans la salle de musique du Saint Lawrence Hall; à la maison (Fig. 5), il ne se lassait pas d'entendre les duos qui réunissaient la basse L. O. Taillon, ancien premier ministre du Québec, et la soprano coloratura Hortense Villeneuve. La carrière du professeur Desjardins a été évaluée en termes mesurés par le vice-recteur de l'Université Laval de Montréal, quand il a dit: "Le départ de ce maître est ressenti par tous avec le même regret. Nous perdons un spécialiste tenu depuis longtemps en haute estime dans le monde scientifique, un professeur méthodique et disert, un cœur de père pour ses étudiants dont il se faisait autant d'amis. Ouvrier de la première heure, le docteur Desjardins fut toujours un fervent de notre œuvre universitaire, pour laquelle il a, dans plusieurs circons-

Fig. 5.—Maison, maintenant centenaire, située sur l'avenue Saint-Louis de Terrebonne, où durant 50 ans le docteur Desjardins passa ses vacances à poursuivre son *hobby* de compositeur.

tances difficiles, travaillé ferme et remporté des victoires décisives. Dès sa jeunesse, me disait un condisciple de son cher collège de Nicolet, il s'est montré un solide et fier chrétien, le plus aimable comme le plus joyeux des compagnons."[21]

Tel fut le premier ophtalmologiste francophone de Montréal et l'un des pionniers de l'oculistique au Canada.

Bibliographie

1. Boissonnault, C. M.: Histoire de la Faculté de Médecine de Laval, Les Presses de l'Université Laval, Québec, 1953, p. 280.
2. Craik, R.: Papers and addresses, Gazette Printing Co., Montréal, 1907, p. 114.
3. Heagerty, J. J.: Four centuries of medical history in Canada and a sketch of the medical history of Newfoundland, The Macmillan Company of Canada Ltd., Toronto, 1928.
4. Heagerty, J. J.: The romance of medicine in Canada, The Ryerson Press, Toronto, 1940.
5. Abbott, M. E.: History of medicine in the province of Quebec, McGill University Press, Montréal, 1931.
6. MacDermot, H. E.: One hundred years of medicine in Canada, McClelland & Stewart Ltd., Toronto, 1967.
7. MacDermot, H. E.: History of the Montreal General Hospital, Montreal General Hospital, Montréal, 1950.
8. MacMillan, J. A.: Note on the history of ophthalmology in Canada, *Amer. J. Ophthal.*, **31:** 199, 1948.
9. Mignault, L.-D.: Revue historique: histoire de l'Ecole de Médecine et de Chirurgie de Montréal, *Un. Méd. Canada,* **55:** 597, 1926.
10. Editorial: Les spécialités, *Un. Méd. Canada,* **3:** 280, 1874.
11. Editorial: Les instituts ophtalmiques, *Ann. Oculist.* (*Paris*), **36:** mars-avril, 1874.
12. Buller, F.: Valedictory address. Faculty of Medicine, McGill University, 12th June, 1903, *Montreal Medical Journal,* **32:** 500, 1903.
13. Desjardins, E.: Ophtalmologie, *Un. Méd. Canada,* **1:** 50, 1872.
14. David, L.-O.: Les événements de 1838, *L'Opinion Publique* (*Montréal*), **10:** 433, 1879.
15. Desjardins, L. E.: Sa vision chez les sourds-muets, *Bull. Soc. Franç. Ophtal.*, **24:** 184, 1904.
16. Desjardins, L. E.: Corps étranger du corps ciliaire, *Bull. Soc. Franç. Ophtal.*, **24:** 458, 1904.
17. Lecompte, E.: Le docteur Edouard Desjardins, *La Vie Nouvelle* (*Montréal*), **15:** 74, 1932.
18. Desjardins, L. E.: Chansons populaires au Canada harmonisées par "Bon Vieux Temps", Crevel Frères, Paris.
19. Desjardins, L. E.: Messe de minuit composée sur des airs de Noël, Imp. Moderne, Montréal, 1902.
20. *Ottawa Citizen,* 27 décembre 1903.
21. Dauth, G.: Discours à l'ouverture des cours. *In*: Annuaire, 1909-10, édité par Arbour et Dupont, Université Laval de Montréal, Montréal, 1909, p. 229.

ORIGINAL ARTICLES

LYMPHANGIOSARCOMA AND BREAST CANCER*

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LYMPHANGIOSARCOMA is a distressing, lethal complication of the primary treatment of breast cancer complicated by the swollen arm. Although only 76 cases have been described so far, this tumour has been examined in a number of excellent reviews.[1-4] Lymphangiosarcoma arises from the lymphatic capillaries which proliferate in an attempt to remove the excess fluid from the arm. A similar tumour has been described in association with edematous limbs from other causes.[1, 5] Stewart and Treves,[2] in 1948, described a patient who developed multiple cutaneous lesions on the arm and chest wall several years after a radical mastectomy that was uncomplicated except for lymphedema. One of these lesions, which was biopsied, was described as "Kaposi's sarcoma". Because certain features precluded complete acceptance of this diagnosis, these authors reviewed the records of women in their breast clinic who had had a radical mastectomy complicated by a swollen arm and found six more with similar cutaneous lesions, which they took to be a type of angiosarcoma. These tumours appeared, on the average, 12½ years after the breast operation.

The purpose of the present paper is to describe seven more women with this association—lymphangiosarcoma and breast cancer. These seven cases, which represent the total experience of the Ontario Cancer Institute with this lesion since 1946, are previously unreported. Because this complication is difficult to treat, I have analyzed the experience of the Institute with three objectives: to find some common factors in its cause; to propose methods of prevention; and to make recommendations concerning therapy. The Ontario Cancer Institute is primarily a radiation-therapy centre and the management of these patients was shared between the Institute staff and the referring surgeons.

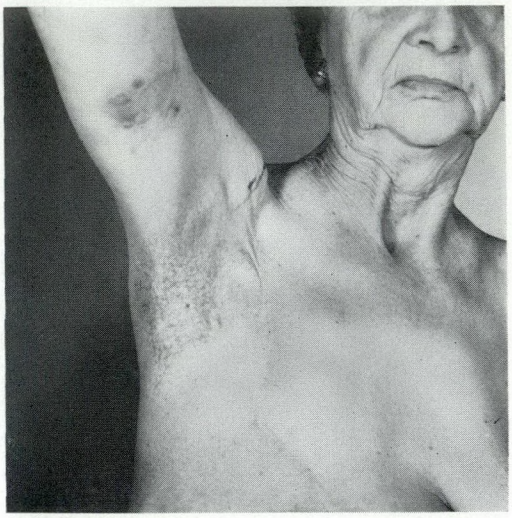

Fig. 1.—Tumour of the upper inner arm, the commonest site of origin for lymphangiosarcoma.

Typically, these lesions are encountered clinically in the following setting: a well patient, on her annual follow-up, is found to have a raised, bluish area on the upper arm (Figs. 1 and 2)—an arm that has been

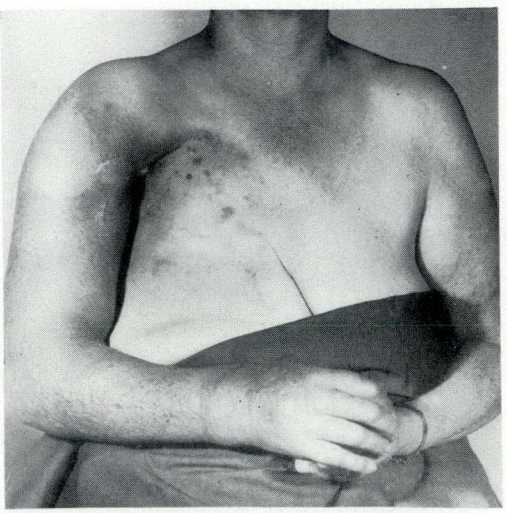

Fig. 2.—Lymphangiosarcoma involving the whole arm and chest wall.

*Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Toronto, Ont., January 1968.

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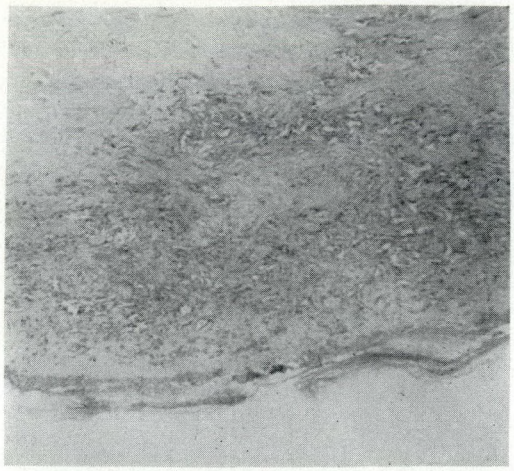

Fig. 3.—Photomicrograph of a biopsy section showing the relation of tumour to skin (original magnification x 45).

swollen since the initial breast treatment and often has limited movement. On casual examination this area might be mistaken for a bruise, but it is indurated and often contains multiple, purplish-red nodules. Subsequently, over a period of weeks or months, satellite tumours appear and it is usually at this stage that a biopsy is taken and a definitive diagnosis made. Untreated old lesions ulcerate, new ones appear and they spread, involving the whole arm and often the chest wall. These lymphangiosarcomas metastasize to distant sites, particularly the lungs. Irrespective of treatment, most of these patients succumb within three years.

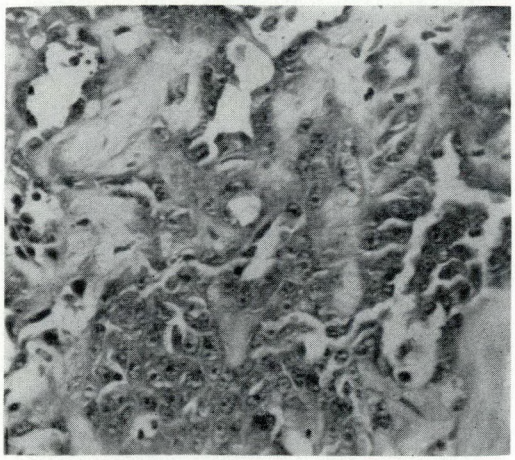

Fig. 4.—Photomicrograph showing a typical tumour with new thin-walled vessels lined by malignant endothelial cells which infiltrate the tissues (original magnification x 256).

The differential diagnosis rests between trauma, Kaposi's hemorrhagic sarcoma, malignant melanoma, recurrent breast cancer and lymphangiosarcoma.

The tissues that have been edematous for a long time show increased amounts of collagen tissue together with proliferating lymphatics and small blood vessels (Fig. 3). There is perivascular lymphocytic infiltration; both lymphatic and vascular capillaries contain red blood cells giving the early tumour its characteristic bruised appearance. The thin-walled vessels are lined by both normal and malignant endothelial cells, the latter infiltrating adjacent tissues (Fig. 4). Hemorrhages occur and the pleomorphic cells, which show frequent mitoses, may mimic anaplastic carcinoma. The tumour is of multifocal origin and the early invasion of vessels leads to widespread metastases.

Clinical Material

The details of stage, treatment and results of treatment in our seven patients are shown in Table I: in six, the breast tumours were staged according to the Steinthal principles—five were Stage I and one was Stage II. In all these patients lymphangiosarcoma developed in the swollen arm following the primary treatment of breast cancer. They were among 9019 patients treated at the Ontario Cancer Institute between 1946 and 1956; however, all were found among the 3000 patients registered in the decade before 1956. Thus, the incidence of lymphangiosarcoma following breast cancer is low, but more such cases can be anticipated from the group treated since 1956 because of the long latent period before lymphangiosarcoma develops.

Of the seven patients, all but one were thought to have been cured of their breast tumour; Mrs. M.C. (Case 6) developed an axillary recurrence 16 years after her initial treatment. Each patient had had a radical mastectomy and postoperative irradiation to the chest wall and regional nodal areas; Mrs. L.M. (Case 5) had preoperative irradiation in addition. In all the operations, the pectoral muscles and axillary contents were excised; unfortunately, postoperative complications could not be assessed. The radiation doses ranged from 4000 to 6500

TABLE I.—Summary of Seven Cases Showing the Relationship Between Breast Cancer and Lymphangiosarcoma

	Breast tumour					*Lymphangiosarcoma*		
Case no.	*Stage*	*Treatment*		*Edema**	*Result (years)*	*Age (years)*	*First site*	*Survival from time of diagnosis (months)*
		Operation	*Radiation*					
1	I right	Radical mastectomy	Postoperative	+++	No evidence of disease 11	78	Upper inner arm	34
2	– right	"	"	++	" 8	61	"	82
3	I right	"	"	++	" 8	72	"	34
4	I right left	" "	" "	+++ +	" 9	76	"	9
5	II left	"	Preoperative plus postoperative	++	" 10	66	"	15
6	I right	"	Postoperative	+Upper arm only	First recurrence, right axilla 15	73	"	22
7	I left	"	"	+++	No evidence of disease 9	64	Upper outer arm	34

*+ = mild; ++ = moderate; +++ = severe.

rads in two to six weeks; all these women developed radiation changes in skin and soft tissue. In all but Mrs. M.C. (Case 6), lymphedema was moderately severe or severe. The mean age at radical mastectomy was 58 and the mean latent period from primary breast treatment to the appearance of lymphangiosarcoma was nine years. In six, the first tumour was on the upper inner arm, and in one, on the upper outer arm. All died of their second malignancy after a median survival of 34 months from diagnosis; only Mrs. M.C. (Case 6) had proved evidence of recurrent breast cancer when she died.

Analysis of Treatment

The treatment of these seven patients is summarized in Table II. In each patient. the lymphangiosarcoma recurred quickly within a few months and in an area or areas adjacent to the primary site. Subsequently, metastases appeared in the skin, particularly the chest wall, and in the lungs, liver and bones (Table III). Unfortunately, because none of these patients died in the Princess

TABLE II.—First Planned Treatment of Lymphangiosarcoma With Result and Further Management

Case no.	*Planned treatment*	*First recurrence*			*Survival from time of diagnosis (months)*
		Months	*Site*	*Treatment*	
1	XT arm	3 3	Axilla plus chest wall Lungs	XT XT	34
2	Excision plus graft (18 months from first symptom)	3	Upper arm plus graft	XT	82
3	XT upper arm	1	Mid-arm	XT	34
4	"	1	Forearm	XT	9
5	XT arm	8	Arm	XT	15
6	"	2	Right axilla	Excision plus graft	22
7	"	6	Upper arm	XT	34

XT = radiation.

TABLE III.—Further Recurrences of Lymphangiosarcoma and Treatment

Case no.	*Subsequent treatment*	*Further recurrence*	*Survival from time of diagnosis (months)*
1	XT, stilbestrol	Bone, skin, lungs, liver	34
2	XT, vinblastine sulfate	Forearm, chest wall, lungs, liver	82
3	Amputation, XT, thio-tepa	Forearm, arm, chest wall, lungs, liver	34
4	Nitrogen mustard	Arm, chest wall, lungs, bone	9
5	XT	Arm, chest wall, lungs	15
6	—	Lungs, liver	22
7	XT, amputation	Upper arm, chest wall, lungs, liver	34

XT = radiation.

Margaret Hospital, Toronto, and none came to autopsy, we cannot establish the total extent of disease at death.

Forequarter amputation was considered in four patients but carried out in only two because of the anticipated clinical course of the chest-wall recurrence and the appearance of distant metastases. Mrs. V.M. (Case 7, Fig. 5) is worthy of comment because after two years, the lymphangiosarcoma, although it had recurred several times, was still confined to the edematous limb. After full investigation, including whole-lung tomography, which showed no evidence of spread beyond the limb, forequarter amputation was undertaken in the hope of cure. The incision healed promptly and the patient adjusted well, although she developed symptoms of phantom limb. Within two months of operation, however, she had a recurrence on the lateral chest wall (Fig. 6) and, a few months later, she developed metastases in lungs and liver. She died eight months after the forequarter amputation.

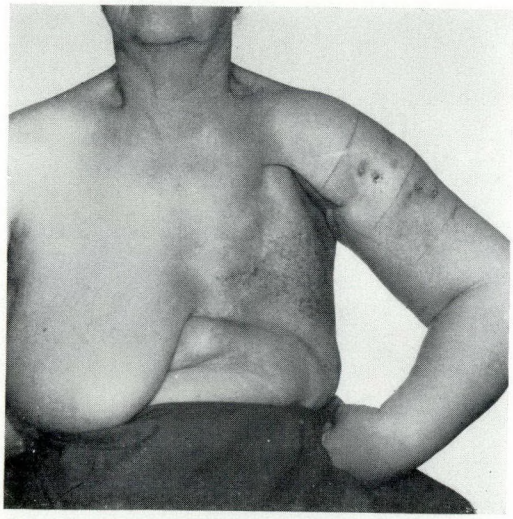

Fig. 5.—Case 7. Lymphangiosarcoma of the upper outer arm. Note the radiation changes in the skin and severe lymphedema.

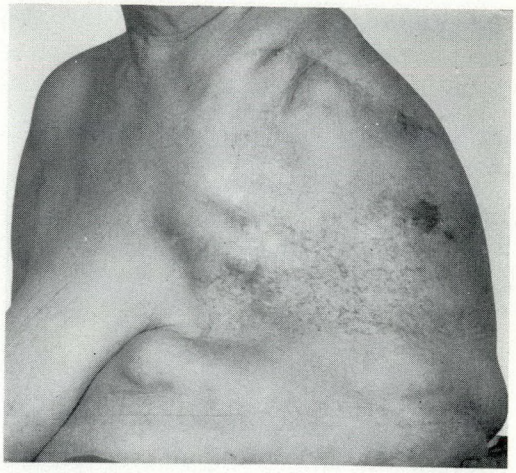

Fig. 6.—Case 7. Recurrence on the chest wall two months after forequarter amputation.

Mrs. A.S. (Case 2) had a different clinical pattern. Although her tumour was the same histologically, it behaved differently from those found in the other six patients. She did not have any treatment until 18 months after her first symptom of lymphangiosarcoma and it was 72 months before a recurrence appeared in the axilla, which then spread rapidly to involve the whole arm, chest wall and distant organs. This patient survived for 82 months from first treatment and 100 months from the first symptom.

Chemotherapy was used in three patients in a somewhat random manner, the agents including vinblastine sulfate, thiotepa and nitrogen mustard, but the dosage and schedules used produced no benefit.

Discussion and Conclusions

Only 6 of 76 patients with lymphangiosarcoma following radical mastectomy for breast cancer reported in the literature survived for more than five years without evidence of tumour following treatment.[6] Of these six, two had been treated solely by irradiation, one by local excision and three by extensive surgical procedures. One of the irradiated patients was treated with

intra-arterial injection of radioactive yttrium microspheres. I do not know the eventual outcome in these patients. With the exception of these six patients, the medial survival period was 18.6 months after the onset of the sarcoma.[6] The reported results are mainly in average figures and for comparative purposes our statistics are averaged too, but the more meaningful median survival figure is included. In all 76 patients the average age at which the breast cancer was treated was 53 years; the lymphangiosarcoma developed nine years later at 62 years—a latent period that varied from 16 months to 24 years. In the seven women in our series, the average age at the time of radical mastectomy was 60 years; the lymphangiosarcoma appeared at 70 years after a latent period of 10 years.

TABLE IV.—Summary of Results

	Number of cases	*Five-year survivors*	*Median survival* (months)*
Princess Margaret Hospital	7	1	34
Literature	76	5	18.6

*Time from diagnosis, which coincided with the first treatment for lymphangiosarcoma.

The median survival from the diagnosis of the lymphangiosarcoma was 34 months. One patient, however, lived more than five years (Table IV). In the literature, the few recorded long-term survivors must have had biologically more benign tumours and been similar to our one long-term survivor, Mrs. A.S. (Case 2).

The etiology of this type of lymphangiosarcoma is unknown but chronic edema is probably significant. The phases of its development are probably as follows: prolonged lymphedema during which lymphatics proliferate to cope with the excessive fluid in the arm; and, as in other hyperplastic conditions, the multiplying cells occasionally escape from the body's control and a malignant process ensues. Six of our patients had marked lymphedema which developed soon after the mastectomy. In this context it is interesting that angiosarcomas have been described in limbs edematous from other causes (Fig. 7).[1,5] Stewart and Treves[2] postulated that a systemic carcinogen was operating because four of six patients in their series had other tumours. As far as I can determine none of our patients had other tumours in addition to breast cancer and lymphangiosarcoma.

Because lymphangiosarcoma is considered to be incurable by any present mode of therapy, attention must be focused on the best type of palliation. In the past, palliative treatment, including various surgical resections, radiation and chemotherapy, has given disappointing results. In our experience none of the treatments altered the eventual prognosis although irradiation produced some local control. Our median survival was nearly twice as long as those reported in the literature although the series is too small to be of any statistical significance. In view of the fact that the tumour involves the whole arm, limited treatment is useless. Because the tumour is moderately radiosensitive, I recommend that radiation be given using large fields that include the whole arm from wrist to shoulder. A dose that is free of significant morbidity is 3500 rads to the whole arm given in three weeks, supplemented by 1500 rads in a single treatment to the known tumour sites, bringing the tumour dose to 5000 rads. Limited irradiation can be repeated and useful palliation obtained. Because the tumour is multifocal, local excision is pointless and wide excision with skin graft difficult because of edema. Forequarter amputation is formidable and, in my opinion, when there is no reasonable chance of cure, should be done only when

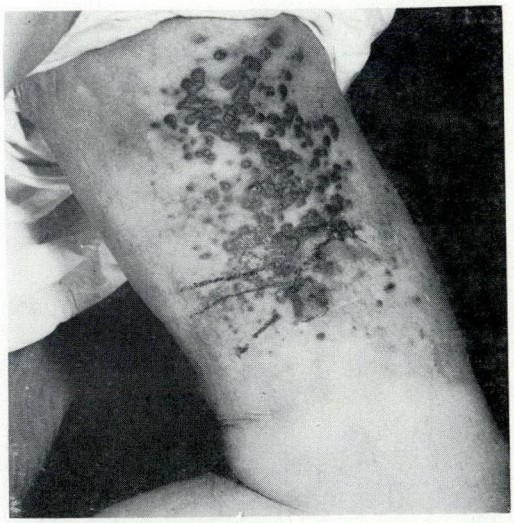

Fig. 7.—Lymphangiosarcoma in an edematous leg of many years' duration.

the patient has a painful, useless limb. It seems unlikely that chemotherapy will be effective, although in the light of recent information[7] the drug, dosage and regimen used in our patients is now considered to be inadequate. Following administration of chemotherapeutic agents, Eby, Brennan and Fine[8] reported that these tumours regressed but this improvement lasted less than two months.

Lymphangiosarcoma can be prevented by avoiding lymphedema. In early breast cancer, the prognosis is independent of the method of treatment:[9-11] equal results are achieved by radical mastectomy, modified radical mastectomy, simple mastectomy or local excision with postoperative irradiation. Lymphedema, a serious complication of treatment, is more likely to follow Halsted's classical operation, and the risk of this complication increases if the axilla is irradiated postoperatively. The incidence of lymphedema after radical mastectomy ranges from 13% to 57%.[12] The swelling varies from a small increase in the width of the upper arm to gross swelling of the whole arm and hand, producing discomfort and interfering with movement and function. Chronic, massive edema of the arm is more common in women who have positive axillary nodes, delayed wound healing, secondary infection, obesity, or thrombophlebitis in addition to operation and radiotherapy. Thus, in treating curable breast cancer, we must be careful to avoid operative and radiotherapeutic techniques that lead to lymphedema and indirectly to the development of this highly malignant, lethal tumour.

My thanks go to the clinical staff of The Princess Margaret Hospital, Toronto, who have treated these patients and to Dr. T. C. Brown for providing the histological sections; to Mrs. Helen Worton and Miss Doris Hunter who performed the secretarial tasks admirably, and to members of the Department of Medical Photography who produced the slides and tables.

References

1. Taswell, H. F., Soule, E. H. and Coventry, M. B.: Lymphangiosarcoma arising in chronic lymphedematous extremities. Report of thirteen cases and review of literature, *J. Bone Joint Surg.* [*Amer.*], **44A:** 277, 1962.
2. Stewart, F. W. and Treves, N.: Lymphangiosarcoma in postmastectomy lymphedema; a report of 6 cases in elephantiasis chirurgica, *Cancer,* **1:** 64, 1948.
3. McConnell, E. M. and Haslam, P.: Angiosarcoma in post-mastectomy lymphoedema; a report of 5 cases and a review of the literature, *Brit. J. Surg.,* **46:** 322, 1959.
4. Fisher, J. H.: Postmastectomy lymphangiosarcoma in the lymphedematous arm: a review of four cases, *Canad. J. Surg.,* **8:** 350, 1965.
5. Whittle, R. J. M.: An angiosarcoma associated with an oedematous limb; a case report, *J. Fac. Radiol.* (*Lond.*), **10:** 111, 1959.
6. Herrmann, J. B. and Ariel, I. M.: Therapy of lymphangiosarcoma of the chronically edematous limb. Five-year cure of a patient treated by intra-arterial radioactive yttrium, *Amer. J. Roentgen.,* **99:** 393, 1967.
7. Bruce, R.: Normal and malignant stem cells and chemotherapy. *In*: The proliferation and spread of neoplastic cells, 21st Annual Symposium on Fundamentals of Cancer Research, 1967, The Williams & Wilkins Company, Baltimore, 1968, p. 409.
8. Eby, C. S., Brennan, M. J. and Fine, G.: Lymphangiosarcoma: a lethal complication of chronic lymphedema, *Arch. Surg.* (*Chicago*), **94:** 223, 1967.
9. Philip, J. F.: Results of treatment of breast cancer at Aberdeen Royal Infirmary 1940-1955, *Brit. Med. J.,* **1:** 323, 1967.
10. Hayward, J. L. and Atkins, H.: A comparison of radical mastectomy with wide excision in the treatment of early breast cancer, *Annals of the Royal College of Physicians and Surgeons of Canada,* **1:** 62, 1968 (abstract).
11. Peters, M. V.: Carcinoma of the breast. Stage II—radiation range. Wedge resection and irradiation. An effective treatment in early breast cancer, *J. A. M. A.,* **200:** 134, 1967.
12. Britton, R. C. and Nelson, P. A.: Causes and treatment of postmastectomy lymphedema of the arm: report of 114 cases, *J. A. M. A.,* **180:** 95, 1962.

Résumé

Le lymphangiosarcome, une complication rare du cancer du sein, apparaît dans le bras œdématié après mastectomie totale. On n'a jusqu'à présent rapporté que 75 cas. Le présent article fait état de sept nouveaux cas trouvés parmi les 9019 malades enregistrés au Ontario Cancer Institute entre 1946 et 1966. On fait remarquer que les sept cas se trouvaient tous parmi les 3000 premières malades, probablement parce que le lymphangiosarcome ne se manifeste qu'après une période latente de 10 ans. Six des sept malades paraissaient guéries de leur cancer du sein. La tumeur apparaît initialement sous forme d'ecchymose sur le bras. Ensuite des nodules multifocaux, gagnent l'avant-bras et la poitrine; des métastases peuvent se produire, surtout aux poumons. La survie moyenne, à partir du moment où le diagnostic de sarcome est établi, est de 34 mois. Seulement une malade a survécu plus de cinq ans. La chirurgie, la radiothérapie et la chimiothérapie n'ont guère influencé le cours de la maladie, mais la radiothérapie a été utile pour enrayer localement la tumeur. Les méthodes actuelles ne nous permettant pas de guérir le lymphangiosarcome, il nous faut trouver des moyens d'empêcher l'apparition de cette tumeur rare et fatale.

SIGNIFICANCE OF CONTRALATERAL AXILLARY METASTASES IN CARCINOMA OF THE BREAST*

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In patients with breast cancer, contralateral, axillary, lymph-node metastases have biological and therapeutic significance.

Biologically, such metastases indicate that there is direct lymph drainage from the breast to the opposite axilla. At the turn of the century, according to Haagensen,[1] Oelsner showed that, in two of nine patients, cutaneous lymphatics of the breast drained to the opposite axilla. Turner-Warwick[2] could not find such drainage in the normal breast, but Kreel and George[3] demonstrated contralateral, axillary lymph drainage after radical mastectomy. It has been suggested that lymph drains to the opposite axilla after the ipsilateral lymphatics become obstructed by tumour, operative procedures or irradiation,[2] and that malignant cells can spread through the deep lymphatic fascial plexus beneath the opposite breast.[1]

Therapeutically, no recommendations have been made by the few authors who have acknowledged the existence of these lesions.

Because so little information is available about the contralateral, axillary metastases of breast cancer, the experience of the Civic Hospital Division of the Ottawa Clinic of the Ontario Cancer Foundation was reviewed, seeking some understanding of the biological significance of such metastases. From such studies more appropriate therapy for this form of breast cancer may emerge.

Material and Method

The review included each of 1440 women reported to the Civic Hospital Division of the Ottawa Clinic of the Ontario Cancer Foundation in whom treatment for carcinoma of the breast was started between 1946 and 1961 (inclusive). The Clinic and these patients have been described in detail previously.[4-6] These patients can reasonably be regarded as a typical population of breast cancer victims.

Since contralateral, axillary, lymph-node metastases are seldom operated upon, and few of these patients came to autopsy, the diagnosis was based on the findings at follow-up clinical examinations. The examinations were done by the various radiotherapists, surgeons and internists who have made up the Clinic's breast cancer team since 1946. No patients were included in whom there was clinical evidence of a tumour in the associated breast.

Observations and Discussion

In 52 of these 1440 women (3.6%), contralateral, axillary, lymph-node metastases were recognized. Since the examination of the opposite axilla may have been cursory in some of the patients with advanced recurrent or metastatic disease, the incidence may have been higher. According to Haagensen,[1] contralateral, axillary metastases were recognized on clinical examination in 4.2% of patients with breast cancer.

TABLE I.—Contralateral, Axillary, Lymph-Node Metastases in Relation to Other Manifestations of Breast Cancer

	Number of patients
Simultaneous with primary tumour	2
At first recurrence	18
With local recurrence	5
With distant metastases	3
After local recurrence	14
After distant metastases	10

Table I shows that these lesions can appear simultaneously with the primary tumour, as first recurrences, at the time of other local recurrences or distant metastases, or after the appearance of other local recurrences or distant metastases.

Only 18 of the 1440 patients developed these metastases as the first reactivation of

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TABLE II.—Relation of Contralateral, Axillary Metastases (CAM) to the Initial Clinical Stage *

Presenting stage	*Incidence of CAM†* (%)	*Median time between initial treatment and development of CAM* (*months*)	(*range*)	*Median survival after CAM* (*months*)	(*range*)
I	(a) 2.3	74	5-120	24.0	3-75
II	(b) 3.9	32	6-76	10.5	2-47
III	(c) 5.6	9	3-79	8.0	1-48

*Two patients with new primary tumours and two Stage IV patients were excluded.
†a = 17 of 734; b = 14 of 355; c = 17 of 303.

the breast cancer. In six of these women, the remaining breast was removed even though there was no clinical evidence of tumour in it. A tumour was found on pathological examination in only two of these. (These two are excluded from all subsequent discussion of biological significance.) Thus, two-thirds of the patients who developed contralateral, axillary, lymph-node metastases had (or had had) other evidence of uncontrolled breast cancer.

The development of contralateral, axillary metastases was a particularly ominous event. All but three of the women have died, and the three survivors each have had other evidence of inadequately controlled breast cancer. The poor prognosis, however, is not due to the contralateral, axillary metastases because in one-third of the patients, these metastases appeared with or after other distant metastases, in one-third with or after local recurrences, and in only one-third were they the first evidence of reactivated disease. A similar observation has been made concerning homolateral, axillary, lymph-node recurrences.[5] Thus, contralateral, axillary, lymph-node metastases should be viewed as evidence of an adverse change in the tumour-host relationship, rather than as sites from which the further spread of breast cancer can occur.

Table II shows that patients presenting with lesions in the more advanced clinical stages had a higher incidence of contralateral, axillary, lymph-node metastases than did women with earlier lesions: the more advanced tumours developed earlier and the patients died sooner. This observation suggests that tumours that present in the more advanced clinical stages are biologically more "aggressive" than those in the less advanced stages. (This concept has been discussed elsewhere.[6]) Tumour aggressiveness, then, is an important factor in determining the occurrence and behaviour of contralateral, axillary, lymph-node metastases.

The late appearance of metastases in the opposite axilla in some patients and the long survival of some patients after such metastases emphasize the ability of some women to live "in peaceful coexistence" with incompletely controlled tumours. Late homolateral, axillary, lymph-node recurrences have also been described.[5]

The location, in the breast, of the original tumour bore no relation to the development of a contralateral, axillary metastasis. Table III shows that, in these 50

TABLE III.—Site of Original Tumours*

Site	*Number of patients*
Outer	28
Central (subareolar, 6 and 12 o'clock positions)	8
Inner	9
Whole	2
Unknown	3

*Two patients with new primary tumours excluded.

patients, the distribution of the primary tumours was normal.[1] If direct lymphatic spread from the skin of the inner one-half of the breast or the areola was the chief cause of contralateral, axillary metastases, most of the women in this series should have had medially located primary tumours.

Forty-nine of the 52 patients have died of their breast cancer. The remaining three have or have had other evidence of metastatic disease. Six patients in whom contralateral, axillary metastases were the first evidence of reactivation, underwent mastectomy even though no tumour was

palpable in the breast. In only two of these did the pathologist find a tumour; both of these women died of metastases—one after 8 months, the other after 16 months.

In view of the eventual outcome in these women, it is clear that patients who develop contralateral, axillary metastases cannot be cured. Therefore, if no tumour is palpable in the breast, mastectomy is contraindicated. Therapy—whether local resection, local radiotherapy, endocrine therapy, chemotherapy or masterful inactivity—is chosen that will provide the best palliation for the whole woman and her disease. In any event, these metastases are not sources of major morbidity and only simple measures are warranted.

Summary

Of 1440 patients with breast cancer followed for a minimum of six years, 52 developed contralateral, axillary, lymph-node metastases. These were detected on clinical examination.

"Tumour-host potential" appeared to be more important than direct lymphatic channels in determining the development of these metastases.

The treatment of patients with contralateral, axillary metastases should never be more than palliative.

References

1. Haagensen, C. D.: Diseases of the breast, W. B. Saunders Company, Philadelphia, 1956.
2. Turner-Warwick, R. T.: The lymphatics of the breast, *Brit. J. Surg.*, **46:** 574, 1959.
3. Kreel, L. and George, P.: Post-mastectomy lymphangiography: detection of metastases and edema, *Ann. Surg.*, **163:** 470, 1966.
4. Devitt, J. E. and Beattie, W. G.: The rational treatment of carcinoma of the breast?, *Ann. Surg.*, **160:** 71, 1964.
5. Devitt, J. E.: The significance of regional lymph node metastases in breast carcinoma, *Canad. Med. Ass. J.*, **93:** 289, 1965.
6. Devitt, J. E.: The clinical stages of breast cancer—what do they mean?, *Canad. Med. Ass. J.*, **97:** 1257, 1967.

Résumé

Sur les 1440 malades souffrant de cancer mammaire qui ont été suivies pendant au moins six ans, 52 ont présenté des métastases des ganglions axillaires contra-latéraux (MGAC) qui ont été diagnostiquées lors d'un examen clinique. Ces MGAC étaient survenues auparavant, coïncidant avec ou survenant après d'autres signes locaux ou éloignés d'une réactivation du cancer mammaire. On a noté que les malades qui souffraient de tumeurs primaires dans leurs phases tardives avaient plus de chances de présenter des MGAC, que celles-ci apparaissaient plus tôt et que la survie était abrégée. Chez aucune des malades ayant des MGAC, le cancer mammaire n'avait pu être enrayé. Ces métastases axillaires n'avaient aucune relation avec le siège de la lésion mammaire primaire. Les auteurs en ont conclu que le "potentiel tumeur-hôte" jouait un rôle plus important que celui des voies lymphatiques directes dans l'apparition de ces métastases. C'est pourquoi le traitement de ces cas ne doit jamais dépasser le stade de la palliation.

IN VITRO CONSERVATION OF THE KIDNEY

The only acceptable criterion to test the efficiency of a method of *in vitro* preservation of the kidney is the function of the implanted kidney in the same dog, removing the contralateral kidney at the time of implantation of the first kidney. The following preservation methods have been investigated: (1) hypothermia; (2) hypothermia combined with hyperbaric oxygen; (3) hypothermia and perfusion with Ringer's lactate solution; and (4) hypothermia and perfusion with diluted, homologous, oxygenated serum.

Even when the kidney is cooled to a temperature of 4°C., the metabolism of the isolated organ is not reduced to zero. After perfusion for 6, 17 or 24 hours, the kidneys are macroscopically normal. Histological examination 24 to 48 hours after implantation has shown abnormality in each case, vascular congestion, and arterial and arteriolar lesions. The best results have been obtained with the perfusion with oxygenated, diluted, homologous serum at 12°C. hypothermia, but even in this series only two of four dogs survived the procedure.—Wuyts, J. L. *et al.*: La conservation in vitro du rein. Etude expérimentale de quelques procédés, *Lyon Chir.*, **64:** 770, 1968.

NORMAL RANGES OF INTERVERTEBRAL-JOINT MOTION OF THE CERVICAL SPINE*

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ALTHOUGH considerable work has been done to record the total range of motion of the cervical spine, our knowledge of intervertebral-joint motion is still incomplete. Ferlic[1] developed a quantitative method for measuring the total range of motion in the cervical spine, using reflected shadows from the head when it was fitted with a metallic helmet. A pointer is fixed to the helmet which in turn is fixed to the subject's head. The patient stands before a horizontal and vertical grid and the range of motion is then recorded by measuring the deflection of the pointer during various movements of the neck. A bright light is used so that the movement of the pointer is measured by the oscillation of its shadow on the grid. Ferlic measured the range of motion in 199 subjects who had no cervical complaints. The age range in his study was between 15 and 74 years. His method is elaborate and although it is an excellent research tool, it is not convenient for office practice. He found that the total range of motion in flexion and extension was 127° (mean value), lateral bending 73°, and rotation 142° from right to left. Overall motion decreased by 21% during the six decades studied.

Jackson[2] described a clinical method of measuring the total range of motion in the cervical spine using a protractor attached to a headband (Cervigon MK 2). The total range of motion in various directions that she reported is approximately the same as that described by Ferlic.

Although these studies indicate the total range of movement in the cervical spine, the range of motion between the individual vertebral bodies at different levels has not been ascertained. The medical literature is lacking in this aspect of the normal anatomy of the cervical spine. Hohl[3] described the normal motions of the upper portion of the cervical spine at the atlanto-occipital and atlantoaxial joints. Fielding[4, 5] described normal and selected abnormal motions of the cervical spine from the second to the seventh cervical vertebra, based on cineradiography. He indicated that, occasionally, adults who have not had previous trauma or neck disability have more motion in flexion between the fifth and sixth cervical vertebrae than at other levels. Exact measurement of ranges of motion by cineradiography is difficult because the image on the screen is ever changing and magnified.

In recent years, greater emphasis has been placed on the movement at individual

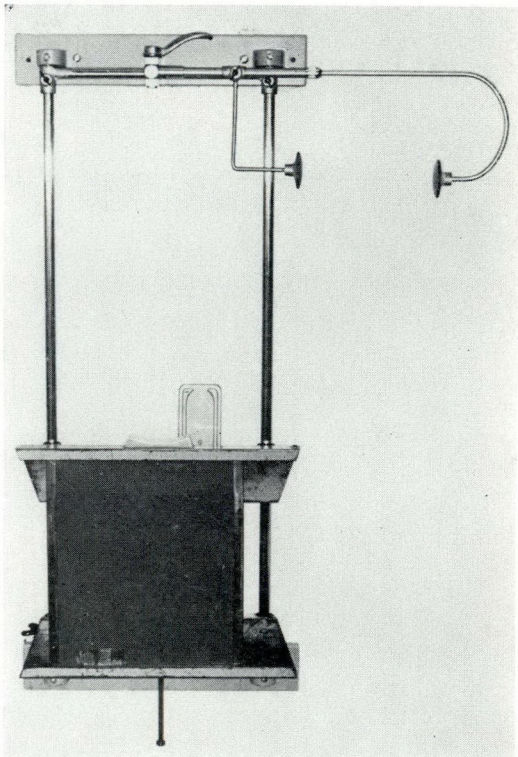

Fig. 1.—The head attachment for the roentgenogram plate stand; the plate holder slides into position.

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Supported by a grant from the Rehabilitation Foundation for the Disabled of Ontario, and the Toronto East General and Orthopaedic Hospital Research Foundation.

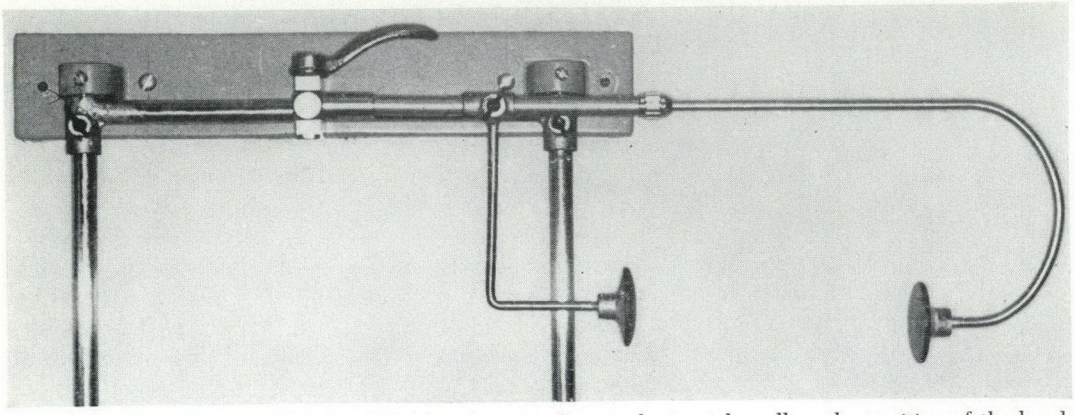

Fig. 2.—Close-up of the head holder showing the attachments that allow the position of the head to be adjusted.

levels. Excessive movement has been used as a criterion to identify the level or levels from which symptoms are arising, and thus to indicate the site for fusion.[6] For this reason, the following clinical and experimental study was carried out to calculate the normal range of motion at individual levels.

The purpose of this study was to record, objectively, the normal ranges of motion at the different intervertebral-joint levels of the cervical spine from C2-T1. This we

Fig. 3a

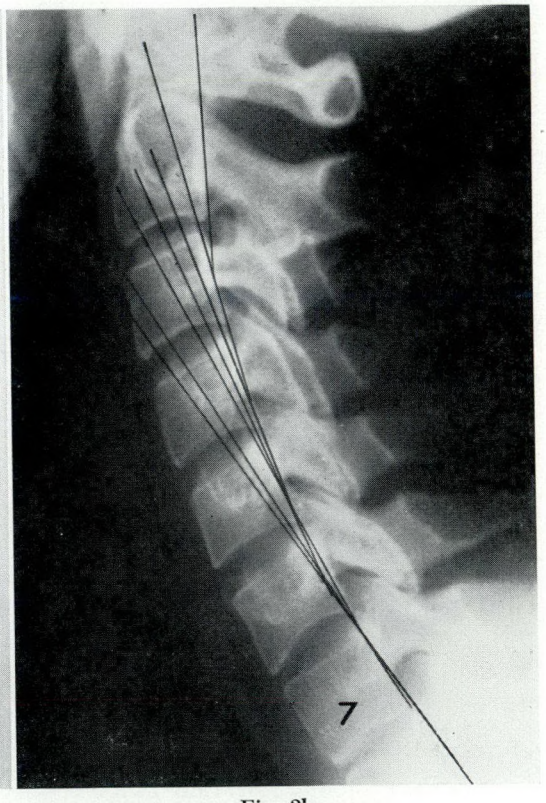

Fig. 3b

Fig. 3.—(a) Subject with the apparatus holding the head firmly in the neutral position. (b) Lateral roentgenogram in the neutral position. The extension angulation of each cervical vertebra in relation to the first thoracic vertebral body is indicated by the inked lines.

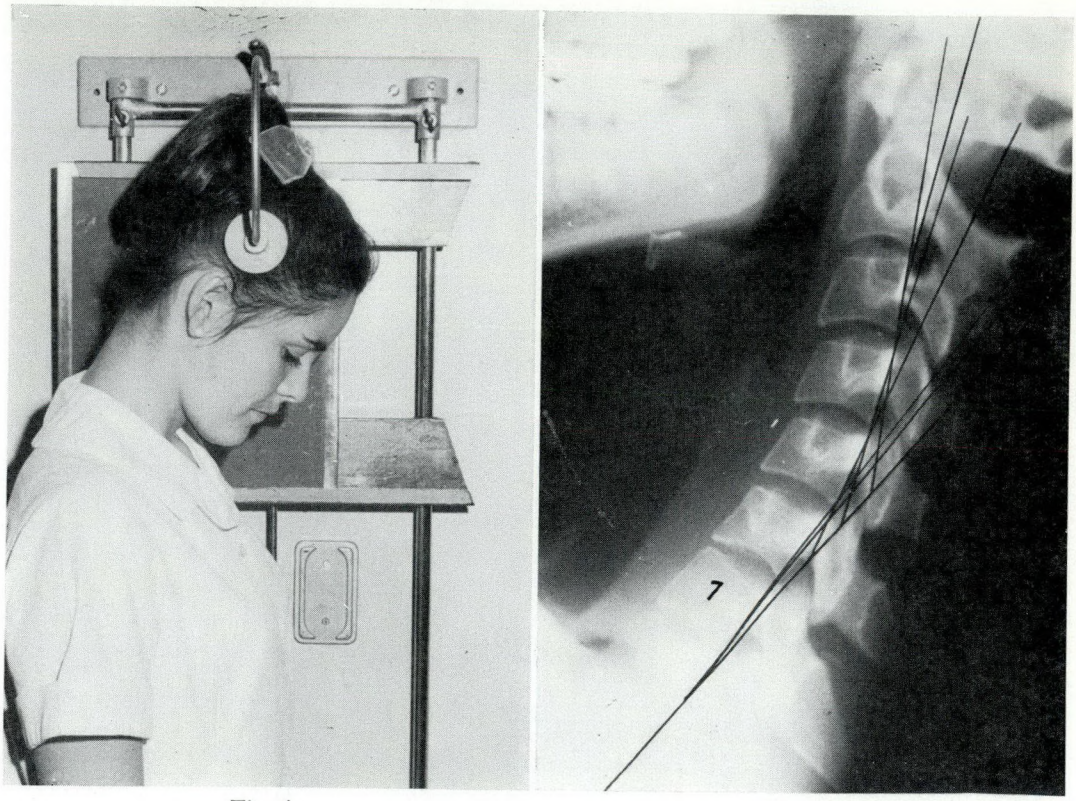

Fig. 4a Fig. 4b

Fig. 4.—(a) Subject with the head in flexion. (b) Lateral roentgenogram of the same subject in flexion.

have endeavoured to do in normals and we then corroborated this by the same study in specimens that covered all age groups. The results of the two studies paralleled.

Clinical Study

In the usual roentgenograms of the cervical spine in flexion and extension, there is often an element of rotation of the vertebral bodies which makes it difficult to measure the correct degree of movement. Also, due to variation in distance of the cervical spine from the radiographic plate, there is a variable magnification factor in each film.

To eliminate significant rotation of the head during flexion and extension of the cervical spine, and to maintain a constant distance between the spine and the radiographic plate, we designed a simple apparatus, which was attached to the radiographic film stand and was easily adjusted around the head. This apparatus (Figs. 1 and 2) consisted of two cups: the outer one was fixed 16 inches from the radiographic plate—a distance found to give the best roentgenograms in this study; the inner cup was adjusted according to the size and position of the subject's head. The subjects were volunteers, student-nurses between 18 and 23 years of age who had no obvious neck abnormalities or history of previous trauma. In each subject, three roentgenograms were obtained in the neutral, flexion, and extension positions of the cervical spine (Figs. 3-5). By keeping the distance from the head to the radiographic plate constant and the focal length fixed, the magnification factor was largely eliminated. Also, the rotation in the three views was minimal because the head was fixed by the two cups. Our anatomical definition of the neutral position was that in which a straight line connecting the mastoid process and the second thoracic vertebral body passed through the lumbosacral junction.

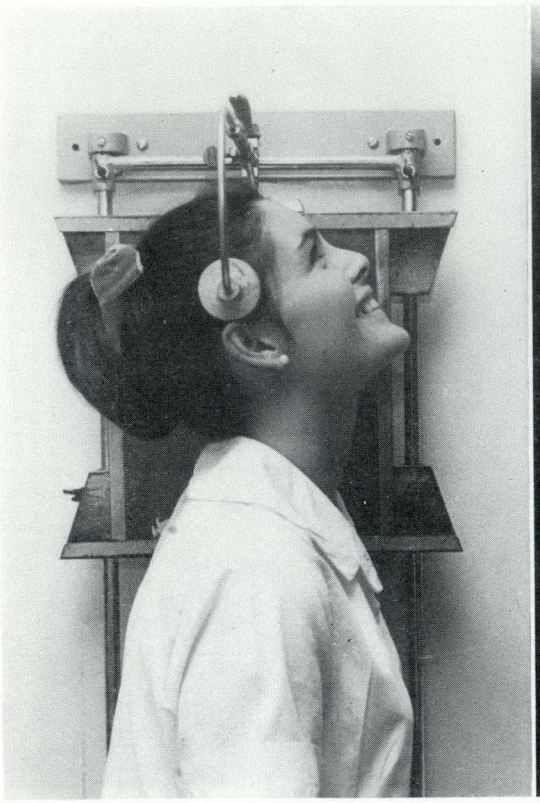

Fig. 5a

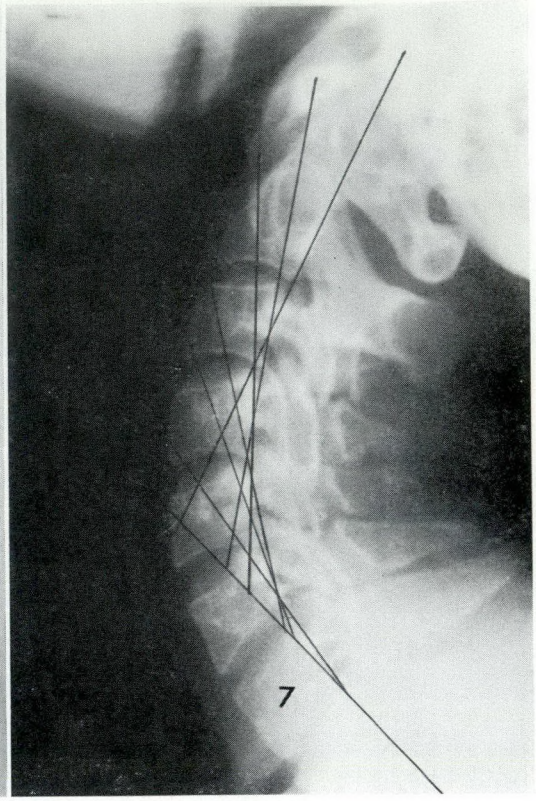

Fig. 5b

Fig. 5.—(a) Subject with the head in extension. (b) Lateral roentgenogram of the subject in extension.

Movement at individual levels was measured on the three roentgenograms of each subject. To determine the range of motion in any part of the body, one must know the "plane" or "axis" of motion—the "line of zero velocity". This was studied in dissected specimens, and was found to pass through the origins of the various nerve roots. When this finding was applied to the roentgenograms of the 20 "normal" subjects, it was found that, for practical purposes, the line of zero velocity passed along the posterior margins of the vertebral bodies.

First, the average angulation at various levels was measured in the neutral posi-

TABLE I.—Average Range of Angulation at Various Levels in the Cervical Spine (C2-T1) in 20 Normals (in Degrees)

	Level	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*	*17*	*18*	*19*	*20*
C2-3	Neutral	13	12	11	12	12	11	11	12	13	12	13	11	13	13	14	14	13	14	12	13
	Flexion	6	6	5	5	6	5	5	6	6	6	6	5	5	5	7	8	6	7	6	6
	Extension	15	14	15	15	15	14	13	14	15	14	15	14	15	15	16	15	14	15	14	15
C3-4	Neutral	5	7	6	5	5	7	7	7	5	7	5	7	5	7	6	6	5	6	7	5
	Flexion	4	5	4	5	4	5	5	5	4	5	4	5	5	5	7	8	4	5	5	4
	Extension	9	12	9	10	9	11	12	11	9	12	9	11	10	12	10	9	10	11	11	10
C4-5	Neutral	4	5	5	4	4	5	5	5	4	5	4	5	4	5	6	4	4	6	5	4
	Flexion	9	10	9	10	9	10	10	9	9	10	9	10	10	10	10	8	9	11	9	9
	Extension	13	14	13	14	13	13	13	14	13	14	13	13	14	13	14	11	13	15	14	13
C5-6	Neutral	4	5	4	5	4	4	4	4	4	5	4	4	5	4	6	5	4	4	4	6
	Flexion	11	12	11	11	11	11	12	11	11	12	11	11	11	12	14	11	11	12	11	13
	Extension	8	8	8	7	8	7	8	7	8	8	8	7	7	8	10	9	8	7	8	9
C6-7	Neutral	6	6	5	6	6	6	5	5	6	6	6	6	6	5	4	5	6	4	5	4
	Flexion	3	4	3	3	4	4	4	4	3	4	3	4	3	4	3	3	3	3	4	2
	Extension	15	16	15	16	15	15	16	15	15	16	15	15	16	16	14	15	15	14	15	13
C7-T1	Neutral	3	3	4	3	4	3	4	3	3	3	3	3	3	4	2	4	4	2	3	3
	Flexion	3	0	0	1	1	0	2	1	0	0	1	2	1	0	2	0	3	2	1	2
	Extension	9	8	10	9	8	10	9	10	9	8	9	10	9	9	10	9	10	9	9	10

TABLE II.—Range of Movement at Various Levels in the Cervical Spine (C2-T1) in Flexion and Extension in 20 Normals (in Degrees)

Level	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*	*17*	*18*	*19*	*20*	*Average*
C2-3																					
Flexion	7	6	6	8	6	6	6	6	7	5	7	6	8	8	7	6	7	7	6	7	7
Extension	2	2	4	2	3	3	2	2	2	2	2	3	2	2	2	1	1	1	2	2	2
C3-4																					
Flexion	9	12	10	10	9	12	12	12	9	12	9	12	10	12	13	14	9	11	12	9	10
Extension	4	5	3	5	4	4	5	5	4	5	4	4	5	5	4	3	5	5	4	5	4
C4-5																					
Flexion	13	15	14	14	13	15	15	14	13	15	13	15	14	15	16	12	13	17	14	13	13
Extension	9	9	8	10	9	8	8	9	9	9	9	8	10	8	8	7	9	9	9	9	9
C5-6																					
Flexion	15	17	15	16	15	15	16	15	15	17	15	15	16	16	20	16	15	16	15	19	15
Extension	4	3	4	2	4	3	4	3	4	3	4	3	2	4	4	4	4	3	4	3	3
C6-7																					
Flexion	9	10	8	9	10	10	9	9	9	10	9	10	9	9	7	8	9	7	9	6	9
Extension	9	10	10	10	9	9	9	10	9	10	9	9	10	9	10	10	9	10	10	9	10
C7-T1																					
Flexion	6	3	4	4	5	3	6	4	3	3	4	5	4	4	4	4	7	4	4	5	4
Extension	6	5	6	6	4	6	5	7	6	5	6	7	6	5	8	5	6	7	6	7	6

tion. As shown in Figs. 3b, 4b and 5b, intersecting lines were drawn along the posterior borders of the various vertebrae from T1 upwards, and the angulation at each level was calculated, relative to the posterior margin of the vertebral body of T1. T1 was selected as the first stable vertebra.

Measurements were carried out between C2 and T1 because the motion at the two levels above C2 is well documented.[1, 5] The average angulation at each level is shown in Table I.

To determine the range of motion, the angle at each level was measured on the roentgenograms in flexion and extension. The total range of movement from the fully flexed to fully extended positions in these 20 normal subjects was calculated (Tables II and III). The average range of flexion and extension for each level is shown in Fig. 6. The same figures can be obtained in consecutive trials on the same patient. This was demonstrated several times during the study when more than one set of radiographs was done during the course of our evaluation and the development of the technique.

Experimental Study

In addition, studies were carried out on dissected specimens of the cervical spine from which the musculature had been removed. Each specimen was prepared by inserting radiopaque metallic pins both anteriorly and posteriorly to record the range of motion, and the specimen was then

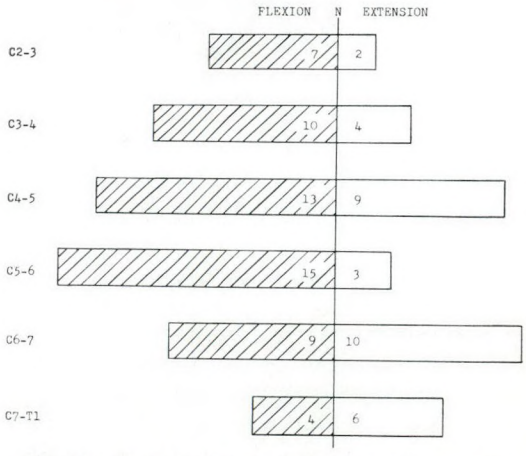

Fig. 6.—Average range of flexion and extension (in degrees) at each vertebral level in 20 normal adults.

TABLE III.—Average Range of Total Movement From Flexion to Extension Position at Various Levels in the Cervical Spine (C2-T1) in 20 Normals (in Degrees)

Level	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*	*17*	*18*	*19*	*20*	*Average*
C2-3	9	8	10	10	9	9	9	8	9	8	9	9	10	10	9	7	8	8	8	9	9
C3-4	13	17	13	15	13	16	17	17	13	17	13	16	15	17	17	17	14	16	16	14	14
C4-5	22	24	22	24	22	23	23	23	22	24	22	23	24	23	24	19	22	26	24	22	22
C5-6	19	20	19	18	19	18	20	18	19	20	19	18	18	20	24	20	19	19	19	19	18
C6-7	18	20	18	19	19	19	18	19	18	20	18	19	19	18	17	18	18	17	19	15	19
C7-T1	12	8	10	10	9	9	11	11	9	8	10	12	10	9	12	9	13	11	10	10	10
Total	93	87	82	86	91	84	98	86	80	97	91	97	96	97	103	90	94	97	96	89	92

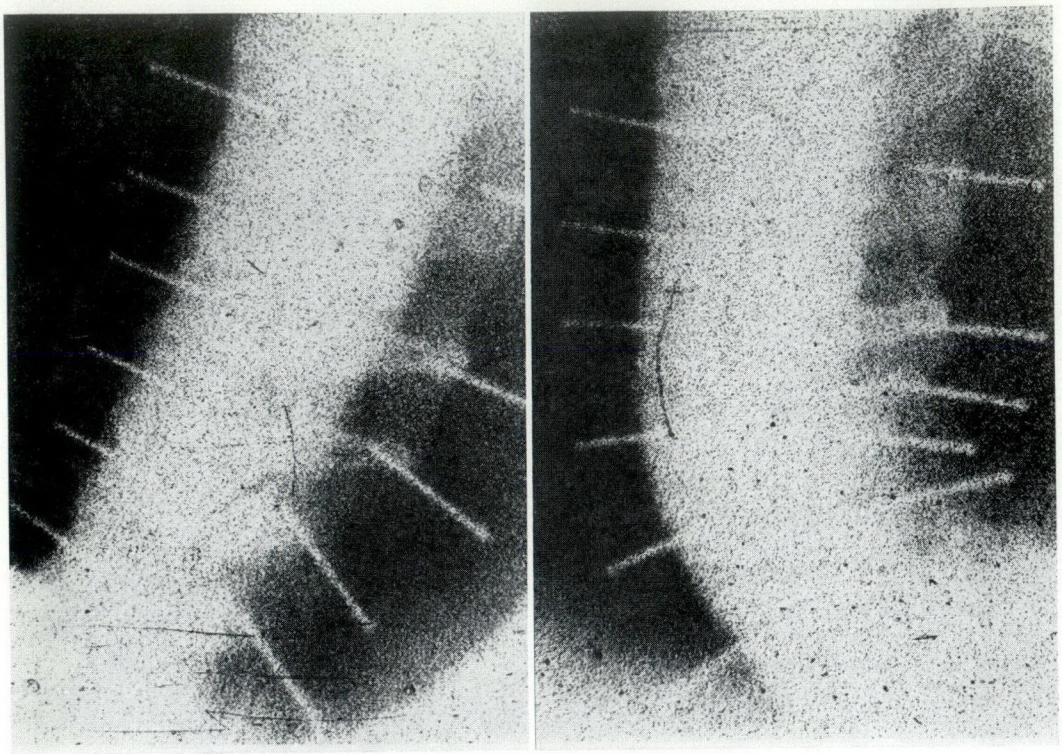

Fig. 7a Fig. 7b

Fig. 7.—(a) Enlarged cineradiographic exposure of a dissected cadaver specimen mounted in the stress machine in flexion. Metallic radiopaque pins have been fixed into each vertebra anteriorly and posteriorly, to record the range of motion. (b) The same specimen in extension.

mounted in a stress machine. The ranges of flexion and extension were recorded cineradiographically. Every fifth frame of the film was enlarged and printed, and the range of movement studied in the fully flexed and extended positions (Fig. 7). The ranges of motion in the fresh cadaver specimens corresponded consistently to the ranges of motion recorded radiographically in the 20 living normal individuals.

Results

It was found that there are only two levels, C6-7 and C7-T1, where there is greater movement during extension than during flexion from the neutral position. At all other levels, C2-3, C3-4, C4-5 and C5-6, the movement is greater during flexion. At C7-T1, the movement in flexion was negligible while there was a fair range of motion during extension. Also, it was observed that the maximum range of total movement occurred at the C4-5 level. Analysis of motion at individual levels shows that, at the C5-6 level, the average range of flexion is 15° and of extension, 3°. In clinical practice the C5-6 level is that most commonly fused.[7-9] This may be related to the fact that, in most extension and extension-flexion injuries, the C5-6 level may be vulnerable to injury because its range of extension is limited. One might postulate that, if the initial trauma forces the neck into flexion, injury will occur at a level where there is less motion in flexion (greater motion in extension), that is C6-7 and C7-T1. On the other hand, if the initial trauma extends the neck, injury will occur at a cervical level where there is less motion in extension (greater motion in flexion), that is at C2-3, C3-4, and C5-6.

Discussion

It appears that the criterion used by some authorities to select the level for fusion—the site of maximum motion—may be misleading. If this criterion has value it would appear important to determine

not only the level at which maximum motion occurs, but to compare this with the motion noted in normals. In extension injuries of the cervical spine, it appears that the C2-3, C3-4 and C5-6 levels are most vulnerable because from the neutral position, their range of extension is limited. Conversely, in flexion injuries of the cervical spine, the C7-T1 level should be the most vulnerable because its range of flexion from the neutral position is limited. We do not suggest that the range of motion should be taken as an indication for surgery in older people. One of the purposes of this paper is to point out that this is a dangerous fallacy. In our opinion, clinical assessment supported by discography, performed with the patient awake, is still the most reliable method of identifying the level or levels of the cervical spine that are producing symptoms.

Summary

The normal range of motion of the cervical spine at each intervertebral level between the second cervical and first thoracic vertebrae has been calculated in 20 normal individuals.

A simple radiographic technique was used to determine the range of motion at each level. A simple apparatus was specially devised to control rotation and magnification.

The average range of flexion and extension was determined from the neutral position at each level.

The clinical significance of these findings is discussed in relation to cervical spine injuries, with respect to the recognition of abnormal areas of mobility.

The authors would like to acknowledge the invaluable assistance given by Mr. Henry Garside of the St. Joseph's Hospital Research Foundation, Toronto, in the mechanical studies described in this paper.

References

1. Ferlic, D.: The range of motion of the "normal" cervical spine, *Bull. Hopkins Hosp.*, **110:** 59, 1962.
2. Jackson, R.: The cervical syndrome, 3rd ed., Charles C Thomas, Publisher, Springfield, Ill., 1966.
3. Hohl, M.: Normal motions in the upper portion of the cervical spine, *J. Bone Joint Surg.* [*Amer.*], **46A:** 1777, 1964.
4. Fielding, J. W.: Cineroentgenography of the normal cervical spine, *J. Bone Joint Surg.* [*Amer.*], **39A:** 1280, 1957.
5. Fielding, J. W.: Normal and selected abnormal motion of the cervical spine from the second cervical vertebra to the seventh cervical vertebra based on cineroentgenography, *J. Bone Joint Surg.* [*Amer.*], **46A:** 1779, 1964.
6. Buonocore, E., Hartman, J. T. and Nelson, C. L.: Cineradiograms of cervical spine in diagnosis of soft-tissue injuries, *J. A. M. A.*, **198:** 140, 1966.
7. Robinson, R. A. *et al.*: The results of anterior interbody fusion of the cervical spine, *J. Bone Joint Surg.* [*Amer.*], **44A:** 1569, 1962.
8. Cloward, R. B.: New method of diagnosis and treatment of cervical disc disease, *Clin. Neurosurg.*, **8:** 93, 1962.
9. Simmons, E. H. and Bhalla, S. K.: Anterior cervical discectomy and fusion with eight year follow-up—a clinical and biomechanical study, *J. Bone Joint Surg.* [*Brit.*], In press.

Résumé

Les auteurs ont calculé l'échelle de la mobilité normale de chaque articulation inter-vertébrale à partir de la seconde vertèbre cervicale jusqu'à la première vertèbre thoracique; les mesures ont été faites sur 20 individus normaux. La mobilité de chaque articulation a été mesurée par une technique radiographique simple, un appareil d'emploi facile, inventé par les auteurs, contrôlant la rotation et le grossissement.

L'échelle moyenne de la flexion et de l'extension à partir de la position neutre a été déterminée à chaque niveau. Les auteurs discutent la signification clinique de leurs découvertes par rapport aux lésions des vertèbres cervicales; elles aideront à identifier les régions à mobilité anormale.

MYOELECTRIC STUDIES ON THE TIBIALIS ANTERIOR MUSCLE IN BELOW-KNEE AMPUTEES*

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ALTHOUGH myoelectric control systems have an assured role in prosthetics, several problems remain unsolved. One important problem that cannot be solved by engineering skill or technical refinement is the availability of ideal control sites. A myoelectric control system requires one or more muscles, which are subject to good voluntary control but are not required for frequent bodily movements. For cosmetic reasons this muscle should be relatively inconspicuous. Below-elbow amputees possess many suitable muscles for myoelectric control and the natural activity of the flexors and extensors of the forearm is to produce the motions required in the prosthetic hand. These factors account for the success of "the Russian hand". Above-elbow amputees, however, have lost not only the elbow joint, whose function must be replaced, but also the good myoelectric control sites afforded by the forearm muscles.

In above-elbow amputees the most suitable muscles for myoelectric control purposes are the biceps and triceps brachii. The disadvantage of these muscles is that the biceps and long head of the triceps have scapular attachments and are activated during movements of the shoulder joint. The shoulder musculature, when it is called into action, causes unwanted operation of the controlled devices (powered hook, wrist or elbow). This is the major reason why myoelectric control systems have not been used extensively or efficiently on high-level upper-extremity amputees.

R. N. Scott of the Bio-Engineering Institute, University of New Brunswick, consulted us about the problem. As a solution, we suggested that a muscle or part of a muscle be isolated to create "neutral" or "non-articular" muscles in the stump. With training, these "surgically neutralized" muscles might then remain inactive except when, on command, they would operate the controlled devices. Would such a muscle still retain good voluntary control? Would atrophy be a serious problem? Would the muscle still contract when the joint is moved?

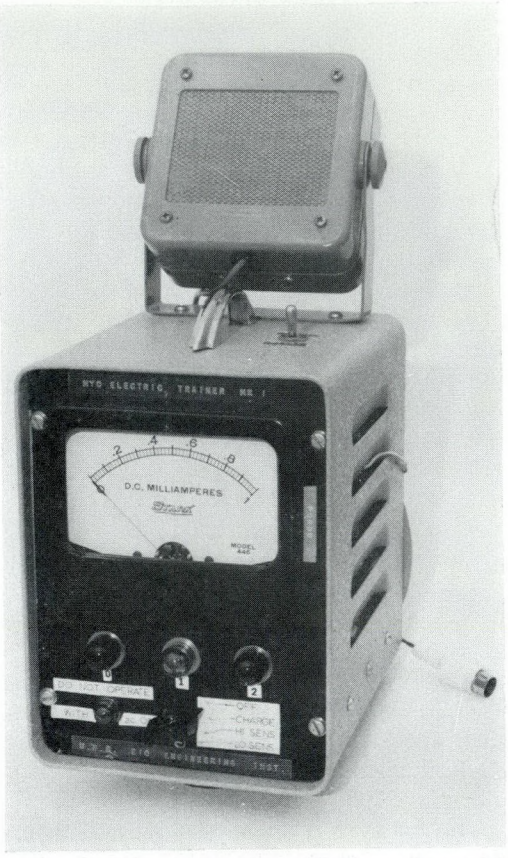

Fig. 1.—The University of New Brunswick Myoelectric Trainer.

EXPERIMENTAL PROCEDURE

As a preliminary, we decided to investigate the activity of the anterior tibial muscle group in below-knee (shank) amputees. These muscles are surgically neutralized and, in addition, function phasically,

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automatically and synchronously with other limb muscles.

The studies were carried out on 6 normal subjects and 20 below-knee amputees.

We evaluated the performance of the pre-tibial muscles as myoelectric control sites using the University of New Brunswick "Myoelectric Trainer Mark I" (Fig. 1)—a device which indicates two levels of muscle activity by turning on coloured lights. We modified it to produce audible tones as well, so that the subject did not have to watch the lights during training and testing. The myoelectric behaviour of the muscles under study was recorded using a single-channel electromyograph which consisted of a preamplifier, oscilloscope and camera (Fig. 2). Beckman surface electrodes, applied with Beckman

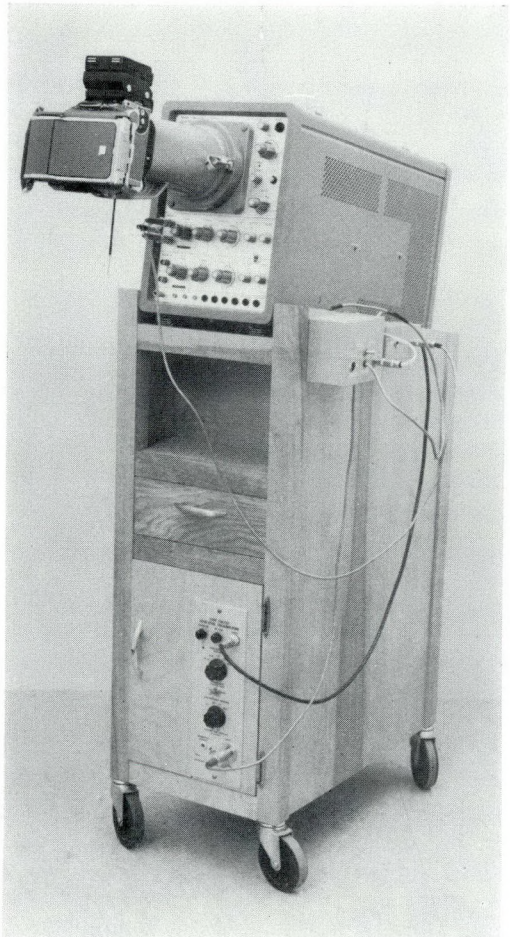

Fig. 2.—Apparatus for taking electromyograms. The preamplifier is in the base of the cart.

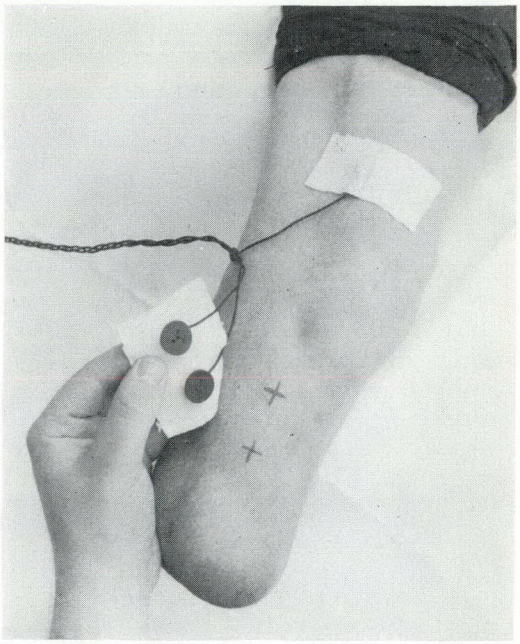

Fig. 3.—Placement of electrodes on a below-knee stump.

electrode jelly, were spaced one inch apart over the belly of tibialis anterior and held in place with an Elastoplast bandage (Fig. 3). To allow the subject to walk about freely, the myoelectric signal was conducted from the subject to the trainer or electromyograph by a cable riding on an overhead trolley. The surface electrodes recorded predominantly the activity of the tibialis anterior but some small potentials from the other pre-tibial muscles were also detected.

In the test procedure, the electrodes were applied and the subject put on his prosthesis in the usual fashion. He then received five minutes of instruction on the use of the myoelectric trainer. After this, the subject was asked to turn on the first threshold (amber light or low-pitched tone) for a count of five; relax and then maintain the second threshold (red light or high-pitched tone) for a count of three. If the test subject could achieve this level of performance, he was rated "good". If the subject could perform this test, but not hold the lights or tones steady, the performance was rated "fair". If the subject was unable to achieve this degree of myoelectric control, he was rated "poor".

In the second part of the test we re-

corded the myoelectric activity of the tibialis anterior during level walking, using the oscilloscope and camera. We also observed whether muscle activity was recorded in turning, mounting a step, holding the prosthesis off the floor or regaining balance after the subject had been given a slight backward shove.

Results

The performance of all normal subjects on the myoelectric trainer was good. They exhibited phasic activity of the tibialis anterior similar to the typical electromyogram (Fig. 4). All showed a burst of activity

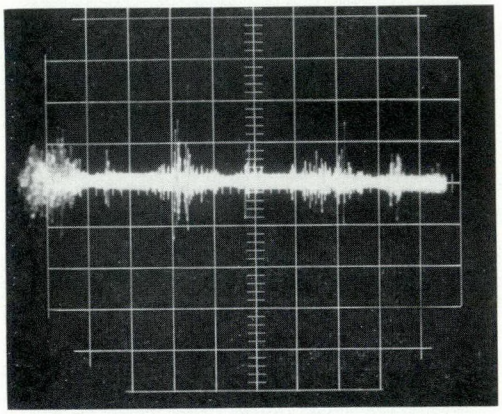

Fig. 4.—Electromyogram of the tibialis anterior from a normal subject during level walking. Note the phasic activity (time scale—0.2 seconds per division).

when unbalanced by a slight backward push on the shoulders.

Seventy per cent of amputees displayed good myoelectric control. Only two amputees (10%) performed poorly; one was a hemiplegic and the other a psychiatric patient. The performance of voluntary control is recorded in Table I.

In 40% of amputees, the muscle was phasically inactive (Fig. 5), exhibiting small bursts of activity only on turning or holding the prosthesis off the floor. In 60% of the amputees, the muscle was phasically active at every step. A typical electromyogram (Fig. 6) is similar to the electromyogram from a normal subject. In almost every amputee who exhibited phasic activity of the tibialis anterior during walking, we could elicit a burst of activity from the muscle by pushing backwards on the subject's shoulders.

TABLE I.—Myoelectric Control Performance in 20 Below-Knee Amputees

Time since amputation (years)	*Number of amputees*	*Good*	*Fair*	*Poor*
Less than 1	5	3	1	1
1 - 2	6	4	2	—
2 - 3	5	3	1	1
More than 3	4	4	—	—
Total	20	14	4	2

Age spread 17 to 84 years; median 50 years.

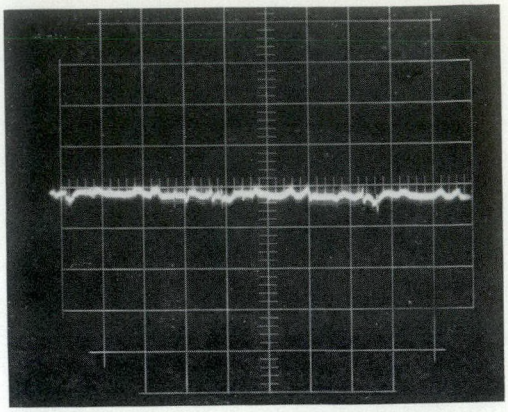

Fig. 5.—An electromyogram from a below-knee amputee shows no activity during walking (bumps and ripples are due to changing pressure on the electrodes inside the socket).

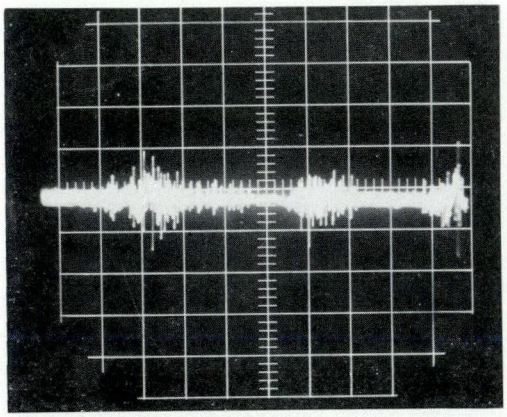

Fig. 6.—Electromyogram from a below-knee amputee displaying phasic activity during level walking.

The important factor governing the behaviour of the pre-tibial stump muscles in below-knee amputees is the length of time since amputation. The data in Table II indicate that, with increasing time following amputation, there is a definite trend towards phasic inactivity during level walking. Our observations indicate that a

TABLE II.—Phasic Activity of the Tibialis Anterior During Walking in 20 Below-Knee Amputees

Time since amputation (years)	*Number of amputees*	*Active*	*Quiet*
Less than 1	5	4	1
1 - 2	6	4	2
2 - 3	5	3	2
More than 3	4	1	3
Total	20	12	8

decline in the phasic activity of the tibialis anterior muscle occurs from one to three years after amputation.

It is especially interesting to note that two amputees, 7 and 35 years after amputation, still had good voluntary control and no phasic activity of their pre-tibial muscles.

Discussion

Ideal muscles for myoelectric control sites must retain good voluntary control. In addition, they may perform a function identical or similar to that carried out in the preamputation state, or may be so altered by operation that they no longer have any mechanical function. The Russian myoelectrically controlled prosthesis uses flexor muscles of the forearm to flex the fingers and the extensors to extend the prosthetic digits. The cerebral cortex, peripheral nerves and muscle fibres function essentially as before the amputation. The loss of tendon and joints prevents the muscles from producing motion. The electrical potentials released from the contracting muscle fibres, however, can be collected, magnified and used to operate the electrically driven motor, which produces the required motion in the prosthesis. The muscle performs the same motion; its electrical reactions are now harnessed electronically to do work that was previously mechanical. With respect to amputations proximal to the elbow joint, the superficial stump muscles have their own natural functions to perform and training does not eliminate these functions.[1]

Other myoelectric control sites have been considered, such as the facial, temporal, neck and sphincter muscles, but none of these seemed cosmetically, physiologically or socially desirable. Our attention was then directed to the possibility of modifying certain proximal stump muscles so they would not pass over a joint and be fixed proximally and distally to the adjacent bone. Obviously the neurovascular supply to the muscle belly must be left inviolate. Biceps, triceps, coracobrachialis and the scapular muscles are all possible candidates for this surgical neutralization.

Our present findings concerning tibialis-anterior function in patients with below-knee amputations support the hypothesis that surgically neutralized muscles can be used to control myoelectric systems. With minimal training, the muscles come under good voluntary control. We propose to study the effects of more intensive training to see if voluntary control can be further improved. Discouraging, at first sight, is the time required for the muscle to lose its phasic activity. We do not know if phasic activity is a characteristic of the muscles in the upper limb. We think that the development of phasic inactivity in the lower limbs can be accelerated by training and we propose to study this matter in upper and lower extremity muscles in the future.

We have used skin electrodes because they are simple and because they reveal adequately the electrical activity of the subjacent muscle. We realize that percutaneous electrodes are more precise and that, if motor units are used to activate many of these systems, percutaneous or implant electrode transmitters will be required.

Basmajian, Baeza and Fabrigar[2] have demonstrated that patients can be trained to fire off independently up to 11 single motor units in a small muscle-like abductor pollicis brevis. The ability of patients to control single motor units greatly enhances the potential value of surgically neutralized muscles. Basmajian states that any skeletal muscle may be selected and that most people can isolate and master one or two units and some can master three, four, six or more units. Therefore, if muscles can be divorced from their natural function by operation or other means and the motor unit activity trained, a single muscle may provide many control sites. Theoretically, two or more functions can be controlled in the prosthesis or orthosis, the number being commensurate with the patient's ability to master control of many motor units.

SUMMARY AND CONCLUSION

In a preliminary study to determine the feasibility of surgically prepared myoelectric control sites, we tested the tibialis anterior in 20 below-knee amputees and 6 normal subjects. This muscle was chosen because it is superficial, and also in below-knee amputees it is a surgically neutralized (non-articular) muscle. Myoelectric control was assessed while the subject performed a simple exercise with a myoelectric trainer. The activity of the muscle during walking was recorded on an electromyograph.

A surgically neutralized muscle retains good voluntary control and generates a signal sufficient to operate a myoelectric control system. For an indefinite period after operation the muscle continues to contract during the activity for which it formerly was required. After one to three years, the tibialis anterior muscle of many below-knee amputees remains inactive except on command.

REFERENCES

1. SCOTT, R. N.: Personal communication.
2. BASMAJIAN, J. V., BAEZA, M. AND FABRIGAR, C.: Conscious control and training of individual spinal motor neurons in normal human subjects, *J. New Drugs*, **5:** 78, 1965.

RÉSUMÉ

Dans une étude préliminaire visant à établir la possibilité de préparer chirurgicalement des régions de contrôle myoélectriques, nous avons étudié le jambier antérieur de 20 sujets amputés en-dessous du genou et de 6 sujets normaux. Ce muscle a été choisi parce qu'il est superficiel et aussi parce qu'il est déjà "neutralisé" chirurgicalement (n'est plus articulé) chez les amputés en-dessous du genou. Le contrôle myoélectrique a été essayé en demandant au sujet de faire un exercice très simple avec un entraîneur myoélectrique. L'activité du muscle durant la marche a été enregistrée sur un électromyographe.

Un muscle neutralisé chirurgicalement garde un bon contrôle volontaire et il produit un signal assez fort pour faire fonctionner un système de contrôle myoélectrique. Pour un temps indéfini après l'opération, le muscle continue à se contracter chaque fois que l'activité musculaire aurait réclamé sa collaboration avant l'intervention. Après une période allant de un à trois ans, le jambier antérieur d'un grand nombre d'amputés en-dessous du genou devient inactif, sauf sur commande.

PREVENTION OF THE HEMORRHAGIC ENTERITIS PRODUCED BY IRREVERSIBLE HYPOVOLEMIC SHOCK

It has been shown by Bounous *et al.*, that the exclusion of the pancreatic exocrine secretion from the intestinal lumen prevents the occurrence of the hemorrhagic enteritis that in dogs is associated with experimental hemorrhagic shock. As a continuation of this work, the authors investigated two series of dogs. In a first group of six dogs, a segment of ileum 40 to 60 cm. in length was isolated and the two ends were brought out through the abdominal wall. The continuity of the bowel was re-established and a hemorrhagic shock procedure was carried out two to three weeks afterwards. The response of the animals to the experimental procedures was identical to that of untreated animals. At post mortem it was found that the hemorrhagic enteritis was only present in that part of the intestinal tract which was left in continuity but not in the isolated loop of ileum. In a second series consisting of seven dogs, a subtotal duodenal pancreatectomy was carried out. The tail of the pancreas was left behind but not anastomosed to the bowel. The common bile duct was ligated and the gallbladder was anastomosed to the stomach. During the postoperative period, the animals' diet was supplemented with pancreatic enzymes, and after three to four weeks the dogs were subjected to the same experimental shock procedure. In this group the survival following the experiment was prolonged as compared with a control group and at post mortem no evidence of hemorrhagic enteritis was found. Thus, the observation made by Bounous *et al.* has been confirmed.—Tiberio, G. *et al.*: Sur la prévention de l'entérite nécrotico-hémorragique provoquée par le choc hypovolhémique irréversible expérimental, *Lyon Chir.*, **64:** 605, 1968.

DILUTED ACD BLOOD FOR CARDIOPULMONARY BYPASS IN CHILDREN*

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THE use of diluted donor blood to prime a pump oxygenator is now generally accepted.[1] In addition, the difficulty of collecting heparinized blood, which must be fresh, has led many surgical groups[2-4] to accept citrated blood up to four days old[5, 6] and to heparinize it shortly before operation. Having adopted these two modifications of our earlier practice (that of insisting on fresh heparinized blood) we have made hematological and biochemical observations on the effect of such hemodilution on the blood of children. Since no previous publication has confined itself to children, we believe the results reported herein may be of interest.

THE PRIME AND THE PATIENTS

Acid-citrate-dextrose (ACD) blood was drawn into plastic bags within 48 hours of operation and each unit was modified so that the final volumes were as follows:

Constituent	*Volume (ml.)*
Blood	450
ACD solution (National Institutes of Health Formula A)	67.5
Heparin 1000 units/ml.	2.5
10% $CaCl_2$	5
$NaHCO_3$ 0.9 mEq./ml.	12
5% Glucose in 0.2% NaCl	110 or 150
Total	647 or 687 ml.

The smaller volume of diluent (110 ml.) was used with the 10-screen oxygenator (2280 ml. prime) when the predicted flow was under 2000 ml./min. The larger volume of diluent (150 ml.) was used with the 20-screen oxygenator (2750 ml. prime). Heparin, calcium chloride and sodium bicarbonate were added to each unit of ACD blood in the blood bank and the glucose in 0.2% sodium chloride was added to the pump oxygenator. Samples of the prime were taken for analysis after at least 10 minutes of recirculation.

The first 12 patients who underwent bypass using this prime were extensively studied. These children ranged in age from 1 to 13 years and in weight from 8.6 to 42.4 kg. In eight, the bypass was done using an oxygenator containing 10 screens (110 ml. of 5% glucose in 0.2% NaCl added to each unit of blood); in the four larger children, the oxygenator contained 20 screens (150 ml. of 5% glucose in 0.2% NaCl to each unit of blood). In these children the bypasses, which lasted from 17 to 110 minutes (average 56 minutes), were done to correct the following lesions: aortic stenosis—two patients, atrial septal defect—one, mitral valve repair—one, pulmonary stenosis—one, tetralogy of Fallot—five, and ventricular septal defect—two patients. Eleven of the 12 children survived; the one death was clearly not related to the priming solution. Subsequently, another 45 patients were evaluated, although with less extensive laboratory investigations; the findings in these patients corresponded with those in the 12 who provided the data for this report. In none of these did we encounter hemorrhage or tamponade that required reoperation.

OBSERVATIONS

Biochemistry

The superiority of 5% glucose in 0.2% NaCl to 5% glucose in water, which has been recognized before,[7] was confirmed by dividing a bag of ACD blood and adding one of these diluents to each half. The bags were allowed to stand at room temperature for two hours and then were stored overnight at 4°C. The plasma and red cell morphology of specimens from each bag were then determined. A typical result is shown in Fig. 1.

The prime was recirculated through the pump oxygenator for 10 minutes and a sample was drawn for biochemical determinations (Table I). Further samples were

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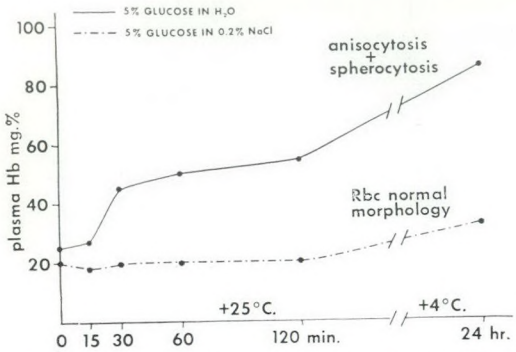

Fig. 1.—Plasma hemoglobin levels in blood stored in 5% glucose in water, and in 5% glucose in 0.2% NaCl. The hemolysis is three times as great with the first solution. The dilution in each case was identical to that in the pump oxygenator.

drawn 10 minutes after starting bypass, and 1 hour and 24 hours after operation. These values are compared with the patients' preoperative values in Table II. The findings can be summarized as follows: (1) sodium levels remained constant, within the normal range. (2) Serum potassium tended to be low during bypass, but had resumed almost normal levels by the end of the first postoperative hour. Digitalis toxicity due to hypokalemia would therefore not present a hazard, although few of our patients are digitalized at the time of operation. (3)

TABLE I.—Priming Solution in the Pump Oxygenator (Mean for 12 Patients)

Constituents		*Hospital normals*
Hb. (g. %)	8.6	—
pH (at 37°C.)	7.30	7.38–7.43
Pco_2	36	36–45
Sodium (mEq./l.)	145	137–144
Potassium (mEq./l.)	4.0	4.7–5.5
Chloride (mEq./l.)	95	95–110
Calcium (mg./100 ml.)	31.5*	9–11
Sugar (mg./100 ml.)	1200	—
Osmolality (mOsm./l.)	324	285–295

* Total calcium.

Serum calcium levels were very high during bypass, but fell to normal one day after operation, except in one patient in whom the level was 7.7 mg./100 ml. 24 hours after operation. The transitory high values are due to the $CaCl_2$ which is added to the prime to bind the citrate—the resultant calcium complex is therefore not ionized. (4) The bypass and one-hour postoperative glucose levels were high, due to the high glucose concentration of the priming solution, but levels returned to normal in the succeeding 24 hours. (5) Blood gases and acid-base determinations were made frequently. The results were similar to those previously found in fresh blood. Abnormalities were related to low perfusion rates or hyperventilation and could not be attributed to the priming solution.

TABLE II.—Open-Heart Operations With ACD Priming (Average of 12 Patients)

Test	*Before operation*	*Patient on bypass*	*1 Hour after operation*	*24 Hours after operation*
Hb. (g.%)	13.5 (8.3-20.1)	9.9 (8.3-11.9)	13.3 (11.7-15.3)	12.6 (9.6-13.7)
Platelets (in 1000)	230 (164-356)	—	78 (42-144)	89 (35-117)
Prothrombin time (sec.)	14 (12-21)	—	15 (14-18)	—
Sodium (mEq./l.)	139 (132-143)	139 (130-150)	142 (134-154)	139 (133-147)
Potassium (mEq./l.)	4.4 (3.9-5.2)	3.6 (2.3-5.3)	4.2 (3.1-6.0)	4.1 (3.7-6.4)
Chloride (mEq./l.)	103 (100-106)	99 (97-104)	104 (99-110)	99 (92-105)
Calcium (mg./100 ml.)	10.1 (9.2-10.7)	18.3 (14.0-24.0)	12.0 (9.6-14.4)	9.5 (7.7-10.2)
Sugar (mg./100 ml.)	105 (68-168)	652 (464-924)	300 (124-480)	130 (68-258)

Hematology

Red blood cell morphology was examined during bypass. The number of crenated cells increased as the duration of the bypass increased, but this was not considered significant (Fig. 2).

A transient fall in hemoglobin concentration was rapidly followed by a return to normal levels. Platelets were markedly reduced up to 24 hours after operation, but to no greater degree than that observed after bypass with heparinized fresh blood.

Prothrombin activity remained normal in all patients. As noted above, we encountered no hemorrhagic complications that made reoperation necessary. This we attribute in large part to the routine prevention of fibrinolysis by the prophylactic administration of epsilon aminocaproic acid.[8]

After these studies were completed, 45 more patients were operated upon using the same diluted modified blood mixture for oxygenator priming but with a slight modification—heparin and calcium chloride were added to the ACD blood in the blood bank the night before operation, and the other

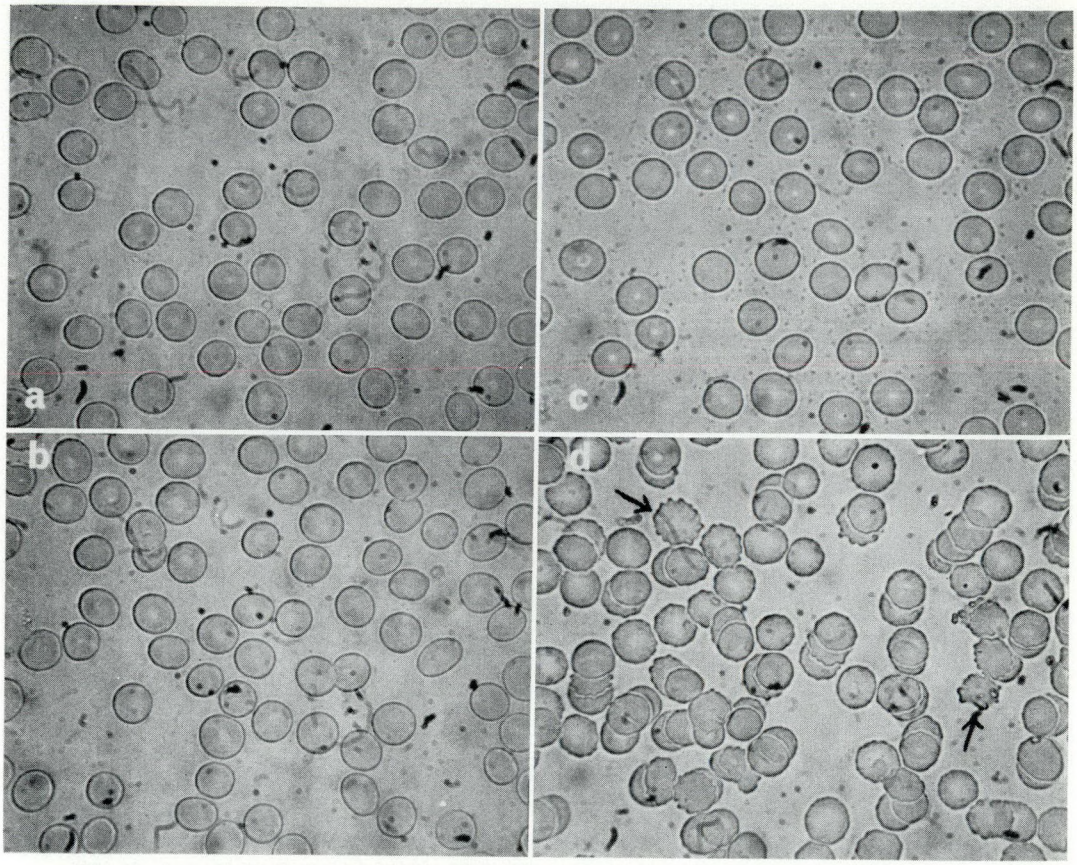

Fig. 2.—(a and b) Red blood cells from the oxygenator immediately before bypass and 17 minutes later. (c and d) Blood from another patient immediately before bypass and after 73 minutes of bypass (x 800).

diluents (5% glucose in 0.2% NaCl, and $NaHCO_3$) were added by the anesthetist directly to the pump just before operation.

Studies of red blood cell size and morphology revealed that 12-hour exposure to heparin/calcium chloride without the alkalinizing effect of the bicarbonate produced no shrinkage, crenation or other recognizable changes.

In this group of 45 patients, the results observed were identical to those of the first 12 patients. None of these 45 patients died.

Conclusion

Using standard ACD bank blood (less than 48 hours old), modified by heparinization, recalcification, alkalinization and dilution with 5% glucose in 0.2% saline, for the priming of the pump oxygenator for cardiac surgery in children, undoubtedly subjects the patients to unusual biochemical and hematological stresses which would not be present if we used fresh, undiluted, heparinized blood. Because diluted ACD blood has great practical advantages, we were reassured to find that the biochemical and hematological changes that result are tolerable and transitory. In no definable way did this diluted blood prejudice the child's ability to tolerate cardiac surgical procedures with pulmonary bypass. It seems apparent that the added sodium bicarbonate (in the amounts used) adequately buffered the fixed acids of the ACD blood, and that the accumulated carbonic acid was effectively eliminated by the pump oxygenator during recirculation.

Summary

In 12 children, diluted, modified, ACD bank blood was used to prime the pump oxygenator for cardiac operations. Biochem-

ical and hematological characteristics of the priming blood and of the patients were measured and showed transitory anemia, thrombocytopenia, hyperglycemia, hypocalcemia and hypokalemia. However, within 24 hours the patients achieved satisfactory compensation.

As a diluent, 5% glucose in 0.2% NaCl is superior to 5% glucose in water, doing less damage to donor erythrocytes. Studies in an additional 45 patients gave similar results.

References

1. Malm, J. R. *et al.*: The use of ACD blood titrated with THAM in extracorporeal circulation, *Trans. Amer. Soc. Artif. Intern. Organs,* **9:** 216, 1963.
2. Foote, A. V., Trede, M. and Maloney, J. V., Jr.: An experimental and clinical study of the use of acid-citrate-dextrose (ACD) blood for extracorporeal circulation, *J. Thorac. Cardiovasc. Surg.,* **42:** 93, 1961.
3. Nahas, G. G. *et al.*: Control of acidosis and the use of titrated ACD blood in open-heart surgery, *Ann. Surg.,* **160:** 1049, 1964.
4. Sessler, A. D. *et al.*: Heparinized blood versus ACD blood for cardiopulmonary bypass, *Transfusion,* **6:** 360, 1966.
5. Britten, A. *et al.*: The use of ACD bank blood and fresh heparinized blood in open-heart surgery: a comparative coagulation study, *Transfusion,* **3:** 368, 1963.
6. Salzman, E. W.: In discussion: Sessler, A. D. *et al.*: Heparinized blood versus ACD blood for cardiopulmonary bypass, *Transfusion,* **6:** 364, 1966.
7. Kirklin, J. W.: In discussion: Sessler, A. D. *et al.*: Heparinized blood versus ACD blood for cardiopulmonary bypass, *Transfusion,* **6:** 365, 1966.
8. Kevy, S. V. *et al.*: The pathogenesis and control of the hemorrhagic defect in open heart surgery, *Surg. Gynec. Obstet.,* **123:** 313, 1966.

Résumé

Du sang citraté dilué a été employé dans des opérations à cœur ouvert chez des enfants de plus d'un an. Le sang avait été héparinisé et le citrate était neutralisé avec du chlorure de calcium la nuit précédant l'intervention. Dans la salle d'opération, le pH était ajusté au moyen de bicarbonate de soude et le sang dilué avec du glucose à 5% dans 0.2% de sérum physiologique. Les auteurs passent en revue les modifications biochimiques et hématologiques relevées au cours de 12 opérations de ce genre. L'emploi de ce sang dilué et été bien toléré en circulation extra-corporelle et aucune complication n'a pu lui être imputée. Les gros avantages pratiques du sang citraté dilué, par comparaison à la cueillette de sang hépariné, ont décidé les auteurs à se servir habituellement du premier.

PULMONARY ARTERY BANDING

The authors report the results of palliative banding of the pulmonary artery in 47 children who had correctable ventricular septal defects, but were either too small or too ill to have corrective surgical treatment. Palliative banding was used as the definitive procedure in a second group of 57 patients who had noncorrectable lesions.

Indications for operation were based upon the presence, in both groups, of a large intracardiac shunt with increased pulmonary flow and complicated by intractable failure that was not responsive to vigorous medical management, of repeated upper respiratory infections, pneumonia, failure to thrive, and excessive limitation of activity.

The operative technique consisted of a left, anterolateral, fourth interspace incision, although a median sternotomy and right anterolateral incision were used for patients with anomalies of the great vessels. The pulmonary artery was wrapped with tape 5 to 7 mm. wide. Initial narrowing was accomplished by clamping the tape with a right-angled clamp, and once the proper degree of tightness was obtained the tapes were fixed in place with interrupted silk sutures. Pressure measurements were taken proximal and distal to the tape when possible.

There were 15 deaths in the palliative group with ventricular septal defects, and 18 deaths in the definitive group. In both groups the patients who survived showed substantial improvement in cardiac status, with a decrease in pulmonary vasculature and heart size, and many have had subsequent total correction. Only 1 of 13 patients with a single ventricle and 2 of 14 patients with transposition died. However 7 of 15 patients with atrioventricular canal and four of six patients with truncus arteriosus died.—Idriss, F. S., Riker, W. L. and Paul, M. H.: Banding of the pulmonary artery; a palliative surgical procedure, *J. Pediat. Surg.,* **3:** 365, 1968.

LEUKEMIC INFILTRATION OF TESTIS

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UNILATERAL enlargement of the testis due to acute leukemia is extremely rare, although microscopic infiltration is frequently observed in postmortem material. In a study of acute leukemia based on 340 necropsies, Thomas and Berard[1] noted that 48% of the males had leukemic infiltration of the testes and 24% of females had infiltration of the ovaries. In a review of 1073 "lymphoblastomas" over a 35-year period (456 lymphosarcomas, 383 Hodgkin's disease and 234 leukemias), Watson, Sauer and Sadugor[2] found only three with testicular involvement. Two had metastatic lymphosarcoma and one, a 64-year-old man, had leukemic infiltration causing bilateral enlargement of the testes. Stegagno, Digilio and Felici[3] described leukemic infiltration of the testes in two 5-year-old boys, one of whom was treated by x-ray therapy with gradual return of both testes to normal size. Zuelzer and Flatz[4] reported unilateral leukemic infiltration of the left testis in a 17-month-old male infant who was treated by orchiectomy. Dameshek and Gunz[5] referred to a 6-year-old boy with leukemic involvement of both testes, who died in renal failure. Donohue[6] has studied a child with acute leukemia and testicular enlargement. His patient is similar to the three boys described in the present paper.

CASE REPORTS

Case 1.—D.H., a 10-year-old boy, was admitted to Chedoke Hospital, Hamilton, Ontario, on August 9, 1962 because he was thought to have subleukemic leukemia. His hemoglobin on admission was 11 g./100 ml., the leukocyte count was 1900/c.mm. with a differential of 83% lymphocytes and 17% neutrophils. The platelet count was 190,000/c.mm. and the sedimentation rate was 54 mm. in one hour (Wintrobe). The bone marrow revealed acute lymphoblastic leukemia. He was treated with prednisone and 6-mercaptopurine until November 1963 and obtained an excellent remission. In July 1964 he was admitted to the South Waterloo Memorial Hospital, Galt, Ontario, because of an enlarged right testicle; it was subsequently removed. The testis, epididymis and spermatic cord weighed 94 g. The testis measured 6 x 4 x 3.5 cm. and the epididymis 4.5 x 2 x 1.5 cm. The cut surfaces were uniformly grey and firm with a fish-flesh appearance. Microscopically, the testis and epididymis were diffusely infiltrated by cells of the lymphocytic series. The interlobular connective tissue septa were well preserved.

His postoperative course was uneventful. On July 24, 1964, he was referred to the Hospital for Sick Children, Toronto for further assessment. At this time his hemoglobin was 11.5 g./100 ml. and his leukocyte count was 1700/c.mm. with 82% lymphocytes. The bone marrow, which was almost completely replaced by lymphocytes, was reported as "acute lymphocytic leukemia". He was again treated with prednisone and 6-mercaptopurine. In April 1965 he struck his left ankle and it became red, swollen and painful. The right shoulder and left elbow were similarly affected in August. He was admitted to the Hospital for Sick Children in September. His leukocyte count had dropped to 300/c.mm. and was 650 on discharge. His hemoglobin was 9.6 g./100 ml. and the platelet count was 35,000/c.mm. He was maintained on daily doses of methotrexate.

On September 26, 1966 he was again admitted to the South Waterloo Memorial Hospital, Galt, because of fever, vomiting and abdominal cramps. He died on September 28, 1966. The postmortem examination revealed leukemic infiltration of the meninges, spleen, lymph nodes, bone marrow and left testis. The right testis had been removed earlier. Other findings included pulmonary emphysema with cor pulmonale, hemosiderosis of the spleen and fatty metamorphosis of the liver.

Case 2.—T.R., a 19-month-old male infant, was first admitted to St. Joseph's Hospital, Hamilton on February 11, 1965 with fever and pallor of recent onset. His hemoglobin was 3.8 g./100 ml., hematocrit 10 ml./100 ml., leukocyte count 2440/c.mm., platelet count 122,000/c.mm. and sedimentation rate 120 mm. in one hour (Wintrobe). On peripheral blood films, 100% of the cells were of the lymphocytic series and a diagnosis of acute leuke-

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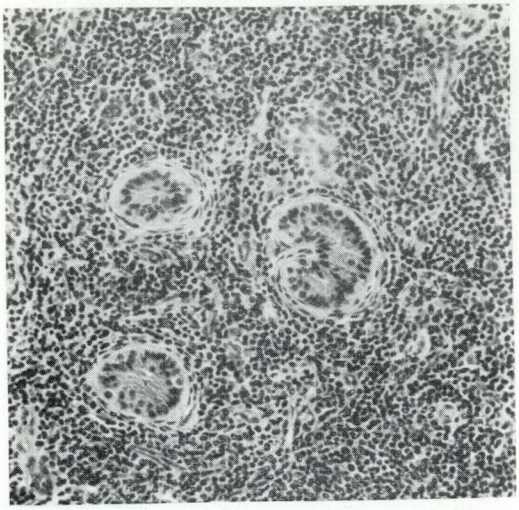

Fig. 1.—Case 2. Diffuse lymphocytic leukemic infiltration of the testis. The seminiferous tubules are isolated by the infiltrate (original magnification x 240).

mia was made. He was transfused with packed cells and treated with prednisone and 6-mercaptopurine. He was discharged 17 days after admission with a hemoglobin of 12.8 g./100 ml., leukocyte count 8550/c.mm. and a differential of 51% neutrophils, 45% lymphocytes and 4% monocytes. The platelet count was 268,000/c.mm. He was given daily doses of methotrexate and over the next 13 months remained clinically well. In April 1966 the left testicle was noted to be enlarging rapidly. It was firm and almost twice as large as the right and did not transilluminate. There were no other clinical findings. When he was admitted to hospital on April 21, 1966 his hemoglobin was 11.0 g./100 ml., hematocrit 31 ml./100 ml., leukocyte count 8900/c.mm. with a differential of 86% neutrophils, 8% lymphocytes and 6% monocytes. The left testicle, which was removed on April 22, 1966, measured 3.3 x 2 x 1.6 cm. and weighed 7 g. On section, it was yellowish-grey and fleshy. Microscopically, the entire testis was diffusely and extensively infiltrated by cells of the lymphocytic series (Fig. 1). The interlobular connective tissue septa were preserved. The interstitial tissue of the epididymis was infiltrated by leukemic cells but the spermatic cord was not involved. A lumbar puncture done at operation showed that the cerebrospinal fluid contained undifferentiated stem cells. Intrathecal methotrexate was administered.

His postoperative course was uneventful and he was maintained at home on 6-mercaptopurine. During the summer of 1966 it was noted that the right testicle was slowly enlarging and, by September, it measured 6 x 4 x 4 cm. From September 23 to 30, the right testicle was treated with deep radiation on six occasions—a total of 550 rads. In October, he was in hospital again with exacerbation of acute leukemia and bronchitis but by November he was once again in remission following a course of methotrexate and prednisone. He was maintained on this therapy and continued fairly well except for bronchopneumonia in December 1966 and the appearance of ulcers of the buccal mucosa in July 1967. At the present time (December 1967) he is in clinical remission.

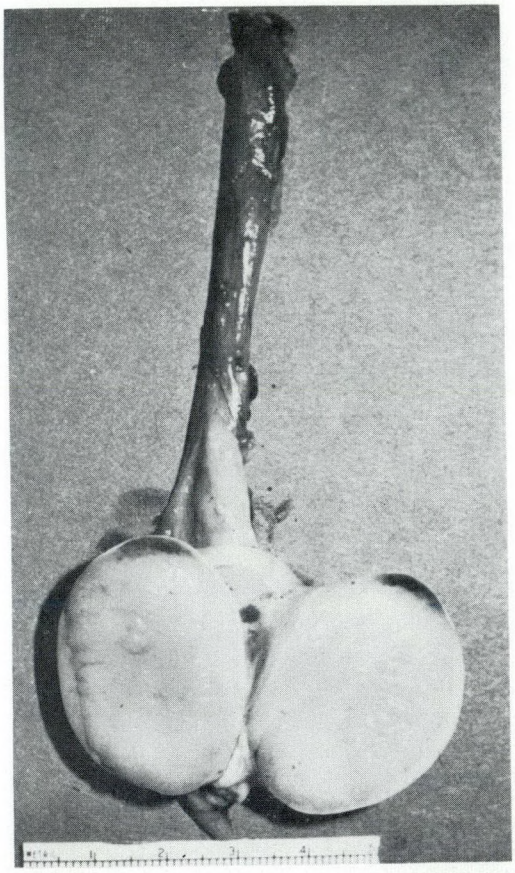

Fig. 2.—Case 3. Gross appearance of the testis illustrating diffuse leukemic infiltration (approximately full size).

Case 3.—M.H., a 6-year-old boy, was first admitted to St. Joseph's Hospital, Hamilton on March 21, 1966. A diagnosis of acute lymphyocytic leukemia had been made in June 1964 at the Hospital for Sick Children in Toronto. He had been treated with prednisone and 6-mercaptopurine and was on daily methotrexate. On admission his hemoglobin

was 11.3 g./100 ml., hematocrit 33 ml./100 ml., leukocyte count 3740/c.mm. with a differential of 74% neutrophils, 14% lymphocytes and 12% monocytes. The platelet count was 234,000/c.mm. and the sedimentation rate 105 mm. in one hour (Wintrobe). The left testicle was about twice as large as the right, firm and did not transilluminate. There were no other clinical abnormalities. On March 23 a left orchiectomy was performed. The testis measured 3.6 x 2.4 x 2.0 cm. and weighed 19 g. (Fig. 2). The cut surfaces were firm, grey and glistening, and microscopically the organ was diffusely infiltrated by cells of the lymphocytic series. Practically all spermatogonia had been lost but islands of sertoli cells persisted. The interlobular connective tissue septa were preserved (Fig. 3). The epididymis and spermatic cord were not involved.

His postoperative course was uneventful and he was maintained on daily 6-mercaptopurine. On June 30, 1966 his leukocyte count was 2500/c.mm. with a differential of 36% neutrophils, 49% lymphocytes, 4% eosinophils, 7% monocytes and 4% stem cells. The platelet count was 104,000/c.mm. A bone marrow aspirate revealed extensive displacement by undifferentiated stem cells. He was given prednisone and methotrexate and again went into clinical remission. On January 21, 1967 he was admitted to hospital with severe chicken pox and acute tracheobronchitis with bronchopneumonia. He was treated with antibiotics and gamma globulin but died three days after admission.

The postmortem examination revealed hundreds of vesicles of chicken pox over the face, chest and back. The cause of death was tracheobronchitis, with obstruction by mucus plugs, and focal bronchopneumonia. Leukemic infiltration was present in the meninges, lungs, liver, spleen, lymph nodes and bone marrow. The left testis had been removed earlier and the right was not involved by leukemic infiltration.

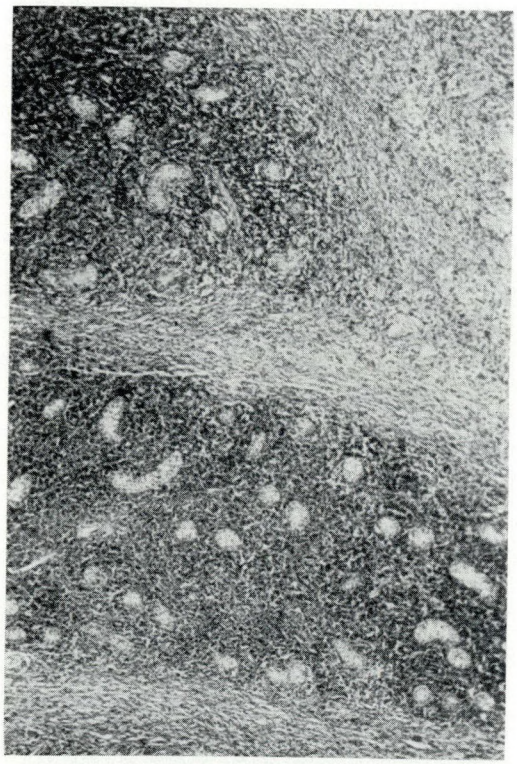

Fig. 3.—Case 3. Diffuse leukemic infiltration of the testis illustrating preservation of interlobular connective tissue septa (original magnification x 96).

Discussion

The unilateral enlargement of the testis in these three patients was due to extensive infiltration by leukemic cells of the lymphocytic series. In all microscopic sections, the testis was diffusely infiltrated with leukemic cells and the tubules, which were separated, appeared to be isolated and surrounded by malignant cells. There was remarkable preservation of the connective tissue septa which formed the normal boundaries of the lobules. This appearance is in striking contrast to that observed in primary malignant tumours which grow by expansion and destroy the peripheral, normal tissue elements by compression. Leukemic infiltration causing testicular enlargement will probably become more common in the future as newer antimetabolic agents prolong the life expectancy in children with acute leukemia. Indeed, the incidence of leukemic involvement of the testis has changed during the past half century. In their review, which covered a period of 35 years from 1913 to 1948, Watson, Sauer and Sadugor[2] described 234 patients with leukemia, one of whom had testicular infiltration. The other two patients with testicular involvement in their series were associated with generalized lymphosarcoma. Thomas and Berard[1] studied 340 necropsies of patients with acute leukemia and found microscopic evidence of leukemic infiltration of the testes in 48% of males and of the ovaries in 24% of the females.

The three patients in the present paper were seen during a three-year period, 1964 to 1967, during which time 22 children with acute leukemia were studied. Donohue[6] believes that this high incidence can probably be explained by the efficacy of modern therapy and the fact that leukemic cells, which are sequestrated in such organs as the testis and central nervous system, are not reached by the antimetabolic chemotherapeutic agents and can therefore proliferate and eventually cause clinical effects. It is unlikely that this apparent increase in incidence is due to any change in the natural history of the disease.

In D.H. (Case 1) the enlarged testicle was removed because we believed the enlargement was probably due to a primary malignant tumour. In the other two boys the testicular enlargement was attributed to leukemic infiltration. The testes were removed because both children were otherwise in clinical remission. The same end result could probably have been achieved by high-voltage radiation therapy since, in Case 2, irradiation returned the testis to normal size and there has been no clinical evidence of recurrence in a 14-month follow-up period.

Removal of the involved testis has the following advantages: it is definitive, rapid treatment and it allows the physician to obtain a firm pathological diagnosis. Although there is practically no morbidity, the patient does require a general anesthetic. The testis involved by leukemic infiltration responds rapidly to a short course of low-dosage radiotherapy. In this, the patient experiences no discomfort and very little inconvenience. However, the physician does not obtain a definitive pathological diagnosis.

Mathé *et al.*[7] investigated 31 leukemic patients in "complete remission" by doing a series of needle biopsies of the kidney, liver and testis, and by examining the bone marrow and the cerebrospinal fluid. Nineteen of them had completely negative findings; of these only one had an early relapse. However, of the 12 remaining patients with leukemic involvement in one or more of the sites studied, 6 had an early relapse. This study suggests that leukemia in a single accessible site, such as the testis, should be treated either by surgical removal or irradiation.

Primary tumours of the testis are rare in children and secondary tumours are very uncommon. Turley and Moore,[8] in a review of 5500 testicular tumours reported between 1803 and 1942 found only 131 in children. Phelan, Woolner and Hayles[9] reviewed nine malignant testicular tumours in children seen at the Mayo Clinic from 1932 to 1952. Five of these were germinal tumours and four were sarcomas which were considered to have arisen from the tunica or supportive muscle of the testis. Waddell[10] reviewed the literature concerning malignant lymphomas of the testis in children and found seven that had been reported as either primary or secondary tumours. He added one case of his own—a 7-year-old boy with a reticulum-cell sarcoma which was treated by orchiectomy. The patient died with disseminated disease two months later. Dixon and Moore,[11] who do not include lymphomas in their classification of tumours of the testis, state that "Although a few instances of primary lymphosarcoma of the testis have been reported, all have been associated with the generalized disease. The occurrence of primary testicular lymphoma is at least questionable."

Varney[12] described lymphosarcoma of the testis in five adults; he considered that the tumour was primary in two of them, although these patients had disseminated disease at the time of death. Collins and Pugh,[13] who analyzed the records of 63 patients with malignant lymphomas of the testis, state that "It is only in retrospect, after a patient has been without evidence of recurrence for some years, that a malignant lymphoma of the testis may be accepted as a primary." However, these authors do not indicate whether any of the 63 cases in their series could be so classified. Wescott,[14] who reviewed 3000 testicular tumours recorded between 1952 and 1966, considered that, with respect to lymphomas, "most authorities have felt that testicular involvement represents a part of a generalized disease process, with metastases involving multiple organs". The consensus appears to be that there is no such entity as a primary testicular lymphoma.

SUMMARY

Three boys with unilateral testicular enlargement due to leukemic infiltration were treated by orchiectomy. Although the same end result could probably have been achieved by high-voltage x-ray therapy, no definitive pathological diagnosis would have been possible. Surgical removal is probably the treatment of choice.

Leukemic infiltration of the testis may be a manifestation of modern therapy and will probably occur more frequently in the future.

Primary malignant tumours of the testis in children are extremely rare and secondary tumours are very uncommon. There is probably no such entity as a primary testicular lymphoma.

We wish to thank Drs. A. Horne and G. Mertens for the use of Case 1 and Dr. A. Zipursky for his assistance with the references.

REFERENCES

1. ROBERTS, W. C.: Personal communication.
2. WATSON, E. M., SAUER, H. R. AND SADUGOR, M. G.: Manifestations of the lymphoblastomas in the genito-urinary tract, *J. Urol.*, **61:** 626, 1949.
3. STEGAGNO, G. A., DIGILIO, G. AND FELICI, W.: On the testicular localization of infiltrative processes of the tumoral type as the first sign of recurrence in leukemic children during treatment, *Arch. Ital. Pediat.*, **21:** 502, 1960.
4. ZUELZER, W. W. AND FLATZ, G.: Acute childhood leukemia: a ten-year study, *A.M.A. J. Dis. Child.*, **100:** 886, 1960.
5. DAMESHEK, W. AND GUNZ, F.: Leukemia, Grune & Stratton, Inc., New York, 1964, p. 194.
6. DONOHUE, W. L.: Personal communication.
7. MATHÉ, G. *et al.*: Extensive histological and cytological survey of patients with acute leukaemia in "complete remission", *Brit. Med. J.*, **1:** 640, 1966.
8. TURLEY, H. K. AND MOORE, T. D.: Malignant lymphoma primarily manifested as a testicular tumor, *J. Urol.*, **68:** 744, 1952.
9. PHELAN, J. T., WOOLNER, L. B. AND HAYLES, A. B.: Testicular tumors in infants and children, *Surg. Gynec. Obstet.*, **105:** 569, 1957.
10. WADDELL, R. W.: Testicular lymphoblastomas in children: report of a case and review of the literature, *J. Urol.*, **85:** 956, 1961.
11. DIXON, F. J. AND MOORE, R. A.: Tumors of the male sex organs. Atlas of tumour pathology. Sec. 8, Fasc. 31B and 32, United States Armed Forces Institute of Pathology, Washington, D.C., 1952, p. 111.
12. VARNEY, D. C.: Lymphosarcoma of the testis, *J. Urol.*, **73:** 1081, 1955.
13. COLLINS, D. H. AND PUGH, R. C. B.: The pathology of testicular tumors, *Brit. J. Urol.*, **36** (Suppl.): 1, 1964.
14. WESCOTT, J. W.: Reticulum cell sarcoma with primary manifestation in the testis, *J. Urol.*, **96:** 243, 1966.

RÉSUMÉ

Trois enfants âgés respectivement de 19 mois, de 6 ans et de 10 ans ont présenté une hypertrophie testiculaire unilatérale, secondaire à une infiltration leucémique. Tous trois ont été traités par orchidectomie. Dans l'un des cas, l'autre testicule commença à grossir et fut traité par radiothérapie profonde (550 rads au total). Le meilleur traitement pour une hypertrophie testiculaire unilatérale chez des enfants atteints de leucémie aiguë est probablement l'orchidectomie, parce qu'elle est rapide et définitive; la morbidité est négligeable et l'on peut obtenir un diagnostic sans équivoque. Dans nos trois cas, le testicule était le siège d'infiltration généralisée de cellules leucémiques, mais le tissu conjonctif interlobulaire et quelques tubules séminifères étaient bien conservés, ce qui contraste fortement avec l'apparence des tumeurs malignes qui prolifèrent en détruisant les tissus périphériques normaux. L'hypertrophie testiculaire secondaire à une infiltration leucémique, qui est la conséquence de la thérapeutique moderne, deviendra plus fréquente à mesure que la survie de l'enfant atteint de leucémie aiguë sera mieux prolongée par de nouveaux antimétabolites. Les tumeurs malignes primaires du testicule sont rares chez l'enfant. Une revue critique de la littérature permet de croire que le lymphosarcome primaire du testicule est une entité probablement inexistante.

SEAT-BELT TRAUMA TO THE ABDOMEN*

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OVER the past 10 to 15 years, with increasing use of the automobile there has been a corresponding increase in automobile accidents. During this time the use of seat belts has also increased. Such use is now well accepted and the value of these belts is well documented.[1-5] Injury to the abdomen due to seat belts has been reported in only 38 instances. The three cases in the present report bring the total to 41. The details of these 41 cases are summarized in Table I.[1, 2, 6-21]

Our three patients were seen in a single year at the Victoria General Hospital, Halifax, Nova Scotia; in the same period, 15 patients with blunt abdominal injury were operated upon in this hospital.

Although shoulder-type belts are worn much less frequently, these were responsible for 5 of the 38 injuries reported to date. Of 29 in which a lap-type seat belt was implicated, there was a tear or perforation of the bowel and/or its mesentery in 22.

Some workers believe that the abdominal injuries associated with seat belts are the result of improper application of the belts.[17] In our patients, the seat belts were properly applied as indicated by the patients' statements and by the distribution of the seat-belt abrasions, all of which were below the iliac crests. Despite this,

TABLE I.—DETAILS OF 41 REPORTED CASES OF SEAT-BELT TRAUMA

Authors	*Number of cases*	*Type of belt*	*Abdominal injuries*	*Delay in treatment*	*Course*
Kulowski and Rost[6]	1	Lap	Contusion of distal ileum adherent to pelvic brim—intestinal obstruction	None	Cured
von Bahr and Erikson[7]	4	Shoulder (all improperly applied)	(1) Rupture of left kidney, left renal vein torn from aorta	None	(1) Cardiac arrest on way to operating room
			(2) Rupture of liver and spleen	None	(2) Transferred to other hospital, dead on arrival
			(3) Rupture of right kidney	None	(3) Recovered
			(4) Fractured ribs and tear of liver	1 hour	(4) Recovered
Engberg[8]	1	Shoulder	Hepatic veins torn from vena cava	1 hour	Died in shock at operation
Garrett and Braunstein[9]	2	Lap	(1) Rupture of duodenum and pancreas	Unknown	(1) Prolonged hospital recovery
			(2) Renal and bladder contusions	Unknown	(2) Recovered
Cocke and Meyer[10]	1	Lap (loose)	Shattered spleen, fractured ribs	5 hours	Complicated recovery
Aiken[11]	1	Lap	Perforation of jejunum	6 days	Recovered
Tolins[12]	1	Lap	Perforation of jejunum	4 days	Complicated recovery
Rubovits[13]	1	Lap	Rupture of pregnant uterus	2 days	Recovered
Fisher[14]	1	Lap	Lacerated spleen	None	Recovered
Fish and Wright[15]	4	Lap (air crash)	(1) Rupture of ileum	9 days	(1) Recovered
			(2) Rupture of ileum	13 days	(2) Recovered
			(3) Mesenteric rent	None	(3) Recovered
			(4) Mesenteric rent	None	(4) Died of hemorrhage
Hurwitt and Silver[16]	1	Lap (loose)	Hernia of colon, small bowel and stomach	None	Recovered
Williams, Lies and Hale[17]	4	Lap	(1) Rupture of ileum	3 hours	(1) Recovered
			(2) Multiple hematomas of small bowel, amputation of portion of omentum, laceration of mesentery	5 hours	(2) Recovered
			(3) Tear of duodenum, junction of second and third portions	5 hours	(3) Recovered
			(4) Tear of mesentery of ileum, tear of mesosigmoid	12 hours	(4) Recovered

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TABLE I (*continued*)

Authors	*Number of cases*	*Type of belt*	*Abdominal injuries*	*Delay in treatment*	*Course*
Gerritsen, Frobese and Pezzi[18]	2	Unknown	(1) Laceration of jejunum, multiple lacerations of mesentery, amputation of portion of omentum	Unknown	(1) Unknown
			(2) Laceration of ileum, tears of mesentery and serosal tear of sigmoid	Unknown	(2) Unknown
Sube *et al.*[19]	2	Lap	(1) Mesenteric rent	None	(1) Recovered
			(2) Transection of jejunum	8 days	(2) Recovered
LeMire, Earley and Hawley[20]	2	Lap	(1) Hernia of colon and small bowel	None	(1) Recovered
			(2) Perforation of proximal jejunum	10 hours	(2) Recovered
Porter and Green[21]	3	Lap	(1) Avulsion of terminal ileum, perforation of upper ileum and mid-jejunum, necrosis of sigmoid	3 days	(1) Stormy and prolonged convalescence, eventual recovery
		Unknown	(2) Transection of jejunum, transection of rectus abdominis	2 days	(2) Wound infection, recovered
		Unknown	(3) Perforation of jejunum, avulsion of mesentery, rupture of spleen	2 hours	(3) Enteric fistulas, died of pulmonary embolism
Blumenberg[1]	1	Lap	Sigmoid perforation and mesosigmoid tear	4 days	Recovered
Witte[2]	6	Lap	(1) Perforation of duodenum	24 hours	(1) Recovered
			(2) Perforation of small bowel	24 hours	(2) Recovered
			(3) Avulsion of ileum, tear of mesentery	2 hours	(3) Recovered
			(4) Tears of mesentery, avulsion of greater omentum	3 hours	(4) Recovered
			(5) Tears of mesentery, perforation of jejunum	2 hours	(5) Recovered
			(6) Perforation of ileum	8 days	(6) Died
MacLeod and Nicholson (1969)	3	Lap	(1) Perforation of ileum, seromuscular tears of small bowel and sigmoid, small mesenteric rent	12 hours	(1) Recovered
			(2) Perforation of jejunum	17 hours	(2) Recovered
			(3) Transection of distal ileum, mesenteric rent	19 hours	(3) Recovered

these patients sustained serious intra-abdominal injury.

Case Reports

Case 1.—W.B., a 32-year-old man, was admitted to hospital following an automobile accident one hour earlier. He was driving and had been wearing a lap-type seat belt at the time of his accident. The car was completely demolished. His major complaint was lower abdominal pain.

On examination, he was alert but in obvious pain. He had a small laceration over his right eye. His blood pressure was 190/110 mm. Hg and his pulse was 92/min. The abdomen was diffusely tender but more so over the lower portion, where there was some guarding and minimal rebound tenderness. The abdomen was also abraded. His hemoglobin on admission was 14.5 g./100 ml. The urine contained red blood cells. Radiographs of the chest, abdomen, kidneys and bladder were within normal limits. A radiograph of the spine showed a slight compression fracture of L3. Over the next eight hours his blood pressure and pulse did not change; however, his abdominal pain increased. Repeated examination showed only generalized abdominal tenderness, guarding and rebound tenderness. His leukocyte count at this time was 20,700/c.mm. with 27% band cells.

Twelve hours after injury his abdomen was explored through a left paramedian incision. There was obvious peritonitis. He had multiple seromuscular tears involving the sigmoid colon and small bowel. In the small bowel, three feet from the ileocecal valve, there was a 2-cm. circular perforation on the antimesenteric side. This was closed with one layer of 00 silk Lembert sutures and the other seromuscular tears were repaired. We explored the remainder of the abdominal cavity and found no further abnormality. The abdomen was then closed in layers. His postoperative course was uncomplicated and he was discharged eight days after the operation.

Case 2.—L.R.A., an 18-year-old man, was seen one hour after an automobile accident. A passenger in the front seat, he had been wearing a lap-type seat belt which was so

tight that he could not move or twist his body; in addition, he was asleep when the accident occurred.

On admission to hospital he complained of generalized abdominal pain. There was generalized guarding, a moderate degree of tenderness over the lower abdomen, and skin abrasions below the iliac crests. There was no evidence of other injuries. His blood pressure was 110/70 mm. Hg and his pulse was 92/min. His hemoglobin was 15.8 g./100 ml., his hematocrit 45 ml./100 ml., leukocyte count 13,025/c.mm. and his serum amylase 65 Somogyi units/100 ml. Radiographs of the abdomen showed no free air. Our diagnosis at this time was abdominal-wall contusion with a possible intraperitoneal injury. Gastric suction and intravenous therapy were begun at once. Ten hours after the accident, his serum electrolytes were normal, his hemoglobin was 15.4 g./100 ml., leukocyte count 12,975/c.mm. and hematocrit 43.5 ml./100 ml. An intravenous pyelogram was normal. However, his pain and tenderness on palpation persisted and the rigidity and rebound pain increased. Bowel sounds disappeared. His abdomen was explored 17 hours after the accident.

The abdomen was opened through a left paramedian incision. There was a small amount of clear intraperitoneal fluid, and a thick exudate on the serosal surface of the small bowel and its mesentery. We found a 1-cm. perforation on the antimesenteric surface of the mid-jejunum which was closed in one layer with interrupted Lembert sutures of 000 silk. The peritoneal cavity was drained through a stab wound and the abdomen closed in layers. His postoperative course was uncomplicated and he was discharged nine days after operation.

Case 3.—W.F.M., a 22-year-old white man, was admitted following the same accident as L.R.A. (Case 2). He was driving the car and said that his belt was applied as tightly as possible. He estimated his speed at the time of impact was approximately 70 m.p.h. The car was demolished.

He complained of steady aching pain in the right flank and right hip regions. There were abrasions below the iliac crests, more marked on the right side. Slight tenderness was noted on the right side of the abdomen and the right flank. He had no evidence of other injuries except for a contusion of the right frontal region. Radiographs of the skull and pelvis were negative. A flat plate of the abdomen showed no evidence of free air. However, there was a suggestion of some displacement of the ascending colon which was interpreted as a retroperitoneal hematoma. At this time we believed he had a retroperitoneal hematoma and perhaps intraperitoneal injury.

Ten hours after the accident, his pain had increased slightly, but the tenderness was not marked. He had mild abdominal distension, and some ecchymosis had appeared in the right flank. Bowel sounds were present. At this time, his hemoglobin was 18.5 g./100 ml., hematocrit 55.4 ml./100 ml., leukocyte count 15,200/c.mm. An intravenous pyelogram was normal. Six hours later, because of increasing pain and tenderness, moderate distension and diminished bowel sounds, we decided to operate.

When a right paramedian incision was made in the lower abdomen, we found less than 100 c.c. of free blood in the peritoneal cavity. In the mesentery of the proximal ileum, there was a rent 3 cm. in length. At this point, a 2-cm. segment of the ileum was denuded of its mesentery. However, the circulation of the bowel appeared to be satisfactory and the rent in the mesentery was repaired. On further search, we found another rent in the mesentery of the distal ileum that extended from its root to the bowel wall. At this point the bowel was completely transected. A hematoma in the root of the mesentery extended behind the posterior peritoneum. We resected this portion of the ileum and did an end-to-end anastomosis. The tear in the mesentery was repaired. Examination of the remainder of the abdomen showed no further injury. The peritoneal cavity was drained through a stab wound and the abdomen closed in layers. The patient's postoperative course was uncomplicated and he was discharged nine days later.

Mechanism of Injury

During thorough questioning, these three patients insisted that they had been wearing their seat belts properly and that the belts fitted tightly. The abrasions on their bodies confirmed these statements. A properly fitted seat belt, however, still rides above the symphysis pubis and is therefore in direct contact with the lower abdominal wall. In all three there was rapid deceleration. The mechanism of injury in these cases was probably a combination of bowel compression by the seat belt, acute flexion of the trunk, an increase in intra-abdominal pressure and, as a consequence, entrapment of a segment of bowel. Compression of the bowel between the seat belt and spine probably produces the seromuscular and mesenteric tears so fre-

quently seen. The circular perforation (Case 1) must represent a blow-out that resulted from entrapment and the high intraluminal pressure. With tight or properly applied seat belts, injury to the lower abdomen is probably more frequent. With a loose-fitting lap belt or a diagonal-type shoulder belt, the injuries are usually to the upper abdomen and may involve solid organs.

Discussion

Motor vehicle accident victims who have seat-belt injuries and abdominal complaints should be studied for possible intra-abdominal injury. If they have abrasions beneath the seat belt, they have been exposed to significant force and intra-abdominal injury is all the more likely.

The early clinical findings may not be impressive. Pain, tenderness and guarding may be slight, and yet they may have serious intra-abdominal injury. The physician should not readily accept the diagnosis of uncomplicated abdominal-wall contusion. Persisting or increasing abdominal tenderness and guarding are particularly suggestive of intra-abdominal injury and, if managed conservatively, the patient must be observed closely.

With these clinical findings, a laparotomy is justified even if there is no evidence of blood loss or inflammation. Even if the laparotomy does not reveal a lesion requiring operative treatment, the mortality in patients without serious associated injury[22] is not significantly increased. Particularly in patients who have no associated chest injury, localized abdominal findings warrant exploration before generalized peritonitis develops.

Of the patients with intestinal perforation described in the literature, only 50% were explored within the first 24 hours. It is also apparent from the literature that the greater the delay the greater the morbidity. In the collected series to date, the mortality of uncomplicated intestinal perforation has been low; in one instance, exploration was delayed as long as eight days and the patient eventually recovered.[19]

Radiographs have not been helpful in the diagnosis of these intra-abdominal injuries and abdominal paracentesis is of doubtful value in these patients.

Summary

To date, 41 patients have been described with abdominal injury associated with the use of seat belts; 32 of these wore lap-type belts. Of these 32, 25 had perforations of the intestine and/or mesenteric rents. The interval from injury to operation was unduly long in most of these patients.

The early findings, although not marked, are compatible with a localized peritonitis. Generalized peritonitis usually becomes apparent within 12 to 18 hours if the patient has a perforated bowel.

In conscious patients, without chest injuries, who complain of abdominal pain following an automobile accident, intra-abdominal injury should be suspected, particularly if there are abrasions in the distribution of the seat belt. The decision to explore must be based on the clinical findings because radiographs and abdominal paracentesis are not generally helpful.

Conclusions

In an automobile accident, serious intra-abdominal injury may occur despite properly applied seat belts. Following lap-type seat-belt trauma to the abdomen, the most frequent injury is perforation of the bowel and/or mesenteric rent.

Uncomplicated abdominal-wall contusion is a dangerous diagnosis.

In all probability, the three patients reported here would have suffered serious, possibly fatal, injury if they had not been wearing seat belts. The authors of this report do not wish to suggest that seat belts *per se* are hazardous.

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References

1. Blumenberg, R. M.: The seat belt syndrome: sigmoid colon perforation, *Ann. Surg.*, **165:** 637, 1967.
2. Witte, C. L.: Mesentery and bowel injury from automotive seat belts, *Ann. Surg.*, **167:** 486, 1968.
3. Frazier, R. G.: Hazards to health: effectiveness of seat belts in preventing motor-vehicle injuries, *New Eng. J. Med.*, **264:** 1254, 1961.

4. CAMPBELL, H. E.: Role of the safety belt in 19 auto crashes, *Bull. Amer. Coll. Surg.*, **40:** 155, 1955.
5. CLARKE, R.: Closed abdominal injuries, *Lancet*, **2:** 877, 1954.
6. KULOWSKI, J. AND ROST, W. B.: Intra-abdominal injury from safety belt in auto accident. Report of a case, *A.M.A. Arch. Surg.*, **73:** 970, 1956.
7. VON BAHR, V. AND ERIKSON, E.: Injuries caused by safety belts, *Svenska Läk.-Tidn.*, **58:** 141, 1961.
8. ENGBERG, A.: Injuries caused by safety belts. A contribution to the discussion with reference to an unusual case, *Svenska Läk.-Tidn.*, **58:** 884, 1961.
9. GARRETT, J. W. AND BRAUNSTEIN, P. W.: The seat belt syndrome, *J. Trauma*, **2:** 220, 1962.
10. COCKE, W. M., JR. AND MEYER, K. K.: Splenic rupture due to improper placement of automobile safety belt, *J. A. M. A.*, **183:** 693, 1963.
11. AIKEN, D. W.: Intestinal perforation and facial fractures in an automobile accident victim wearing a seat belt, *J. Louisiana Med. Soc.*, **115:** 235, 1963.
12. TOLINS, S. H.: An unusual injury due to the seat belt, *J. Trauma*, **4:** 397, 1964.
13. RUBOVITS, F. E.: Traumatic rupture of the pregnant uterus from "seat belt" injury, *Amer. J. Obstet. Gynec.*, **90:** 828, 1964.
14. FISHER, P.: Injury produced by seat belts: report of 2 cases, *J. Occup. Med.*, **7:** 211, 1965.
15. FISH, J. AND WRIGHT, R. H.: The seat belt syndrome—does it exist?, *J. Trauma*, **5:** 746, 1965.
16. HURWITT, E. S. AND SILVER, C. E.: Seat-belt hernia. A ventral hernia following an automobile crash, *J. A. M. A.*, **194:** 829, 1965.
17. WILLIAMS, J. S., LIES, B. A., JR. AND HALE, H. W., JR.: The automotive safety belt: in saving a life may produce intra-abdominal injuries, *J. Trauma*, **6:** 303, 1966.
18. GERRITSEN, R., FROBESE, A. S. AND PEZZI, P. J.: Unusual abdominal injuries due to seat belts, *J. A. Einstein Med. Cent.*, **14:** 63, 1966.
19. SUBE, J. *et al.*: Seat belt trauma to the abdomen, *Amer. J. Surg.*, **113:** 346, 1967.
20. LEMIRE, J. R., EARLEY, D. E. AND HAWLEY, C.: Intra-abdominal injuries caused by automobile seat belts, *J. A. M. A.*, **201:** 735, 1967.
21. PORTER, S. D. AND GREEN, E. W.: Seat belt injuries, *Arch. Surg. (Chicago)*, **96:** 242, 1968.
22. TOURIN, B.: Ejection and automobile fatalities, *Public Health Rep.*, **73:** 381, 1958.

RÉSUMÉ

Nous avons eu l'occasion de traiter trois personnes voyageant dans des autos et qui, bien qu'ayant attaché convenablement leur ceinture de sécurité, subirent des lésions abdominales. Tous trois avaient des perforations de l'intestin avec déchirures mésentériques. Les auteurs passent la littérature en revue et discutent le mécanisme de la lésion. Le siège de celle-ci peut différer: la bretelle de sécurité, tout comme la ceinture mal fixée ou trop lâche, provoque habituellement des lésions dans la portion supérieure de l'abdomen, particulièrement aux organes fixes, tandis que les ceintures, même bien fixées, causent des lésions dans la portion inférieure de l'abdomen, particulièrement des perforations intestinales et des déchirures mésentériques. C'est surtout le grêle qui est touché, plus rarement le sigmoïde.

Une flexion brusque, un violent coup direct, une augmentation de la pression intra-abdominale et le coincement d'une anse intestinale entre la boucle de la ceinture et la colonne vertébrale peuvent jouer un rôle. Les auteurs brossent le tableau clinique et signalent que les téguments de l'abdomen situés dans la région de contact de la ceinture sont souvent écorchés. Ils soulignent aussi l'importance d'une laparotomie d'urgence pour réduire la morbidité. Enfin, ils rappellent les avantages intrinsèques des ceintures de sécurité.

HYPERBARIC OXYGEN AND IN VITRO CONSERVATION OF THE KIDNEY

Various groups of investigators have reported different degrees of success with *in vitro* conservation of the kidney under hypothermia and exposed to high pressure oxygen. In this series of experiments, the dogs' kidneys were cooled to 2° to 4°C. and exposed to an oxygen pressure of three to eight atmospheres. The kidney was then implanted in the same dog and the contralateral kidney was removed. The addition of the high pressure oxygen to hypothermia did not improve the results. It appears that the oxygen cannot diffuse throughout the organ.—Fontaine, J.-L. *et al.*: Oxygène hyperbare et conservation in vitro du rein. Résultats expérimentaux chez le chien, *J. Chir.*, **96:** 215, 1968.

LYMPHOMAS OF SMALL BOWEL AND THEIR RELATIONSHIP TO IDIOPATHIC STEATORRHEA

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MALIGNANT tumours of the small bowel are relatively rare, and comprise less than 5% of all tumours of the gastrointestinal tract. There is, as yet, no satisfactory explanation for this low incidence although the alkalinity of small-bowel contents, and rapid transit with short exposure of the mucosa to possible carcinogenic agents have been proposed. The effect on carcinogenesis of the rapid turnover of the epithelial cells of the small-bowel mucosa is unknown. In the duodenum almost all the malignant lesions are carcinomas; in the jejunum, carcinomas and leiomyosarcomas occur in about equal frequency along with a few lymphomas; but in the ileum, lymphomas predominate, no doubt because of the greater amount of lymphoid tissue there.

This paper describes a study of malignant tumours of the small bowel that are called "malignant lymphomas" or "reticuloses". In the course of this disease many systems of the body may be involved; however, patients were accepted for this study only if the process appeared to have begun in the small bowel and its mesentery. At the Toronto General Hospital, 45 patients were seen with lymphomas of the small bowel, proved at operation or autopsy; of these, 18 fulfilled the criteria for inclusion in the series. In the remainder, the small-bowel lesions were incidental to extensive involvement elsewhere.

CLINICAL FEATURES

Eleven of these 18 patients were female; one-third were under 40 years of age. All but one had had vague symptoms of ill health such as anorexia, weight loss, fatigue or weakness before more specific complaints developed that were referable to the abdomen. Eight patients were admitted to hospital as "emergencies"; five with perforation, two with massive bleeding, and one with small-bowel obstruction. Of those admitted because of abdominal symptoms, seven had a change in bowel habit and weight loss, and three had a mass with no change in bowel habit. One patient had no symptoms; an abdominal mass was discovered on routine examination. In 11, a portion of bowel was resected; in 2, this was considered curative. In two with intestinal obstruction, a bypass anastomosis was done; only biopsy was possible in two more. The remaining patients were too ill for operative treatment. Two patients are alive and apparently free of tumour, 12 years and 2 years after resection of lymphosarcomas from the jejunum and ileum. The former was given postoperative radiotherapy and the latter nitrogen mustard. Seven patients died in hospital while their initial treatment was being given; the remaining nine survived for an average of four months. Gastrointestinal radiological examination did not contribute to the diagnosis which was usually made by the pathologist after he had examined the resected material, or at autopsy. The most frequent clinical diagnoses were retroperitoneal sarcoma, Crohn's disease, and idiopathic steatorrhea.

PATHOLOGY

It is sometimes difficult for the pathologist to classify these tumours accurately. In the 18 patients, the final diagnosis was reticulum-cell sarcoma in 10, lymphosarcoma in 6, and Hodgkin's disease in 2. The major part of the tumour was in the mesentery in one-half the patients; in the remainder, there was extensive bowel involvement. Two-thirds of the bowel tumours were found in the ileum and, in one-third, more than one area was involved. Lymphosarcomas, because they re-

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sponded better to radiation, had a better prognosis.

Small-Bowel Lymphomas and Steatorrhea

Thirty years ago, Fairley and Mackie[1] suggested that malignant lymphoma of the small bowel might be related to steatorrhea of the type which simulates adult celiac disease—idiopathic steatorrhea. Before 1950, steatorrhea in patients with lymphoma was attributed either to blockage of mesenteric nodes by tumour or diffuse infiltration of the wall of the small bowel. In 1962, Gough, Read and Naish,[2] postulated that small-bowel lymphomas may develop as a complication in patients with idiopathic steatorrhea. A study of four patients in this group with lymphomas and steatorrhea is presented in order to investigate this possible relationship further.

Case Reports

Case 1.—T.S., a 35-year-old spinster, who weighed only 85 lb. was admitted to hospital in February 1954 because of diarrhea (8 to 12 stools per day for six months), abdominal distension, weight loss, and ankle edema. For years, in spite of an excellent appetite, she had failed to gain weight. Six years previously, a sister who had similar complaints was found to have idiopathic steatorrhea and responded to treatment. T.S. had a reduced hemoglobin, low serum proteins, calcium, and cholesterol, a prothrombin time of 25 seconds, and a stool fat excretion of 36%. The curve of the xylose tolerance test was flat. A barium motor meal had the usual features of malabsorption. As a result a diagnosis of idiopathic steatorrhea was made and appropriate treatment was begun. The patient was discharged after 68 days, free of symptoms and with marked improvement in all her biochemical tests. The xylose tolerance curve was normal and her weight had increased to 113 lb. Nine months later her symptoms recurred; over the next four months, in spite of treatment, she went downhill and died, 10 months after her first discharge from hospital. At autopsy, a pleomorphic reticulum-cell sarcoma was found which involved mesenteric nodes, retroperitoneum and, to a slight extent, the jejunum, the kidneys and the adrenals.

Case 2.—Mrs. E.K., a 53-year-old woman, was admitted to hospital in January 1967 because of nausea, belching and intermittent diarrhea which had begun in childhood. In December 1962, she had undergone a partial gastrectomy for duodenal ulcer, after which her symptoms improved, except for the diarrhea which, at the present admission, consisted of 10 foul, fatty stools per day. The patient was cachectic; her blood pressure was 90/50 mm. Hg and she had marked ankle edema. Soft nodes were found in both axillae. Laboratory tests were as follows: normal blood smear, prothrombin time 21 seconds, low serum proteins, cholesterol and calcium, a flat xylose tolerance curve and stool fat excretion of 47%. On light and electron microscopy, a peroral jejunal biopsy showed villous atrophy of the type seen in idiopathic steatorrhea. In an axillary node, we found evidence of Hodgkin's disease. Therapy including gluten-free diet, steroids, and psyllium (Metamucil) brought no improvement and the patient lost appetite and weight, and died 2½ months after admission. Just before death, she had a massive gastrointestinal hemorrhage and peritonitis. At autopsy, we found a malignant lymphoma of the Hodgkin's type which involved the small-bowel mesentery and, to a minimal degree, the jejunum and axillary nodes. The immediate cause of death was acute, fulminating, necrotizing, bacterial gastroenteritis.

Case 3.—C.C., a 64-year-old man, was admitted to hospital in December 1966 because of massive, bright red, rectal bleeding that required multiple transfusions before it subsided five days later. Over the previous six months, he had had diarrhea, abdominal distension, excessive flatus and had lost 30 lb. Other than lower abdominal tenderness, there were no significant physical findings. Investigation revealed no abnormalities except changes in the gastrointestinal barium meal compatible with the malabsorption syndrome. On peroral jejunal biopsy, we found subtotal villus atrophy of the type seen in idiopathic steatorrhea. Later, the patient suddenly developed acute peritonitis. At operation we found that the jejunum had perforated through an area of reticulum-cell sarcoma. This area was resected but nothing was done about many other areas where similar tumours were seen. Later another area of tumour perforated and the patient died. At autopsy, we found multiple tumour nodules that involved the jejunum and, to a lesser extent, the ileum. No normal mesenteric nodes were found.

Case 4.—Miss F.P., a 68-year-old woman, was admitted to hospital in August 1967 with

acute peritonitis. In 1960 she had been treated for anemia and diarrhea. In 1963 a diagnosis of idiopathic steatorrhea was made when a jejunal biopsy showed atrophic villi. At this time her stool fat was 35%, and her xylose tolerance curve was low. Her response to a gluten-free diet and prednisone was excellent. She gained 20 lb. and had only two formed stools each day. She continued this way for four years and then, two months before admission, in spite of treatment, she became ill with abdominal discomfort and stools of increasing frequency. Acute peritonitis developed 12 hours before admission. At operation, we found that a segment of jejunum had perforated through an area of Hodgkin's disease. This segment was resected but the patient died nine days after operation. At autopsy, Hodgkin's disease was found in two areas of jejunum but there was no tumour in the mesenteric nodes or elsewhere in the body.

Discussion

There appears to be a relationship between idiopathic steatorrhea and small-bowel lymphomas. The long history of bowel dysfunction in three patients (Cases 1, 2 and 4) suggests that the steatorrhea preceded the lymphoma. In two more (Cases 2 and 4) the response to treatment for steatorrhea was excellent initially but became poor later, indicating a change in the pathological process in the small bowel. Thus, it might be assumed that the lymphoma occurred after the onset of steatorrhea. Certainly, jejunal biopsies in patients with idiopathic steatorrhea demonstrate a change from normal in architecture and in the cellular pattern of the mucosa. Harris *et al.*[3] followed 202 patients with idiopathic steatorrhea for an average of 8.2 years, and found 14 lymphomas and 13 carcinomas of the gastrointestinal tract. No patient who had a gastrointestinal carcinoma had had steatorrhea for less than 10 years. From their study, they concluded that a gluten-free diet appeared to decrease the risk of malignant complications.

Summary

Patients with lymphoma of the small bowel have a period of chronic ill health before abdominal symptoms begin. Almost one-half these patients are operated upon as emergencies because of perforation, obstruction or massive bleeding. Without earlier diagnosis, the chances of cure or significant palliation by operation, radiation or chemotherapy are poor. There is evidence to support the claim that lymphomas may develop as a complication of idiopathic steatorrhea. This change should be suspected when the patient with the features of idiopathic steatorrhea fails to respond to the usual management or relapses after a period of successful treatment. Lymphomas associated with steatorrhea are more likely to be in the jejunum and usually are reticulum-cell sarcomas or Hodgkin's disease. From this study of 18 such patients, we have concluded that all patients with a diagnosis of idiopathic steatorrhea should be followed closely for many years and early abdominal exploration should be seriously considered if they relapse despite good treatment.

References

1. Fairley, N. H. and Mackie, F. P.: The clinical and biochemical syndrome in lymphadenoma and allied diseases involving the mesenteric lymph glands, *Brit. Med. J.*, **1:** 375, 1937.
2. Gough, K. R., Read, A. E. and Naish, J. M.: Intestinal reticulosis as a complication of idiopathic steatorrhea, *Gut*, **3:** 232, 1962.
3. Harris, O. D. *et al.*: Malignancy in adult coeliac disease and idiopathic steatorrhoea, *Amer. J. Med.*, **42:** 899, 1967.

Résumé

Les malades présentant des lymphomes de l'intestin grêle traversent une période de mauvaise santé chronique avant que n'apparaissent les symptômes abdominaux. Près de la moitié des cas sont opérés d'urgence à cause d'une perforation, d'une occlusion ou d'une forte hémorragie. Sans un diagnostic précoce, les chances de guérison ou de traitement palliatif valable, par intervention chirurgicale, par radiopathie ou par chimiothérapie, sont assez rares. Certains faits semblent prouver que le lymphome serait une complication de la stéatorrhée idiopathique. Cette complication devrait être soupçonnée lorsque le malade souffrant de stéatorrhée idiopathique ne répond plus au traitement habituel ou fait une rechute après un traitement couronné de succès. Les lymphomes associés à la stéatorrhée sont situés principalement dans le jéjunum et sont des réticulo-sarcomes ou des sarcomes de Hodgkin. Nous avons fait une étude de 18 malades et nous en concluons que tous les cas de stéatorrhée idiopathique doivent être suivis pendant de nombreuses années; une exploration abdominale précoce doit être sérieusement envisagée en cas de rechute après un bon traitement.

REVIEW ARTICLE

CLINICAL COURSE AFTER GASTRIC FREEZING: LONG-TERM FOLLOW-UP OF 74 PATIENTS AND A REVIEW OF THE LITERATURE*

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At St. Joseph's Hospital, Toronto, from May 1963 to June 1964, gastric freezing was done for duodenal ulcer in 85 patients who otherwise would have required operation. Gastric secretory tests were performed before and after the freezing. The results obtained and the statistical analysis of these results were reported previously by McIntyre and Brindis.[1] We stopped gastric freezing in June 1964 because the short duration of gastric secretory depression convinced us that gastric freezing would not cure duodenal ulcer.

Subsequently, although the morbidity and mortality of this procedure were extremely low, others also stopped because of the high incidence of recurrence of ulcer.

Even though the procedure has been abandoned in most centres, we wish to report our follow-up of the clinical response to freezing in 74 of these 85 patients and discuss the relationship of this response to the alterations in gastric secretion. The interesting, and as yet unexplained, feature of this follow-up is that 17 of these 74 patients continue to have complete or marked relief of symptoms. The complications associated with this procedure are also discussed and pertinent literature on gastric hypothermia is reviewed.

Materials and Method

Of the 85 patients, 78 were men and 7 were women. They ranged in age from 19 to 62 years—most were between 25 and 40 years. Early in the series, four patients underwent a second gastric freezing; thus, the procedure was done 89 times. Subsequently, we decided to consider the recurrence of symptoms as indicating that this method of treatment had failed and refreezing was no longer recommended.

All patients had radiological evidence of duodenal ulcer, either as a crater or severe deformity of the duodenum. In all, the characteristic symptoms had persisted despite medical treatment. Many had had perforations or hemorrhage, but none had suffered from pyloric obstruction or gastric ulcer.

In these patients, standard gastric freezing was done for 60 minutes using the method of Wangensteen.[2-4] The gastric balloon was filled with absolute alcohol, circulated and cooled with a Swenko unit, maintaining an average inflow temperature of −17° C. and an outflow temperature of −10° C.

Complications of Freezing

Gastrointestinal Bleeding

Five patients (5.9%) bled from the upper gastrointestinal tract; they had either hematemesis on the day of the freezing or hematemesis and melena during the next two days. Two vomited small quantities of blood (less than 250 c.c. of black blood) and did not require a transfusion. Three had more severe blood loss, remained in hospital, and were given several transfusions. One of these latter patients had previously bled three times from a duodenal ulcer but, in this episode, there was a definite relationship between the bleeding and the freezing. All five made an excellent recovery and had no further bleeding.

Gastric Ulcer

Three patients (3.5%) developed a gastric ulcer; later, two were treated by gastric resection and one by vagotomy and

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pyloroplasty. A fourth patient developed a gastric ulcer 3.8 years after freezing; this was treated by vagotomy and subtotal gastrectomy at another hospital. There was no instance of perforation of the stomach.

Circulatory Collapse

During freezing, two patients developed shock—with faintness, pallor, and clammy extremities, associated with a fall in blood pressure and tachycardia. Active, voluntary muscle exercises restored the circulatory hemodynamics to normal.

Damage to Surrounding Organs

Pancreas.—Three patients had elevation of serum amylase, with values of 420, 254 and 208 Somogyi units. None had the clinical signs characteristic of pancreatitis.

Liver.—Four patients had increased bromsulphalein retention (BSP) and three patients had elevated serum glutamic oxaloacetic transaminase (SGOT) levels following freezing. There was, however, no other evidence of liver dysfunction.

Electrocardiographic Changes

Because the stomach and heart are close together, the heart is cooled more during gastric freezing than during systemic hypothermia. We studied the effects of this low temperature upon the myocardium by following the electrocardiographic changes during freezing.

In 53 patients electrocardiograms were made before, during and 24 hours after freezing. Six had abnormal tracings before freezing; in the remaining 47 the electrocardiogram was normal. Of those with abnormal tracings, one had a tracing typical of old anterior-wall infarction, one had a right bundle-branch block, and four had significant T-wave alterations.

Of these 53 patients, 17 (32%) had no electrocardiographic changes during freezing. Of the 36 (68%) who had abnormal tracings during the procedure, all but 1 subsequently reverted to the pre-freezing pattern.

The most frequent change noted during freezing was T-wave alterations indicating myocardial ischemia. Twenty-one patients (39.6%) had inverted T-waves in leads II, III, AVF, AVR, V5 and V6. Eleven (20.8%) had low T-waves in limb leads and V5 and V6. Two patients (3.8%) developed transient auricular fibrillation and one (1.9%) had ventricular premature beats.

Mortality

None of the patients died.

CLINICAL COURSE AFTER GASTRIC FREEZING

Of these 85 patients, 74 were followed for at least three years and ten months. By April 1, 1968, 31 (36.5%) had undergone vagotomy and drainage or partial gastrectomy for one or more of the following reasons: intractability (30), gastrointestinal bleeding (5), pyloric obstruction (4), gastric ulcer (4) and perforation (1). The time interval between freezing and subsequent operation ranged from 2 to 53 months, the average being 14.2 months.

Electrogastrograms—recordings of the changes in electrical potential in the gastric wall—were done on six patients who had gastric freezing and later were operated upon. These tracings, which generally show regular waves, showed a high incidence of disorder in the intrinsic electrical rhythm.[5]

The subsequent course of the other patients who have been followed for 3.8 to 4.9 years is as follows:

Complete or marked relief.—Seventeen patients (20.0%) have had no disability, no dietary restrictions and do not take medications regularly. Their symptoms, when present, have been very mild.

Moderate improvement.—Nine patients (10.6%) have had mild symptoms that were controlled by dietary restriction and occasional medication. These patients consider that gastric freezing has produced moderate improvement in their symptoms.

Slight or no improvement.—Seventeen patients (20.0%) have had persistent disabling symptoms, which are poorly controlled by diet and medication.

It is important to note that five patients deteriorated four years after freezing. In three, their symptoms increased in severity, and two required operation (vagotomy and

drainage in one, and subtotal gastrectomy in the other) after their symptoms had been markedly or moderately relieved for four years.

Relation of Clinical Response to Changes in Gastric Secretion

For the purpose of comparison, we have divided the patients into three groups:

Group A (worst)—31 patients who were operated upon—complete failure of the freezing.

Group B (best)—17 patients who had marked or complete relief of symptoms —freezing clinically successful.

Group C (total)—all 74 patients, regardless of clinical result.

We studied gastric secretion before, 12 hours after and 3 months after freezing, measuring volume, pH, and total acidity in mEq./l. as titrated to pH 7.0, of the eight-hour (overnight) secretion. We measured vagal response by the standard insulin-stimulation (Hollander) test and antral response by peptone-broth stimulation. The Hollander test was considered successfully performed only if the blood sugar fell to 50 mg./100 ml. or less. For comparison of different measurements we only included the results of analyses that had been successfully completed on the three successive occasions.

In comparing the gastric secretory changes, we first calculated the average value for volume, pH and total acidity for

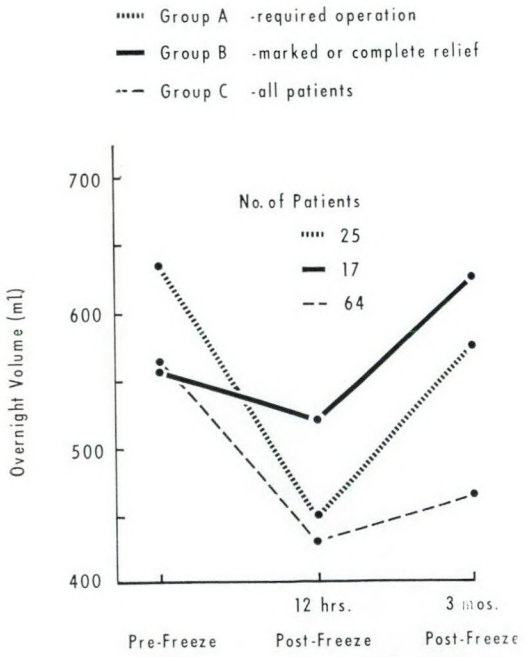

Fig. 1.—Eight-hour overnight volume. Only patients in whom a particular test was performed successfully on all three occasions are included in this and subsequent graphs.

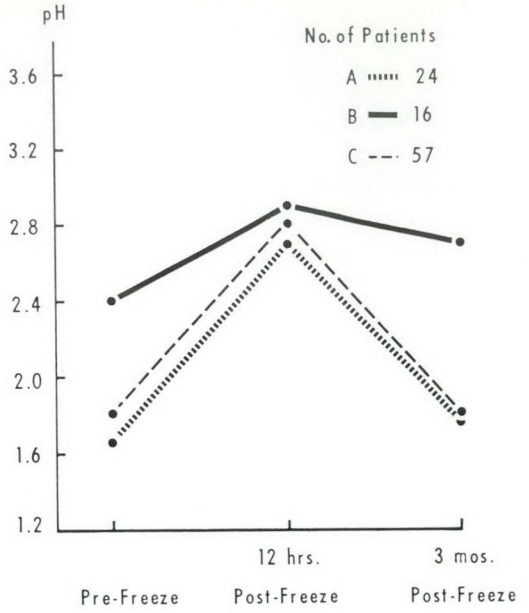

Fig. 2a

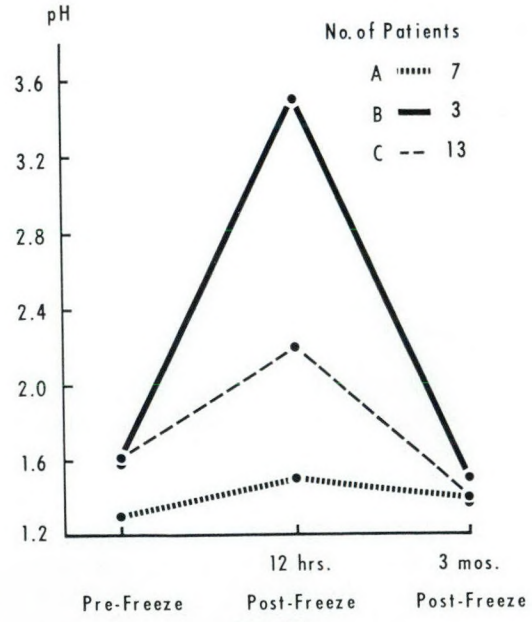

Fig. 2b

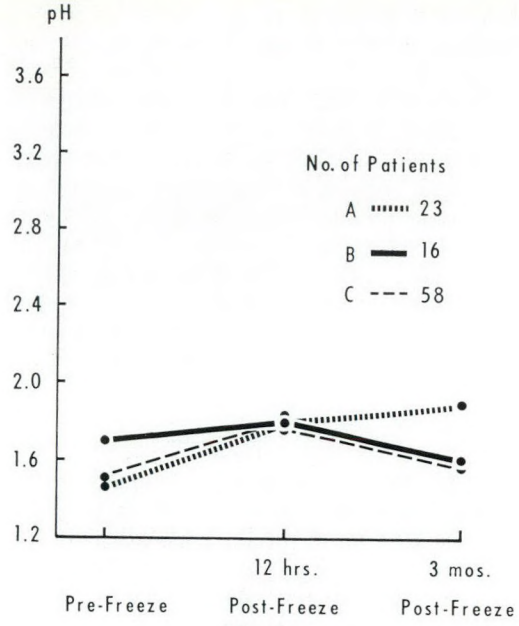

Fig. 2c

Fig. 2.—pH changes. (a) Eight-hour overnight secretion. (b) Insulin test. (c) Peptone test.

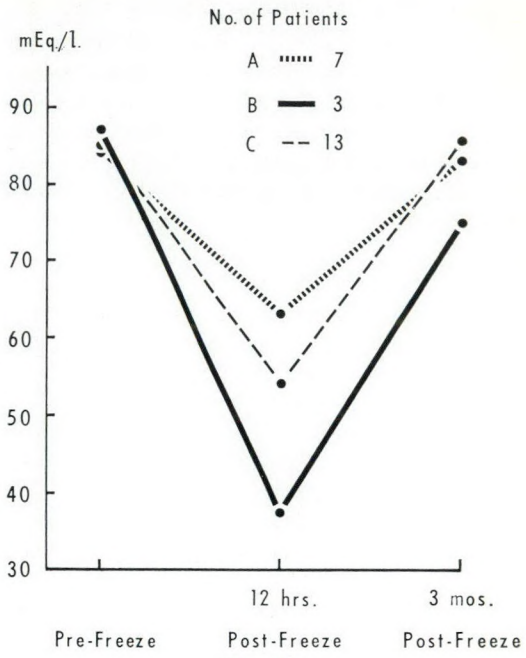

Fig. 3b

Groups A, B and C. The curve for each measurement was plotted for the pre-freezing, 12-hour post-freezing, and 3-month post-freezing values. Finally, to recognize any variation in the gastric secretory changes in the three groups with different clinical responses, the curves of each test of Groups A, B and C were superimposed (Figs. 1-3).

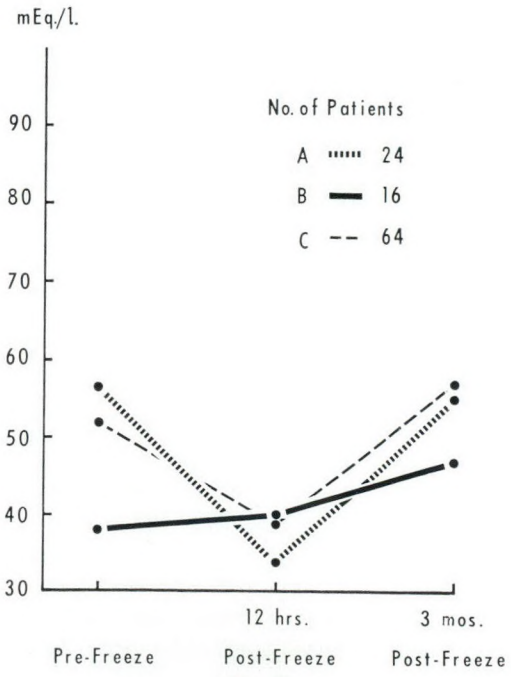

Fig. 3a

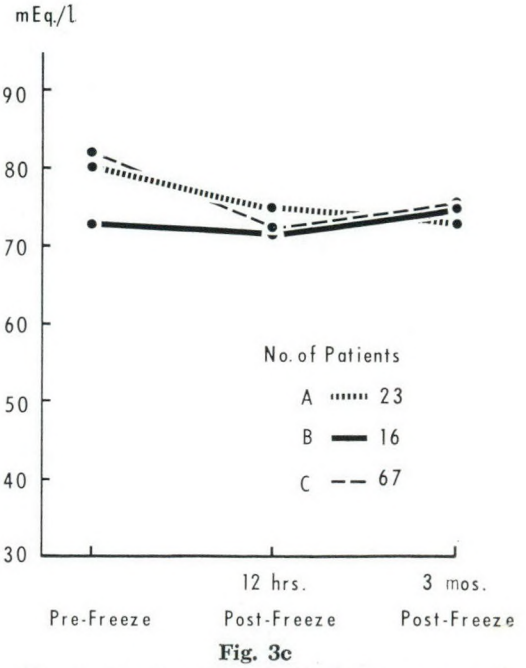

Fig. 3c

Fig. 3.—Total acidity. (a) Eight-hour overnight secretion. (b) Insulin test. (c) Peptone test.

Eight-Hour Overnight Volume (Fig. 1)

In all three groups, the overnight volume dropped in the 12 hours after freezing, and rose by 3 months. The increase in volume in Group C (all patients) was less than in the other groups. In Group B (good clinical result), the volume at three months was greater than it was before freezing.

pH Changes (Fig. 2)

Overnight secretion.—In all patients, the pH increased 12 hours after freezing and decreased to pre-freeze levels after 3 months. In Group B, the pH was higher before freezing and remained slightly higher than in the other groups three months after freezing.

Insulin test.—After insulin stimulation, the pH also increased 12 hours after freezing; however, 3 months later insulin stimulation gave about the same pH as before freezing. These changes were most marked in Group B.

Peptone test.—Very little change occurred in pH or volume after freezing in Groups A, B or C.

Total Acidity (Fig. 3)

Overnight secretion.—In patients in Groups A and C, the total acidity decreased 12 hours after freezing and rose to the pre-freeze level 3 months later. Patients in Group B had a slight increase 12 hours and 3 months after freezing.

Insulin test.—The total acidity fell at 12 hours and returned approximately to pre-freeze levels at 3 months. The fall in total acidity at 12 hours was most marked in Group B.

Peptone test.—No significant change in total acidity occurred in any of the groups after freezing.

Discussion

We found no significant correlation between clinical response and gastric secretory changes when volume, pH, total acidity of overnight secretion and peptone test were compared. The differences between the various groups, when present, were quite subtle and insignificant.

Insulin stimulation after freezing, however, caused a greater increase in pH and less total acidity in those patients who had marked relief (Group B), than in the patients in the other two groups. These patients may have had a greater inhibition of vagal activity after freezing which, though not sustained, gave prompt relief of ulcer symptoms. This marked decrease in acidity with insulin stimulation 12 hours after freezing was not seen in every patient who continued to have lasting relief; hence, insulin stimulation cannot be used to predict which patients will have permanent relief of symptoms after gastric freezing.

There is no logical explanation for the success of freezing in the 17 patients who had marked or complete relief of symptoms. However, the work of Artz, Kent and Winn[6] may shed some light on this improvement. They studied the effects of gastric freezing by interposing 20 cm. of ileum between the stomach and duodenum in 53 dogs—29 controls and 24 dogs who underwent gastric freezing. Twenty-seven of the 29 controls (93%) either died of duodenal ulcer complications or had an ulcer at autopsy three months later. In contrast, only 11 of 24 dogs (46%) subjected to gastric freezing three days before the ulcerogenic operation died of ulcer complications or had an ulcer at autopsy three months later.

This experiment casts doubt on the "witchcraft" concept of freezing. It may yet be shown that there is some real, although as yet unknown, mechanism through which our 17 patients obtained their satisfactory clinical response.

Changes in Gastrointestinal Radiographs

In 58 patients, gastrointestinal series were made before and three months after freezing; the radiographic changes noted are shown in Table I. All patients had evidence of duodenal ulceration—either a crater (the majority) or marked duodenal deformity before freezing. In no patient did the radiographs become normal after freezing. Thirty-one patients still had an ulcer crater three months after freezing; 22 (66.5%) were operated upon subsequently.

TABLE I.—Radiographic Changes Following Gastric Freezing

Radiographic finding before freezing	*Number of patients*	*Radiographic finding after freezing*	*Number of patients*	*Clinical results*
Crater seen	50	Crater persistent	29	18 patients required operation
		No crater, only deformity	21	3 patients required operation
No crater seen, severe duodenal deformity	8	Crater visualized	3	All required operation
		Deformity persistent	5	None required operation, varying degrees of improvement

Review of the Literature

In 1962, Owen Wangensteen *et al.*[2] introduced gastric freezing in an attempt to control intractable duodenal ulcer in patients who would otherwise have required an operation. This procedure, which was studied intensively at several medical centres, was used as definitive treatment for patients with an ulcer that did not respond to conservative therapy or had bled more than one month before freezing. The procedure was abandoned following clinical trial when it was found that the relief from duodenal ulcer was temporary in a large percentage of patients, and that there was no sustained alteration in gastric secretion.

Complications of Gastric Freezing

Gastrointestinal hemorrhage.—Of Manlove's[7] 55 patients, 5 had massive upper gastrointestinal bleeding after freezing, and Spellberg *et al.*[8] reported that 5 of 64 patients bled after freezing. According to Stephen Wangensteen *et al.*,[9] 6 of 31 patients bled from the upper gastrointestinal tract and 4 developed gastric ulcers following freezing. Owen Wangensteen *et al.*[10] reported that, after 810 episodes of gastric freezing in 605 patients, 3.8% had melena and 2.8% had gastric ulceration: none died.

Gastric ulcer.—A man described by Lisker, Stahlgren and Tumen[11] developed two gastric ulcers after freezing. These produced symptoms on the eleventh day after freezing and he was operated upon on the twenty-ninth day. They attributed the ulcers to: (1) the jet effect of the coolant causing a localized area of marked hypothermia; (2) excessively low temperature or overly prolonged freezing; (3) mucosal pressure by the balloon; (4) rapid decompression of the balloon without rewarming, causing adherent mucosa to rip off; and (5) stasis from decreased gastric motility. Barner *et al.*[12] encountered three "jet-thrust" gastric ulcers in 91 patients. Although one of these perforated, none of the patients died following freezing. Lippman, Morgenstern and Panish[13] were able to nullify the "jet-thrust" effect by modifying the infusion tip of the cooling apparatus with multiple pin-point exit holes to dissipate the intense pressure.

Holland and Morkovin[14] described a "post-freeze" gastric ulcer that adhered to spleen and diaphragm, and produced pneumonitis, pleurisy and effusion in the adjacent hemithorax. Another of their patients had lower esophageal perforation due to faulty balloon placement. In describing technique and precautions, Owen Wangensteen's group[4] emphasized that a trained physician must be in attendance and that the inflow temperature must be above −20° C.

In 1965, a panel[15] reviewed 934 freezing procedures done on 669 patients; 26 developed a gastric ulcer, 3 with perforation. Forty patients bled, two developed a fistula, one patient sustained a gastric rupture and one an esophageal leak. No patients died.

Animal studies.—The effects of freezing have been studied in animals. Shingleton *et al.*[16] reported that, after freezing in 32 dogs, 8 developed a gastric ulcer. McIlrath and Hallenbeck[17] noted that, as freezing is performed, the stomach does not actually freeze. When the stomachs of dogs or pigs are frozen solidly, necrosis follows. In dogs, Stephen Wangensteen *et al.*[18] noted that, after one hour, the mucosa appeared

to be extensively frozen and had a shell-like lining of ice. Twelve hours after freezing, the stomach often showed mucosal hemorrhage and, on the second or third day, the mucosa showed multiple superficial erosions. With two hours of freezing, dogs developed free perforations.

After freezing, dogs showed gross gastric injury which consisted of: (1) full-thickness destruction of gastric wall with gangrene; (2) full-thickness injury of gastric wall with extensive mucosal ulceration; (3) injury of the mucosal layer with bullous edema and superficial erosion, with or without small areas of ulceration; (4) minor superficial mucosal erosion; (5) minor ecchymosis of the mucosa without erosion.

Histological studies were done. When a dog's stomach is actually frozen (to a temperature of 0° C. or less, recorded directly) and sacrificed 24 hours later, the findings were similar to those seen in frostbite.[3, 19] For the first two to three days, there are mucosal edema and hemorrhages, and intracellular ice crystals cause vacuolation of the parietal cells. There is also chief-cell injury (shown by diminished digestive power of gastric juice) and some swelling is seen in mucous cells. The submucosal vessels contain microthrombi. The Auerbach's plexus is injured, but there are no dramatic changes in the muscle and other deep tissues. By the fourth day, the hyperemia and edema subside.

According to Kolig and Marx,[19] hemorrhage and gastric ulceration seen in occasional patients indicate that, in the gastric wall, subzero temperature had been reached clinically. Lennard-Jones *et al.*[20] showed that, in dogs at open operation, the mucosa was damaged by direct application for 15 minutes of temperatures below −3.5° C. but not above −3.5° C. He, and Marx and Kolig,[21] found that muscle was more resistant to cold than mucosa, and that acid-secreting mucosa was more sensitive to cold damage than antral or esophageal mucosa.

Benjamin *et al.*[22] believe that the superficial blebs and localized mucosal necrosis are due to shunting of blood from the stomach, and to intravascular clotting in the submucosa. Atik *et al.*[23] found that giving intravenous low molecular weight dextran to dogs during freezing reduced the number and size of gastric ulcers. The mucosa was protected because sludging was prevented, the arterial blood flow declined less and hence the temperature of the gastric wall declined less. Owen Wangensteen's group[10] also found that low molecular weight dextran and ingestion of fluids buffered with sodium bicarbonate minimized mucosal change.

Results of direct examination in patients.—In human beings, this examination was limited to gastroscopy. In an unstated number of patients, Owens, Brown and Sullivan[24] observed intense erythema, diffuse edema and bulla formation, which lasted from 24 to 48 hours, and, in two patients, Berg, Geisel and Necheles[25] noted occasional mucosal hemorrhage and necrosis associated with moderate hemorrhage. According to Spellberg *et al.*,[8] the changes seen 24 to 48 hours after freezing were compatible with superficial gastritis, and were most marked in the body and fundus of the stomach. When the stomach was examined several months later, these findings had disappeared. Nabseth *et al.*,[26] using a Rubin biopsy tube after freezing, found hyperemia and hemorrhage microscopically in five of eight patients; some patients had necrosis of the mucous glands.

According to Allcock *et al.*,[27] the edema produced by gastric freezing usually persists for 7 to 10 days, and during this period precludes any definitive operation. Before this method of therapy is used, therefore, the physician must be certain that the patient will not require an operation, especially a patient who has bled from the gastrointestinal tract.

Freezing was most useful in the management of stomal ulcer, intractable duodenal ulcer, or duodenal ulcer which had bled or perforated more than one month previously.[28] It was contraindicated in gastric ulcer or pyloric obstruction; three of Owen Wangensteen's patients who had gastric freezing for duodenal ulcer with pyloric obstruction required subsequent gastric resection.[3]

Miscellaneous complications.—In Stephen Wangensteen's patients,[9] the serum amylase did not rise with gastric freezing but,

as described earlier, became elevated in three of our patients. One patient in Hitchcock's series[29] developed pancreatitis after freezing.

Hershfield, Lind and Hildes[30] found that gastric hypothermia had no adverse effect on BSP clearance in normal subjects. As noted earlier in this report, a few of our patients had definite elevations of BSP and SGOT after freezing.

One patient of Spellberg's[8] and two of Garcia's[31] went into shock during gastric freezing but responded to vasopressors. Two patients developed pleural effusion and one thrombocytopenia purpura following gastric freezing.[32]

Electrocardiographic alterations. — After freezing, ECG changes are reported in from 77%[31] to more than 90%[33-35] of patients in contrast to Wangensteen's early report that no ECG changes were noted when six patients were monitored.[3] The most frequent change noted[31, 33] was a decrease in voltage or inversion of T-waves in leads II, III and AVF, prolongation of electrical systole, and tachycardia. Left axis deviation of QRS and extrasystoles were less common.[36] Fifteen per cent of patients had a shift in the axis towards horizontal.[33] These alterations disappeared slowly in the first 10 minutes after freezing was finished, except in one patient who had atherosclerotic heart disease.[36] One patient with an old myocardial infarction had no electrocardiographic changes with freezing.[36] Rose and Harrell[33] found that 3 of 26 patients developed atrial fibrillation, which reverted to normal after 24 hours. More severe changes were seen in patients in whom the outflow temperature was lower.[31]

Gastric cooling.—Gastric "cooling" was introduced by Owen Wangensteen *et al.*[37] in 1958 before "freezing" was used for the elective treatment of intractable duodenal ulcer. He used cooling in the emergency treatment of acute, massive, upper gastrointestinal hemorrhage. Wangensteen *et al.*[38] found that intragastric cooling reduced peptic activity, gastric secretion and gastric blood flow. Inflow temperatures of 0° to 10° C. were continued for several hours to several days.[39]

Generally, it was most successful in bleeding from duodenal ulcer, gastric erosion and esophageal varices, and was less effective in bleeding gastric ulcer, steroid ulcer and hemorrhagic gastritis. It was also used in coagulation defects (two hemophiliacs and one vitamin-K deficiency).[40]

Cooling stopped the bleeding in 37% to 88% of patients[39, 41-44] but it recurred six hours to two weeks later in 50% to 71% of those controlled initially.[42, 43] Gastric cooling has been used as a temporary or ancillary measure in acutely ill patients, allowing the surgeon time to put the patient in optimal condition for operation.[39, 41, 45, 46]

McFarland and Gow[47] found that gastric cooling had no advantage over the conservative management of upper gastrointestinal bleeding. Rodgers, Older and Stabler[42] warned that this procedure must be used cautiously in those with pulmonary diseases, because death from pneumonia was frequent.

Cali, Glaubitz and Crampton[48] described a patient who had to have a laparotomy for persistent bleeding after he had had 102 hours of cooling. In addition to a gastric ulcer, the man had a large area of full-thickness gangrene along the greater curvature, which was the result of prolonged hypothermia and the pressure of the balloon against the mucosa. During gastric cooling for uncontrollable hemorrhage, Perey, Helle and MacLean[49] reported that the balloon became disconnected from the tube and alcohol escaped, producing acute alcoholic intoxication, which was successfully managed by hemodialysis.

We believe that gastric cooling is still useful in patients who are very poor risks for operation or have coagulation defects, or when operation must be deferred in the actively bleeding patient for any reason.

Long-Term Results

Artz, Kent and Winn[6] showed that gastric freezing had a definite effect on ulcer disease in dogs that had 20 cm. of ileum interposed between stomach and duodenum. When compared to "non-frozen controls", freezing gave significant protection from ulceration during the early months after the ulcerogenic operation.

In nearly all patients ulcer pain disappears immediately after freezing.[3, 9] Six

weeks later, 75% of patients still have no symptoms.[15] Six months later, asymptomatic patients vary from 12%,[15] 38%,[9] 39.8%,[29] 50%[8] to 60%;[15] and one year later, 21%,[32] 37%,[9] to 44%[12] are free of symptoms.

As noted earlier, 20.0% of our patients have had complete or marked relief after a minimal follow-up of 3.8 years. In the literature, from 23%[12] to 33%[32, 35] of patients after freezing have required operation to control their ulcer. With our longer follow-up, 36.5% of our patients eventually required surgical intervention.

With such a high recurrence rate, the procedure does not appear to alter significantly the eventual course of the disease.[17, 25] However, it may be useful in intractable duodenal ulcer if the patient is a very poor risk for operation.[7]

Bernstein[50, 51] pointed out that gastric freezing is valuable in postgastrectomy stomal ulcer. All but 9 of his 33 patients had relief after one "freeze". Eight were controlled by refreezing and only one patient required operation to control his stomal ulcer. However, his longest follow-up was too short to enable him to draw definite conclusions.

Rutledge and Bronwell[52] emphasized that the surgeon must demonstrate the duodenal ulcer before freezing the stomach. They described a patient with ulcer symptoms and radiologic evidence of duodenal deformity who was treated by gastric freezing. Symptoms persisted and, at operation two months later, the patient had a carcinoma of the antrum and pylorus.

Secretory Studies

Immediately after freezing there is a significant reduction in the volume and free hydrochloric acid produced during overnight secretion and in response to insulin stimulation.[1, 3, 9, 34, 53] However, these values return to the pre-freeze level in three weeks[53] to a few months.[1, 24] Six months later, the gastric acidity tests are similar to the pretreatment values.[8, 9] Of Scott's[32] patients, however, 18% still had reduction in free acid one year later.

No statistical correlation was found between post-freeze gastric secretory values and relief of symptoms.[16, 29, 32, 35] However, as noted earlier with our patients, a lower acidity in insulin-stimulation tests at 12 hours after freezing has been found to correlate with the group which, on follow-up, continues to be asymptomatic.

Mechanism of Pain Relief

The mechanism of pain relief, though not definitely known, has been attributed to temporary sympathetic denervation, decreased motility, destruction of ganglion cells in Auerbach's plexus, temporary elimination of hydrochloric acid and pepsin, and increased mucous secretion.[15, 28] However, in rats, decreased mucin secretion has been found after freezing.[54]

Using cinefluoroscopy, Owens *et al.*[15, 24] showed that gastric motility had returned to normal one day after freezing, but was decreased again the next day; it was normal again six weeks later.

That pyloric emptying is not altered, despite depression of gastric secretory response to insulin, suggests that the intrinsic nerve plexuses were damaged; in effect, freezing produces a selective vagotomy.[3] Klotz[15] attributed the effect of freezing to a temporary incomplete vagotomy because he found no evidence of decrease in gastrin secretion by the antrum. Also, histamine studies do not suggest that cells become unresponsive after freezing. Using augmented histamine tests, Nabseth *et al.*[26] did not find that the gastric acidity was depressed after freezing. By both direct vagal stimulation and sham feeding in dogs after freezing, Klotz and Christodoulopoulos[55] found that the gastric vagal response was markedly decreased but the histamine response was not altered and no significant changes were seen on mucosal biopsy.

Freezing has a different effect from irradiation. Irradiation of the fundus and body of the stomach in the form of 1650 R. ortho voltage or 2000 R. ^{60}Co in 10 divided doses decreases the volume and free acid in basal and stimulated fractions observed three weeks after irradiation; this change is present three months later.[28, 53, 56] Irradiation directly damages the gastric mucosa and destroys parietal and chief cells.[28, 53]

The psychological effect of gastric freezing probably contributes to the relief of symptoms.[8, 29] In a double-blind study in which eight patients had standard Wangensteen freezing and eight controls underwent sham freezing, Zikria *et al.*[57] found that both groups were asymptomatic at the end of a week. Only the freeze group had a significant reduction in acidity at one week after freezing; there was no difference in acidity at three and six months in the two groups. The recurrence of symptoms was higher in the group that had undergone freezing. Apparently, psychogenic influences are extremely important in this dramatic and highly publicized procedure.[28]

Gastric secretory changes and clinical results after multiple gastric freezing show that the alterations are temporary, as with the first freeze.[58] According to Karacadag and Klots,[58] repeat freezing duplicates the temporary selective vagotomy achieved in the first freeze, without enhancing the therapeutic effect.

Summary and Conclusions

A long-term study has been made of 85 patients who enderwent gastric freezing for a duodenal ulcer that otherwise would have required an operation.

All these patients were followed for 3.8 to 4.9 years; 11 were lost to follow-up. Subsequently, 31 patients required an operation for unrelieved or recurrent symptoms. Of the remainder, 17 have had complete or marked relief, 9 have had only moderate improvement, and 17 have had slight or no improvement.

This procedure is associated with several complications. Upper gastrointestinal bleeding occurred after freezing in five patients and gastric ulcer developed in three. There were no deaths.

Except in insulin-stimulation tests, we found no definite correlation between gastric secretory changes and the subsequent clinical results. In patients who continue to have marked improvement, the Hollander test showed slightly less vagal response during the 12-hour post-freeze analysis only. However, this was not a constant finding in patients who achieved lasting relief.

The literature on gastric freezing has been reviewed. Although the late results of gastric freezing are disappointing and, like other workers, we have abandoned the procedure, 17 (20%) of our patients, on long-term follow-up, continue to have complete or marked relief of symptoms.

In spite of low morbidity and mortality, the high incidence of ulcer recurrence after gastric freezing precludes its consideration in the treatment of duodenal ulcer.

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References

1. McIntyre, J. A. and Brindis, R.: Gastric secretory changes resulting from gastric hypothermia, *Brit. J. Surg.*, **53:** 439, 1966.
2. Wangensteen, O. H. *et al.*: Achieving "physiological gastrectomy" by gastric freezing: a preliminary report of an experimental and clinical study, *J. A. M. A.*, **180:** 439, 1962.
3. Wangensteen, O. H. *et al.*: Can physiological gastrectomy be achieved by gastric freezing?, *Ann. Surg.*, **156:** 579, 1962.
4. Peter, E. T. *et al.*: Technique of gastric freezing in the treatment of duodenal ulcer, *J. A. M. A.*, **181:** 760, 1962.
5. McIntyre, J. A. and Deitel, M.: Unpublished data.
6. Artz, C. P., Kent, J. J. and Winn, J. R.: Effect of gastric freezing on an ulcerogenic preparation in dogs, *Amer. Surg.*, **32:** 434, 1966.
7. Manlove, C. H., Jr.: Complications after gastric hypothermia, *Amer. J. Surg.*, **109:** 185, 1965.
8. Spellberg, M. A. *et al.*: Experience with gastric freezing in the treatment of peptic ulcer, *Amer. J. Gastroent.*, **42:** 249, 1964.
9. Wangensteen, S. L. *et al.*: Gastric "freezing". An initial clinical study, *Amer. J. Med.*, **38:** 31, 1965.
10. Wangensteen, O. H. *et al.*: Gastric freezing for duodenal ulcer: potentiation with vasopressin, *Ann. Intern. Med.*, **61:** 636, 1964.
11. Lisker, S. A., Stahlgren, L. H. and Tumen, H. J.: Development of gastric ulcer as a complication of gastric freezing, *New Eng. J. Med.*, **270:** 1001, 1964.
12. Barner, H. B. *et al.*: Clinical gastric 'freezing', *Amer. J. Dig. Dis.*, **11:** 625, 1966.
13. Lippman, H. N., Morgenstern, L. and Panish, J. F.: Etiology and prevention of gross mucosal lesions seen after gastric freezing, *J. A. M. A.*, **187:** 265, 1964.
14. Holland, R. H. and Morkovin, D.: Complications of gastric freezing, *J. A. M. A.*, **186:** 863, 1963.
15. Hightower, N. C., Jr. *et al.*: Panel discus-

sion on gastric freezing, *Amer. J. Gastroent.*, **43:** 523, 1965.

16. Shingleton, W. W. *et al.*: Comparison of instrumentation for experimental and clinical gastric freezing, *Ann. Surg.*, **162:** 1053, 1965.
17. McIlrath, D. C. and Hallenbeck, G. A.: Review of gastric freezing, *J. A. M. A.*, **190:** 715, 1964.
18. Wangensteen, S. L. *et al.*: Gastric "freezing", *Arch. Surg. (Chicago)*, **90:** 286, 1965.
19. Kolig, G. and Marx, F. W., Jr.: Gastric freezing or gastric frostbite?, *Arch. Surg. (Chicago)*, **90:** 182, 1965.
20. Lennard-Jones, J. E. *et al.*: Observations on the temperature causing mucosal damage in the dog during gastric freezing, *Gut*, **7:** 535, 1966.
21. Marx, F. W., Jr. and Kolig, G.: The hazards of gastric freezing, *Amer. J. Dig. Dis.*, **11:** 272, 1966.
22. Benjamin, H. B. *et al.*: Effects of hypothermia and hyperthermia on the vascular supply of the stomach, *Surg. Gynec. Obstet.*, **121:** 1289, 1965.
23. Atik, M. *et al.*: Ulceration from gastric freezing: the protective effect of low molecular weight dextran, *J. A. M. A.*, **191:** 386, 1965.
24. Owens, F. J., Brown, C. H. and Sullivan, B. H., Jr.: Gastric "freezing" for peptic ulcer: evaluation by gastroscopic, cinefluoroscopic, gastric secretory and gastric biopsy studies, *Amer. J. Dig. Dis.*, **10:** 514, 1965.
25. Berg, M., Geisel, A. and Necheles, H.: The treatment of duodenal ulcer by gastric freezing: clinical and physiological observations, *Amer. J. Gastroent.*, **42:** 593, 1964.
26. Nabseth, D. C. *et al.*: Gastric freezing for peptic ulceration in man—a note of caution: the early effects on gastric histology and acidity, *New Eng. J. Med.*, **270:** 603, 1964.
27. Allcock, E. A. *et al.*: Structural changes following gastric freezing, *Surgery*, **53:** 764, 1963.
28. Kirsner, J. B.: Facts and fallacies of current medical therapy for uncomplicated duodenal ulcer, *J. A. M. A.*, **187:** 423, 1964.
29. Hitchcock, C. R., Bitter, J. E. and Sutherland, R. D.: Clinical evaluation of gastric freezing for duodenal ulcer, *J. A. M. A.*, **188:** 409, 1964.
30. Hershfield, N. B., Lind, J. F. and Hildes, J. A.: Gastric cooling and hepatic function, *Canad. J. Surg.*, **8:** 153, 1965.
31. Garcia, A. R., Langsjoen, P. H. and Hightower, N. C., Jr.: Electrocardiographic changes during gastric hypothermia, *Dis. Chest*, **48:** 497, 1965.
32. Scott, H. W. *et al.*: An evaluation of the long term results of gastric freezing for duodenal ulcer, *Surg. Gynec. Obstet.*, **121:** 723, 1965.
33. Rose, H. and Harrell, R.: Cardiovascular effects of gastric hypothermia, *Southern Med. J.*, **60:** 152, 1967.
34. Heineken, T. S. *et al.*: Gastric freezing: preliminary report on ten cases, *Amer. J. Gastroent.*, **39:** 648, 1963.
35. Harrell, W. R. *et al.*: Gastric hypothermia for duodenal ulcer: a long term controlled study, *J. A. M. A.*, **200:** 290, 1967.
36. Escudero, J. *et al.*: Electrocardiographic changes in gastric freezing, *Amer. Heart J.*, **71:** 29, 1966.
37. Wangensteen, O. H. *et al.*: Depression of gastric secretion and digestion by gastric hypothermia: its clinical use in massive hematemesis, *Surgery*, **44:** 265, 1958.
38. Wangensteen, O. H. *et al.*: Studies of local gastric cooling as related to peptic ulcer, *Ann. Surg.*, **150:** 346, 1959.
39. Karacadag, S. and Klotz, A. P.: Gastric hypothermia in the management of massive upper gastrointestinal bleeding, *Amer. J. Gastroent.*, **42:** 505, 1964.
40. Hershfield, N. B., Lind, J. F. and Hildes, J. A.: Gastric cooling for massive upper gastrointestinal bleeding, *Canad. Med. Ass. J.*, **95:** 905, 1966.
41. Schaller, R. T., Jr. *et al.*: Gastric hypothermia for massive upper gastrointestinal hemorrhage: experience with 24 patients and a review of literature, *Arch. Surg. (Chicago)*, **92:** 707, 1966.
42. Rodgers, J. B., Older, T. M. and Stabler, E. V.: Gastric hypothermia: a critical evaluation of its use in massive upper gastrointestinal bleeding, *Ann. Surg.*, **163:** 367, 1966.
43. Gross, A. *et al.*: Gastric cooling—a clinical study, *Canad. J. Surg.*, **8:** 369, 1965.
44. Turner, G. R. *et al.*: Local gastric hypothermia in management of massive upper gastrointestinal hemorrhage, *Surgery*, **53:** 609, 1963.
45. Dollinger, M. R. and Morgan, L. R.: Local cooling in the management of acute upper gastrointestinal bleeding, *Amer. J. Surg.*, **111:** 651, 1966.
46. Alichniewicz, A. *et al.*: Intragastric cooling in temporary treatment of acute hemorrhage from the upper gastrointestinal tract, *Bull. Pol. Med. Sci. Hist.*, **10:** 120, 1967.
47. McFarland, J. B. and Gow, J. G.: Trial of gastric cooling for haematemesis, *Brit. Med. J.*, **3:** 831, 1967.
48. Cali, J. R., Glaubitz, J. P. and Crampton, R. S.: Gastric necrosis due to prolonged local gastric hypothermia, *J. A. M. A.*, **191:** 858, 1965.
49. Perey, B. J. F., Helle, S. J. and MacLean, L. D.: Acute alcoholic poisoning: a complication of gastric hypothermia, *Canad. J. Surg.*, **8:** 194, 1965.
50. Bernstein, E. F. *et al.*: Treatment of postgastrectomy stomal ulcer by gastric freezing, *Arch. Surg. (Chicago)*, **87:** 13, 1963.
51. Bernstein, E. F. *et al.*: Interim report on results of gastric freezing for peptic ulcer: present indications, limitations, and clinical achievement, *J. A. M. A.*, **187:** 436, 1964.
52. Rutledge, R. and Bronwell, A. W.: Incidental freezing of gastric carcinoma, *Texas J. Med.*, **62:** 39, 1966.
53. Karacadag, S. and Klotz, A. P.: Secretory comparison of gastric freezing and radiation therapy, *Amer. J. Gastroent.*, **42:** 604, 1964.
54. Buchan, R. and Clark, C. G.: Gastric function and intestinal absorption after gastric freezing in the rat, *J. Surg. Res.*, **6:** 337, 1966.
55. Klotz, A. P. and Christodoulopoulos, J. B.: The secretory physiology of gastric freezing, *Amer. J. Dig. Dis.*, **10:** 31, 1965.

56. Levin, E. *et al.*: Observations on the value of gastric irradiation in the treatment of duodenal ulcer, *Gastroenterology*, **32:** 42, 1957.
57. Zikria, B. A. *et al.*: Gastric "freezing" — a clinical double-blind study, *Amer. J. Gastroent.*, **47:** 208, 1967.
58. Karacadag, S. and Klotz, A. P.: Multiple gastric freezing, *Amer. J. Dig. Dis.*, **10:** 116, 1965.

Résumé

Soixante-quatorze des 85 patients ayant subi des congélations gastriques entre mai 1963 et juin 1964 au St. Joseph's Hospital de Toronto ont été revus; tous avaient reçu ce traitement pour un ulcère duodénal.

Voici un bref aperçu des complications de la congélation: hémorragie gastro-intestinale, cinq cas; ulcère gastrique, trois cas; collapsus cardio-vasculaire, deux cas. Trois patients avaient une amylase sanguine élevée mais sans preuves cliniques de pancréatite. Quatre malades avaient une rétention accrue de bromosulfonephtaléine (BSP) et trois avaient des taux élevés de transaminase sérique glutano-oxalacétique (SGOT) mais aucun autre signe de lésion hépatique. Les changements trouvés dans les électrocardiogrammes sont esquissés.

Des 74 malades qui ont été suivis, 31 ont dû être opérés parce que les symptômes d'ulcère avaient ré-apparu, 17 n'ont pas été—ou fort peu—améliorés, 9 ont été modérément améliorés et 17 n'avaient virtuellement plus aucun symptôme. Aucune corrélation n'a pu être établie entre l'amélioration clinique et les modifications de la sécrétion gastrique.

La littérature est passée en revue aux points de vue suivants: les complications de la congélation gastrique, hémorragie et ulcère gastrique; les expériences faites sur les animaux; les examens gastroscopiques chez l'homme; les modifications de l'électrocardiogramme; les résultats à long terme; les études sur la sécrétion et sur le mécanisme du soulagement de la douleur.

Les auteurs, en conclusion, déclarent que l'ulcère duodénal ne doit pas être soigné par la congélation gastrique.

MANAGEMENT OF GASTRIC AND DUODENAL ULCERS

The current views in Germany on the operative management of gastric and duodenal ulcers are discussed. It should be recalled that many large series with excellent end results and low mortality rates were reported in the first few decades of this century and that variations of the standard subtotal gastric resections, such as the Billroth II method and the Hoffmeister modification, have been devised and used clinically.

Consequently, a standard two-thirds subtotal gastric resection including the lesser curvature to the left gastric artery is still, in the author's opinion, the correct or definitive procedure for gastric and duodenal ulcers. He anticipates that this operation can be performed electively with a mortality rate of 0.5% to 3% and with good results in about 90%. The familiar early and late complications, and the familiar operative techniques designed mostly to prevent both the early and late dumping syndrome are discussed.

The author points out that vagotomy is limited to duodenal ulcers. He recognizes that a drainage procedure must be carried out because of gastric atony, and that patients with hypersecretion are the best subjects for vagotomy. Again, the anticipated mortality rate is low, that is, from 0.3% to 1%, and the degree of healing is from 85% to 95%.

Vagotomy is, however, not without its drawbacks, and these are discussed extensively. Perhaps the most serious of these is the high rate of relapse and recurrence, in some reported series being as high as 28%. Diarrhea is a well-known post-vagotomy complaint, and the usual 10% incidence rate was noted in the author's experience. Diarrhea combined with atony of the gallbladder, gallstones, and cholecystitis is referred to as the extragastric vagotomy syndrome.

For recurrent ulcer, the preferred treatment is further resection or else standard classic gastric resection if the previous operation has been a vagotomy and drainage procedure. When there is recurrence, the Zollinger-Ellison syndrome with or without multiple endocrine tumours must be considered.

The usual surgical methods of closing perforated ulcers are mentioned briefly, as are combined procedures, without any particular recommendation. Although there is a place for conservative treatment of perforated ulcers, early diagnostic laparotomy is favoured for bleeding. When no source of bleeding can be observed, so-called blind gastric resection is advised.—Kunz, H.: Der heutige Stand der operativen Behandlung des Magen-Zwoelffingerdarm-Geschwuers, *Munschen. Med. Wschr.*, **110:** 1689, 1968.

CASE REPORTS

REPLANTATION OF A COMPLETELY SEVERED ARM FOLLOWED BY REAMPUTATION BECAUSE OF FAILURE OF INNERVATION*

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REPORTS of successful replantation of completely severed extremities are becoming more numerous; however, many of these extremities do not function. In contrast to limbs that are cleanly severed, limbs that have been avulsed can rarely, if ever, be implanted because the nerves are irreparably damaged. Our experience confirms this. The present case report illustrates many of the technical difficulties encountered during replantation or during the postoperative period, and emphasizes some of the factors responsible for the failure of function after replantation.

CASE REPORT

P.S., a 58-year-old workman, was admitted to the emergency ward of St. Michael's Hospital, Toronto on December 13, 1966 following an industrial accident. His right arm had been avulsed completely two inches above the elbow. He also had minor lacerations of the right ear and left leg. Shortly after he arrived, the severed arm was brought to the hospital.

A robust man, in excellent physical condition, he had no evidence of internal injuries. His blood pressure was 130/90 mm. Hg; his pulse rate was 80/min. He was in no acute distress. Bleeding from the stump had stopped and he was not in shock.

While he was being treated, a second team attended to the severed arm. The arm, which was covered in coal dust, was cleaned and immersed in ice. The brachial artery and vein were cannulated, and continuous perfusion was started with Ringer's lactate to which heparin and penicillin had been added.

Approximately 1½ hours after the injury, the patient was taken to the operating room for replantation. The stump and the severed arm were thoroughly cleaned. The bone ends were rongeured but not significantly shortened. The two ends were fixed in apposition by a Küntscher nail introduced through the olecranon fossa. The gaps in the brachial artery and in one deep vein were bridged with saphenous vein grafts. We could restore the continuity of only one other vein—one in the subcutaneous plane. After the circulation was re-established, the radial pulse was bounding. The arterial circulation of the limb was adequate but the venous circulation was precarious. We attempted to anastomose the nerves but never found the ulnar nerve; the median nerve had been avulsed from the forearm and only the radial nerve could be joined. The muscle groups were sutured but there was insufficient skin to close the wound completely and a gap was left posteriorly. The wound was dressed in a bulky dressing and a posterior plaster splint applied. At the end of a five-hour procedure, the patient had a bounding radial pulse but the capillary circulation was sluggish and venous return inadequate. The arm was swollen and slightly cyanotic. The patient received 10,000 c.c. of balanced electrolyte solution and 3000 c.c. blood during the procedure.

After the operation, he was taken to the intensive care unit where the arm was elevated on pillows. He received 500 c.c. of low molecular weight dextran daily, heparin 75 to 100 mg. every six hours according to the clotting time, penicillin one million units every four hours, and sodium oxacillin 500 mg. every six hours.

Transfusions were given volume for volume as measured by estimated loss, the clinical condition of the patient, and daily hemoglobin and hematocrit measurements. The leukocyte

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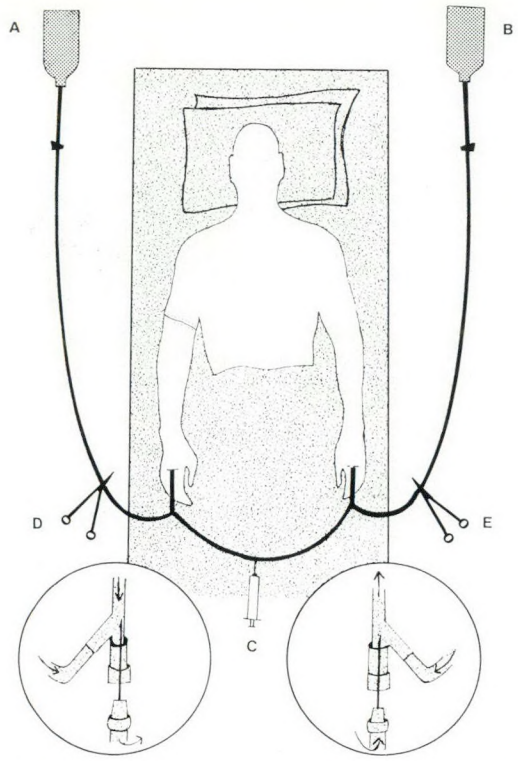

Fig. 1.—Diagram of the venous shunt. Blood flow is indicated by arrows in the inserts. Reservoir bottles, A and B, containing electrolyte solutions are used to irrigate the tubing system. Tubing other than that in the mid-section is clamped at all times (D and E), and opened only to flush the tubing between the two extremities. Heparin (C) is used as necessary. The pressure gradient between the two extremities is sufficiently high to shunt large quantities of blood.

count and clotting times were estimated daily. Several blood cultures were taken from the affected arm and from another systemic vein, and urine was sent to the laboratory for red cell and myoglobin estimations.

During the first day the patient's general condition remained good; the dressing was completely changed and the arm was inspected. The forearm was warm; the radial pulse was strong; however, the arm was markedly swollen, slightly cyanotic and the capillary circulation was poor. The venous drainage was considered to be totally inadequate. To facilitate venous decompression, a cut-down was made on the dorsum of the right hand (affected side) and attached to an open collection bottle. The blood was replaced volume for volume. In 24 hours, 5600 c.c. of blood was drained off. During this period the patient went into shock and the drainage system had to be clamped. It was clear at this point that decompression of the arm in this manner would jeopardize the patient's life; on the other hand, if the blood was not diverted the arm would be lost. The next day the patient's general condition had improved, but the arm had deteriorated; the radial pulse was only faintly perceptible.

Finally, we created a shunt from the previously cannulated vein of the right hand to the veins of the left hand. The venous pressure in the right arm was high enough so that, with care and continuous supervision, enough blood was shunted across to decompress the congested limb. After the shunt had been working for a time, the appearance of the arm markedly improved, swelling decreased and circulation now seemed adequate (Fig. 1).

The patient remained in the intensive care unit for 25 days. During this period he received 42 units of blood, chiefly because of blood loss. His hemoglobin finally stabilized at 13 g./100 ml.

A hematoma that had developed under the posterior skin flap of the proximal stump became infected but the infection subsided after the blood clot was evacuated. An area of pressure necrosis also developed on the medial side of the elbow.

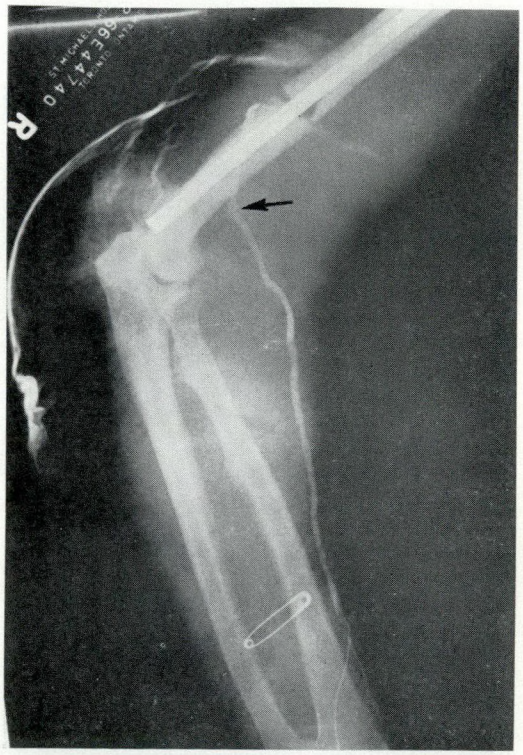

Fig. 2.—Venogram at 16 days showing the patent superficial vein.

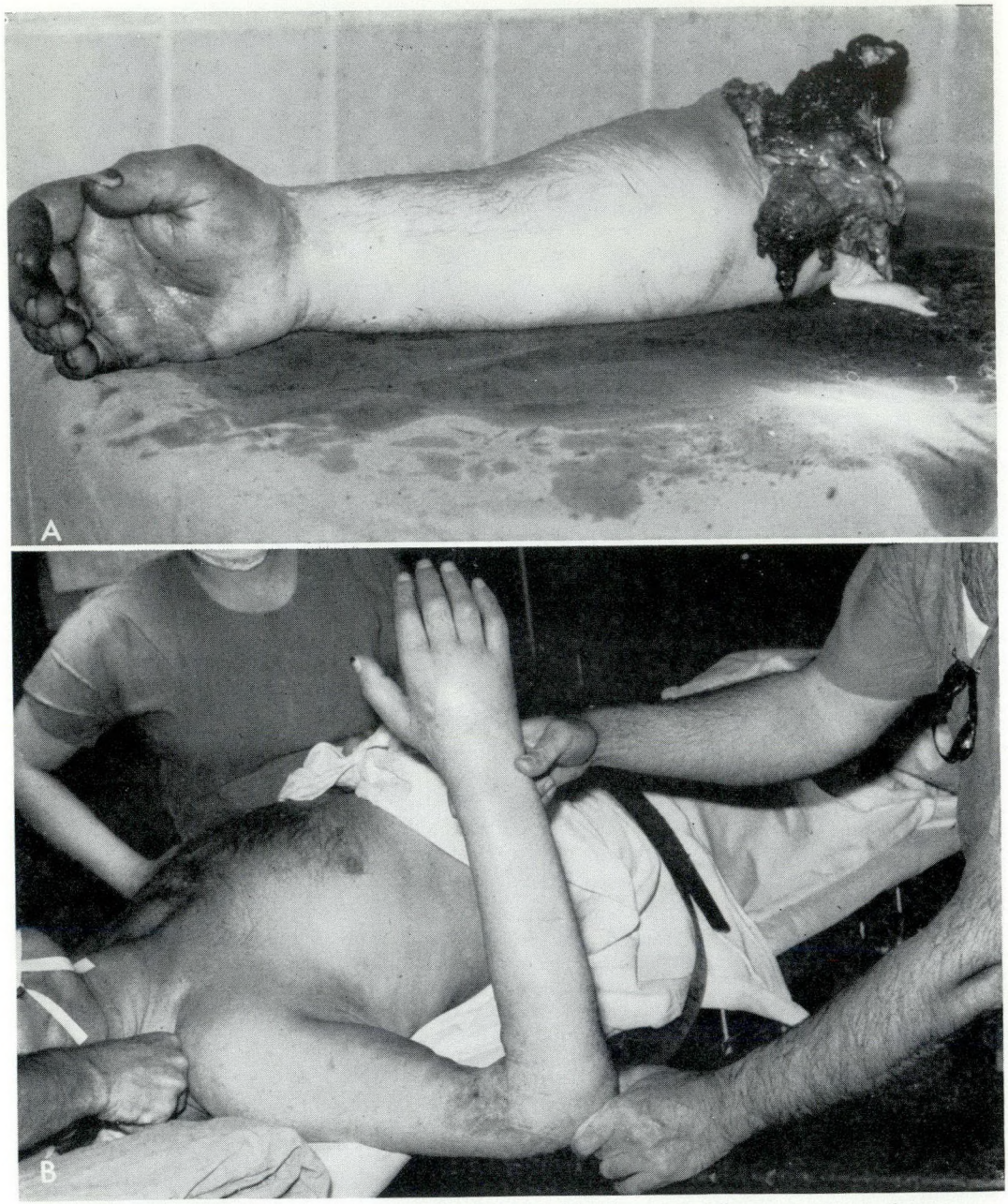

Fig. 3.—(A) The severed arm before replantation. (B) The replanted arm at 5½ months.

During the first 25 days the patient also developed systemic complications. During the first week, he had hematuria, which was controlled by reducing the dose of heparin. Also, a heavy growth of *B. pyocyaneus* was cultured from the posterior skin flap and the patient's temperature rose to 102° F. During the second week he became toxic and the organism was recovered from the blood stream on successive cultures. We had to choose between the patient's general health and the survival of the extremity. Consequently, on the sixteenth day, the shunt was discontinued. A venogram, taken on the preceding day, showed that only one superficial vein crossed the suture line (Fig. 2). Fortunately, by this time, sufficient collateral venous circulation had developed to provide adequate venous return. The septicemia was controlled by adding chloramphenicol to the penicillin and oxacillin. All anti-

biotics were discontinued at the end of the third week. During this period the patient had hemoptysis. A chest film confirmed the clinical impression of pulmonary embolus, which had occurred even though the patient had been receiving heparin since the operation. Eventually, however, the pulmonary changes cleared completely. Low molecular weight dextran was discontinued 25 days after operation.

After the patient left the intensive care unit, we started intensive physiotherapy which included galvanic stimulation. Radiographs showed some evidence of callus formation at the fracture line. Although the arm was slightly swollen, the colour, temperature and nutrition appeared almost normal. Healthy granulation tissue had filled in the area of skin necrosis. On examination of the arm, there was no evidence of motor or sensory nerve function. The patient was in excellent spirits and his general condition was satisfactory.

On January 19, 1967 the skin defect on the medial side of the elbow was covered with a graft. After the operation the patient developed cardiac arrhythmia that was attributed to atherosclerotic heart disease. He was started on quinidine and the arrhythmia improved. Two more grafts were laid on before the area was completely covered.

At the time of the second skin graft on February 22, the median and ulnar nerves were explored. The distal and proximal ends of the ulnar nerve were found and joined end-to-end. Because the distal end of the median nerve could not be found, the proximal end of the median was attached to the proximal end of the ulnar nerve (end-to-side) in the hope that some median nerve fibres might regenerate along the distal ulnar trunk. The last skin graft was done on March 21, three days before the patient was discharged.

At the time of discharge, the edema in the arm was minimal but there was no evidence of nerve recovery. Clinically and radiologically the fracture had not united and, despite intensive daily physiotherapy, the movement in all joints was greatly limited. While in hospital he wore an arm splint which maintained abduction at the shoulder to at least 90°. After discharge, daily physiotherapy was continued. Bony union was slow, but clinically the fracture became stable and the soft tissue healing was complete (Fig. 3) five months after his injury.

His subsequent course was discouraging. He had causalgia in his arm which was very difficult to control. Five and one-half months after the injury, there was still no evidence that any nerve function had returned. On faradic stimulation, there was no innervation of the forearm and the hand. The patient became preoccupied with his arm and quite depressed. Because there was no hope that the arm would recover, an above-elbow amputation was done on June 4.

At operation bony union was satisfactory. A neuroma had formed at the transection site on all three nerves; the muscles were pale, edematous and on stimulation contracted sluggishly. His postoperative course was uneventful. He started a program of rehabilitation immediately and a prosthesis was fitted so that he could return to work as quickly as possible.

On examination of the amputated extremity, there was patchy necrosis of the articular cartilage of the joints (Fig. 4). The tendons had adhered to the tendon sheath and the gliding motion was severely limited. The muscles were pale and edematous. As noted above, neuromas had formed at the transection line of all three nerves and distally there was extensive wallerian degeneration with fibrosis. The muscles showed edema and fibrosis with advanced atrophic changes (Fig. 5).

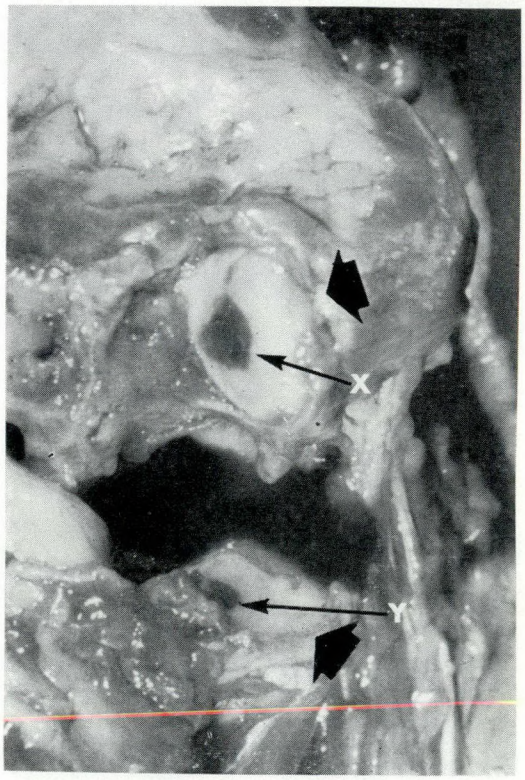

Fig. 4.—Capitellum (X) and the head of the radius (Y) showing gross hemorrhage and necrotic changes involving the articular cartilage.

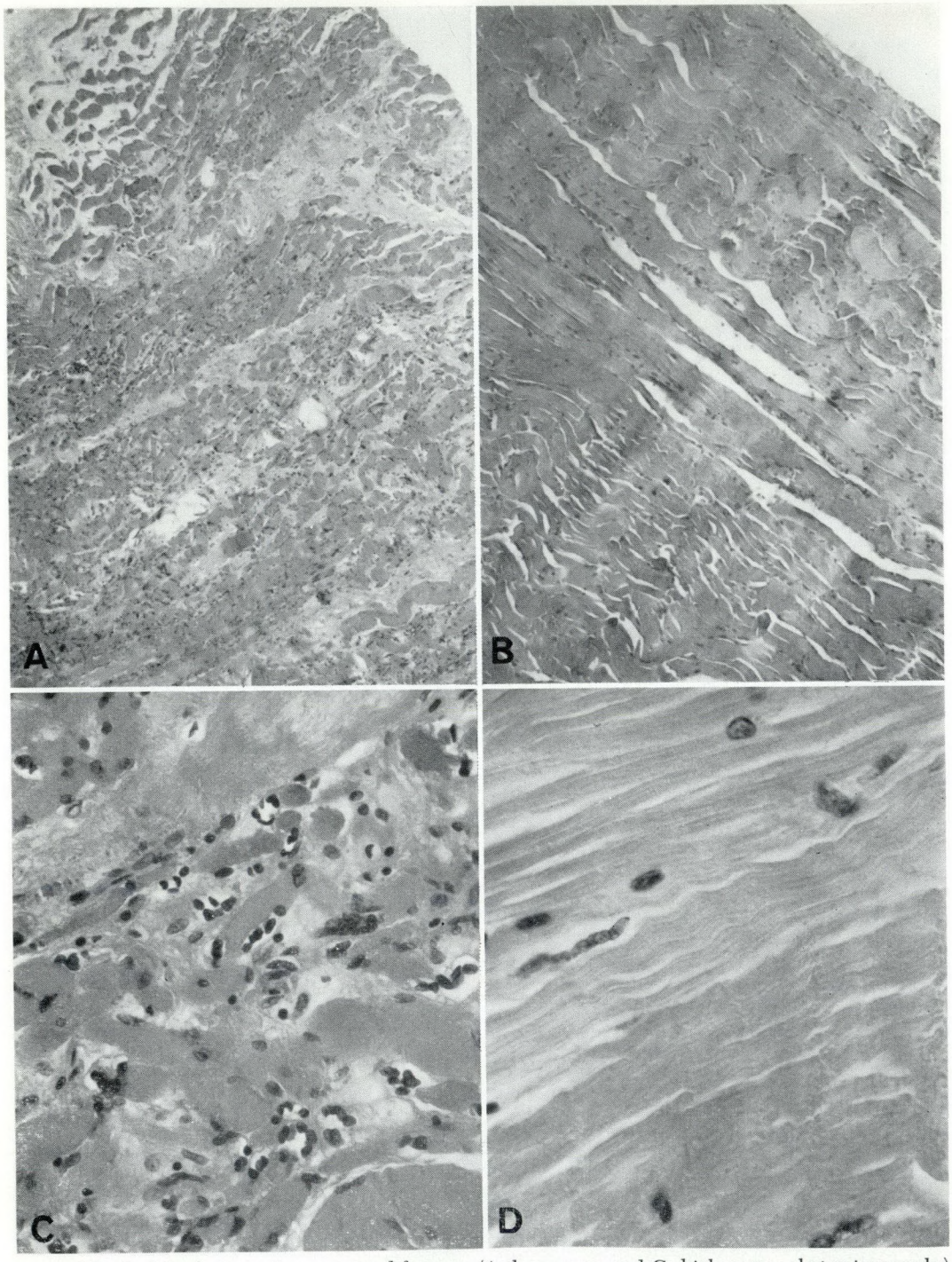

Fig. 5.—Muscle from the amputated forearm (A—low power and C—high power photomicrographs) showing fragmentation and interstitial fibrosis (B and D—normal muscle at the same magnifications for comparison).

Discussion

Malt and McKhann[1] did the first successful replantation of severed extremities in 1964. Eiken *et al.*[2-4] and Lapchinsky[5] discussed the technical aspects of replantation and the physiological changes associ-

ated with the procedure. From Japan, Inoue *et al.*[6] described six patients in whom completely or virtually completely severed limbs had been replanted during a two-year period—October 1962 to October 1964.

In spite of technical advances and the surgeon's familiarity with local and systemic complications, the failure rate in replantation is high. In the patient described in the present report, the replantation was a technical success, but the arm had no function. Although viability depends upon the restored arterial and venous circulation, the replanted extremity is useless without adequate reinnervation.

The basic principles of replantation have been described in detail elsewhere.[1-10] In our opinion, it makes no difference whether the arteries or veins are anastomosed first. Nerve repair is probably much more important. Inoue *et al.*[6] showed that the return of function was significantly improved if the repair was done at once: not only is it more difficult to find the nerves in the dense scar tissue at a second exploration but complications in the immediate postoperative period may significantly delay nerve repair. Our failure in the present case was due chiefly to the fact that we did not repair the nerves at the first operation.

In the immediate postoperative period, the principal problem is inadequate venous return because it is difficult to anastomose enough veins. Several methods have been described by which to decompress an extremity,[11] but to our knowledge, the shunt described in this paper has not been used before. The shunt has several advantages: a suitable systemic vein is readily available on another extremity and the blood can thus be returned to the patient immediately through a closed system. The system itself is simple and requires only standard intravenous tubing. The pressure gradient in the venous shunt is usually sufficiently high so that blood flow from the affected extremity is adequate and enough blood is shunted across to decompress the limb. Without the shunt, our patient's arm would have become gangrenous in a few days. The shunt must be continued until there is adequate regeneration of lymphatic and venous channels. This occurred in 16 days. This method of decompression may be employed not only after replantation, but also in the treatment of acute venous thrombosis, crush injuries, wringer injuries, indeed in any clinical situation where venous return is acutely embarrassed.

The shunt has serious disadvantages, however. First, infection may be spread either by shunting blood to the systemic circulation from a potentially infected area or from an already established infection. Constant supervision is necessary to keep the shunt open. If the pressure gradient is low, frequent manipulations of the tubing may not only introduce infection but dislodge emboli. If silicone tubing is used, thrombus formation may be prevented even if the circulation is sluggish. If cannulation of a vein is prolonged, thrombophlebitis may develop. During the period the shunt is in use the patient's rehabilitation is delayed.

Despite adequate circulation, serious pathological changes developed in the replanted arm in the six months that it was attached. A neuroma formed in each nerve at the transection line. In the nerves themselves there was extensive wallerian degeneration and no myelinization. The muscles became edematous and marked interstitial fibrosis developed. The tendons were almost completely fixed to their sheaths. Adequate new bone formed at the fracture line but the cartilage of the joints showed extensive degenerative changes.

We made several errors: first, at 58, a workman, even though he is physically fit, is too old to undergo the one to two-year ordeal[1, 6, 8-10] necessary to regain satisfactory function. *Replantation should be considered only in children and young adults.* Second, after the avulsion, we did not shorten the proximal humeral shaft sufficiently and, hence, the anastomosis of arteries and veins was made unnecessarily difficult. To bridge the gap we had to use vein grafts. *Bone ends must be shortened adequately so that soft tissues, vessels and nerves can be approximated.* Third, we now know that when we were unable to repair all the nerves at the first operation, the limb was doomed. *Primary nerve repair is essential.*

Conclusions

With present techniques, upper-limb replantation should be attempted only in children and young adults. The proximal humeral shaft must be shortened adequately so that soft tissues, vessels and nerves can be approximated without tension.

Primary nerve repair is essential: the ultimate fate of the replanted limb depends on regeneration of the nerves. Without primary nerve repair, it is highly unlikely that a useful, functioning extremity can be obtained. At replantation, if venous return is inadequate, the shunt described here may keep the extremity alive by diverting trapped venous blood until collaterals develop.

We are indebted to all medical, nursing, laboratory, photography and secretarial personnel at St. Michael's Hospital, Toronto who contributed to the care of this patient and to the preparation of this report.

References

1. Malt, R. A. and McKann, C. F.: Replantation of severed arms, *J. A. M. A.*, **189:** 716, 1964.
2. Eiken, O. *et al.*: Limb replantation. I. The technique and immediate results, *Arch. Surg. (Chicago)*, **88:** 48, 1964.
3. Eiken, O. *et al.*: Limb replantation. II. The pathophysiological effects, *Arch. Surg. (Chicago)*, **88:** 54, 1964.
4. Eiken, O. *et al.*: Limb replantation. III. Long-term evaluation, *Arch. Surg. (Chicago)*, **88:** 66, 1964.
5. Lapchinsky, A. G.: Recent results of experimental transplantation of preserved limbs and kidneys and possible use of this technique in clinical practice, *Ann. N.Y. Acad. Sci.*, **87:** 539, 1960.
6. Inoue, T. *et al.*: Factors necessary for successful replantation of upper extremities, *Ann. Surg.*, **165:** 225, 1967.
7. Herbsman, H., Lafer, D. J. and Shaftan, G. W.: Successful replantation of an amputated hand: case report, *Ann. Surg.*, **163:** 137, 1966.
8. Horn, J. E.: Successful reattachment of a completely severed forearm; a commentary, *Lancet*, **1:** 1152, 1964.
9. Rosenkrantz, J. G. *et al.*: Replantation of an infant's arm, *New Eng. J. Med.*, **276:** 609, 1967.
10. Williams, G. R. *et al.*: Replantation of amputated extremities, *Ann. Surg.*, **163:** 788, 1966.
11. Winfrey, E. W., III and Foster, J. H.: Low molecular weight dextran in small artery surgery; antithrombogenic effect, *Arch. Surg. (Chicago)*, **88:** 78, 1964.

Résumé

Avec les techniques actuelles, la replantation du membre supérieur ne devrait être entreprise que chez l'enfant ou l'adulte jeune. La diaphyse humérale proximale doit être raccourcie de façon adéquate afin que les tissus mous, les vaisseaux et les nerfs puissent être rapprochés sans étirement.

La réparation des nerfs primaires est essentielle: le succès final de la replantation dépend de la régénération des nerfs. Si les nerfs primaires ne recommencent pas à fonctionner, le membre ne pourra pas travailler et sera inutile. Si au cours de la replantation le retour veineux est insuffisant, le shunt décrit dans cette présentation permet de dévier la stase veineuse en attendant que se développe une circulation collatérale.

MANAGEMENT OF ESOPHAGEAL PERFORATION

The availability of effective antibiotics has permitted the physician to treat successfully small perforations of the esophagus. The fact that some small perforations can be treated successfully by conservative management is not questioned; however, difficulty arises in determining whether the perforation is small both in physical size and in the amount of mediastinal soilage that can occur from it. Many patients treated initially by conservative supportive measures are ultimately recorded as operative failures when, two to four days after the institution of antibiotic therapy, deterioration of the patient prompts belated operative intervention.

Nineteen patients with esophageal perforation that occurred between 1952 and 1956 were reviewed at the Minneapolis Veterans Administration Hospital. The overall mortality was 26%. Eleven patients were treated conservatively, with a 36% mortality rate; six required operative intervention one to three days after the institution of medical management. Four (67%) of these six patients died. Of eight who had surgical treatment as soon as the diagnosis was made, one died, a mortality of 12%. The death in this group followed a proximal gastrectomy performed for carcinoma of the stomach.—Youngs, J. and Nicoloff, D.: Management of esophageal perforation, *Surgery*, **65:** 264, 1969.

APPENDICOCECAL INTUSSUSCEPTION: A CASE REPORT*

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INTUSSUSCEPTION of the appendix, an uncommon lesion, is itself an unusual cause of ileocolic intussusception. Fraser,[1] reviewing the literature in 1943, found about 100 cases of intussusception of the appendix. Rarely is this made as a preoperative diagnosis or is a barium study performed with this lesion in mind.

We describe a patient with intussusception of the appendix which progressed to an ileocolic intussusception. Barium examination showed a filling defect in the cecum which suggested that an appendiceal lesion was present.

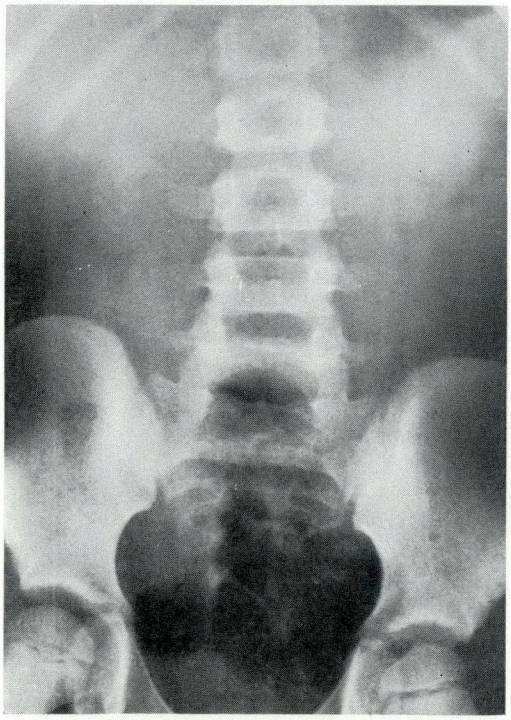

Fig. 1.—Flat plate of the abdomen on admission. There is a moderate amount of gas in the distal small bowel but no gas in the colon.

CASE REPORT

A.P., a 7-year-old boy, entered the Montreal Children's Hospital on July 14, 1962 with a 36-hour history of vomiting and crampy abdominal pain. The pain, which was localized to the right lower quadrant and periumbilical region, was colicky. He had no previous history of abdominal pain and no previous operations.

His temperature was 98° F., pulse rate 96/min. and respiration 22/min. A sensation of emptiness on palpation in the right lower quadrant (Dance's sign) was detected, and a sausage-shaped mass was palpated in the right upper quadrant. During episodes of colic, high-pitched bowel sounds were heard. There was no bloody stool or blood on the examining finger. The hemoglobin was 11 g./100 ml. and the leukocyte count 9000/c.mm. with a normal differential. The child did not appear toxic or acutely ill. The clinical diagnosis was intussusception.

The flat film of the abdomen (Fig. 1) showed a moderate amount of gas in the small bowel which was confined almost entirely to the distal portion; there was no dilatation of individual loops. The colon contained no gas. When the barium enema was done (Fig. 2), barium flowed freely from the rectum to the proximal portion of the ascending colon. A large intraluminal filling defect was seen in the area of the cecum and proximal portion of the ascending colon. The features of the radiograph were characteristic of an intussusception. Initially, barium did not enter the terminal ileum or appendix. After evacuation, fluoroscopy showed barium in the terminal ileum, but a filling defect in the cecum (Fig. 3) remained. (We did not expect to see a residual filling defect in the cecum after barium entered the terminal ileum because we thought the ileocolic intussusception had been reduced. The remaining defect was the appendicocecal intussusception and was the reason for the second barium enema 48 hours later.)

Since the obstruction had been relieved by the barium enema, the only treatment the child received was intravenous fluids and

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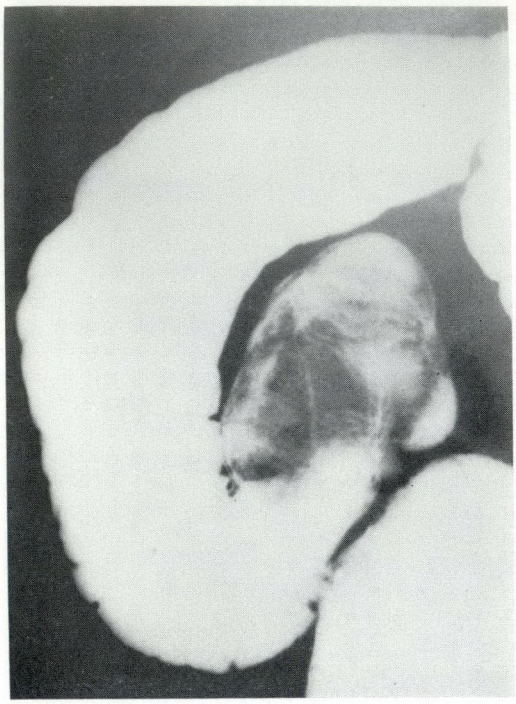

Fig. 2.—Barium enema on admission. Note the intussusceptum and a large filling defect in the cecum.

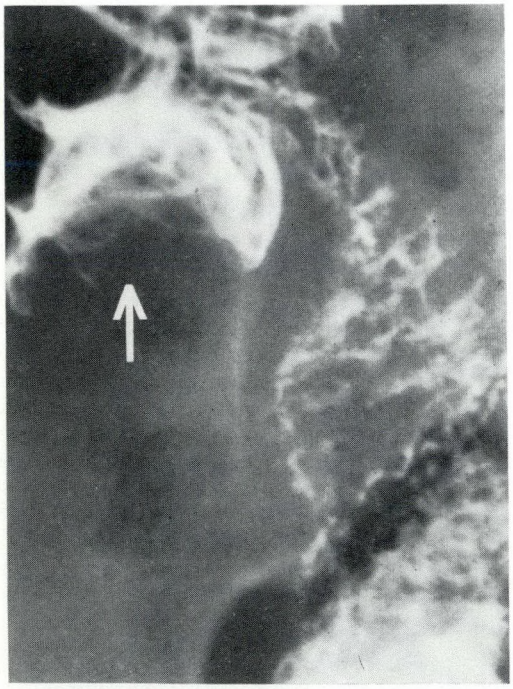

Fig. 3.—Barium enema on admission. The terminal ileum is now filled with barium, but there is a filling defect in the cecum (arrow).

nasogastric suction for the next 24 hours. Bowel movements were normal. The oral fluids were well tolerated. Forty-eight hours later a second barium enema was performed (Fig. 4). The barium flowed up into the cecum and outlined the filling defect seen previously. This filling defect, which was radiolucent, projected into the lumen of the cecum and measured about 4 x 2 cm.

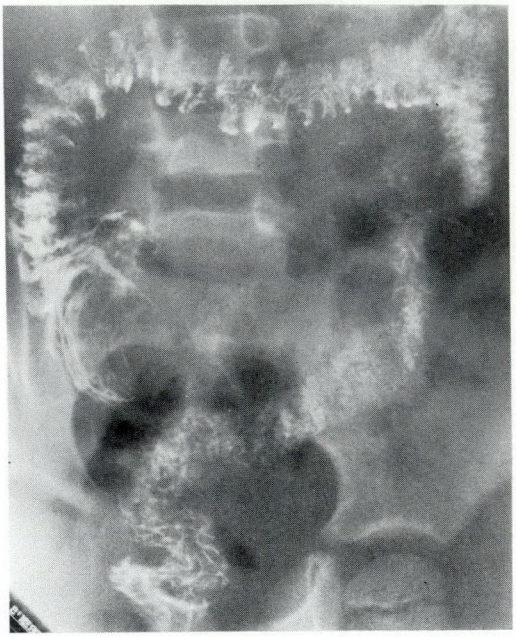

Fig. 4.—Barium enema 48 hours after admission. A rounded filling defect occupies the cecum.

Because the cecal mass had persisted the abdomen was explored on July 18, 1962. No intraluminal or extraluminal masses were found in the cecum except at the caput. Here the appendix had intussuscepted (Fig. 5) and could not be reduced. The base of the appendix could be palpated through the cecal wall and, because a tumour of the appendix could not be excluded, the appendix and a cuff of attached cecum were resected. The cecum was carefully examined and then closed in two layers. The patient made an uneventful recovery and was discharged seven days later.

The base of the appendix had invaginated into the cecum (Fig. 6). The muscular coat of the cecum was continuous with that of the appendix. On microscopy there was marked lymphoid hyperplasia of the appendiceal wall, which produced marked narrowing of the lumen and focal ulceration of the mucosa. The lumen contained exudate rich with polymorphonuclear leukocytes. There was no evidence of malignancy.

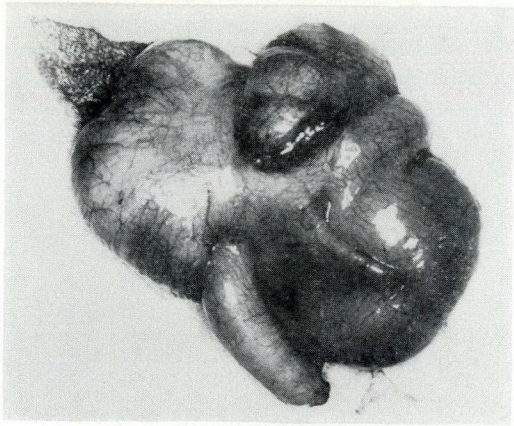

Fig. 5.—Cecum at operation. The base of the appendix is intussuscepted into the cecum and only the distal one-half of the appendix is visible.

Discussion

In 1953 Forshall,[2] having reviewed the literature on appendiceal intussusception,[1,3] classified the various types that may occur. In well over one-half of the recorded cases, the intussusception found at operation was secondary. The patient is usually operated upon as an emergency with a diagnosis of appendicitis or ileocolic intussusception. When a barium enema examination is performed, the radiologist usually interprets the findings as representing: a polyp, lipoma or even carcinoma of the cecum, or carcinoid or mucocele of the appendix.

The etiological factors have been classified by Forshall[2] into anatomical and irritative. Among the anatomical factors are a wide appendicular lumen, a thin, mobile mesoappendix, and the absence of peritoneal folds about the appendix. Irritative factors include worms, foreign bodies, polyps, mucocele, and hypertrophied lymphoid follicles or mucous membranes.

Mitchell[4] described two allergic children with chronic intussusception of the appendix who were successfully treated by appendectomy.

Mucocele of the appendix, which has been reported to cause appendiceal intussusception,[5-10] should be suspected when a filling defect persists in the cecum after the intussusception is reduced following a barium enema.

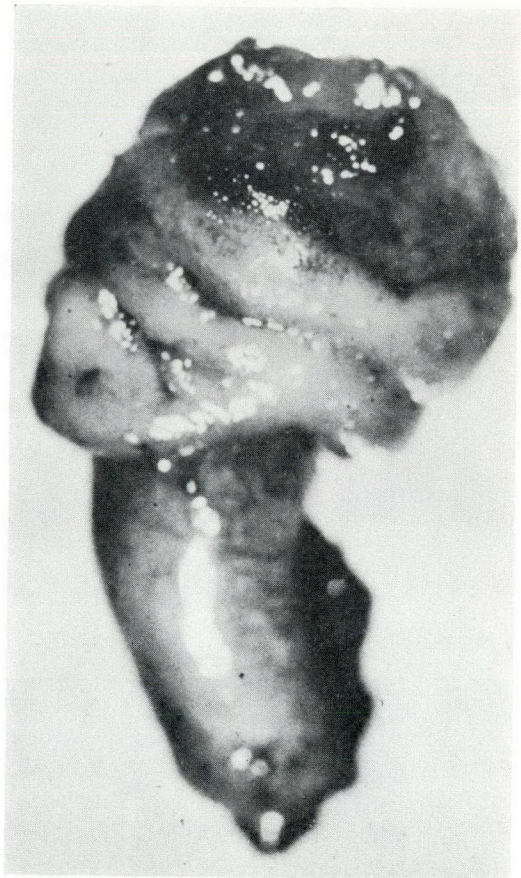

Fig. 6.—Resected appendix including a cuff of cecum, showing the invaginated portion of the appendix.

Summary

Intussusception of the appendix, an uncommon condition, is an unusual cause of ileocolic intussusception. A 7-year-old boy presented with symptoms of ileocolic intussusception. While a barium enema reduced the ileocolic intussusception and showed free flow of barium into the terminal ileum, a filling defect persisted in the cecum even after the obstruction had been relieved and symptoms had disappeared. Repeat barium enema done 48 hours after the first one revealed a persistent filling defect in the cecum. At laparotomy this proved to be an appendicocecal intussusception that was irreducible and required resection of the appendix with a cuff of cecum. Following an uneventful recovery this boy was discharged from hospital, seven days after operation. The pertinent literature is also reviewed.

References

1. Fraser, K.: Intussusception of the appendix, *Brit. J. Surg.*, **31:** 23, 1943.
2. Forshall, I.: Intussusception of the vermiform appendix with a report of seven cases in children, *Brit. J. Surg.*, **40:** 305, 1953.
3. McSwain, B.: Intussusception of the appendix; review of literature and report of case, *Southern Med. J.*, **34:** 263, 1941.
4. Mitchell, R. G.: Intussusception of vermiform appendix in allergic children, *Brit. Med. J.*, **1:** 265, 1955.
5. Adelman, B. P. and Teplick, J. G.: Intussusception of appendiceal mucocoeles, *Amer. J. Roentgen.*, **73:** 966, 1955.
6. Beard, D. D.: Intussusception of mucocele of the appendix, *Med. J. Aust.*, **1:** 43, 1955.
7. Boon, T. H. and Sibley, J. C.: Complete inversion of appendix, *Lancet*, **1:** 1304, 1951.
8. Elson, M. W.: Cecal invagination by a mucocele of the appendix, *Radiology*, **71:** 90, 1958.
9. McCormick, W. F.: Intussusception of the vermiform appendix with a mucocele; review of literature with report of a case, *A. M. A. Arch. Path.*, **64:** 686, 1957.
10. Zeifer, H. D.: Intussusception of appendix; report of case and review of literature, *J. Mount Sinai Hosp. N.Y.*, **18:** 125, 1951.

Résumé

L'invagination de l'appendice est une rareté clinique et c'est une cause exceptionnelle d'une invagination iléo-colique. Les symptômes de cette pathologie se rencontraient chez un garçonnet de 7 ans. Un lavement baryté permit de réduire l'invagination iléo-colique et le lavement baryté s'écoulait librement dans l'iléon terminal. Un défaut de remplissage persistait néanmoins dans le cécum, même après que l'obstacle eut été levé et que les symptômes eussent disparu. Un nouveau repas baryté, donné 48 heures plus tard, révéla la persistance du défaut de remplissage situé dans la tête du cécum. Une laparotomie permit de constater que ce défaut était attribuable à une invagination appendico-cécale irréductible. On procéda à la résection de l'appendice et d'une bande de cécum. Après une convalescence sans incidents, le garçonnet reçut son congé le septième jour postopératoire. Les auteurs passent en revue la littérature pertinente.

CYANOACRYLATE ADHESIVES AS TOPICAL HEMOSTATIC AIDS

This study establishes the efficacy of certain cyanoacrylate monomers as topical hemostatic aids. The model used was an attempt to duplicate a particularly unfavourable type of liver wound—an open area which is not apposed to another body surface. Every effort was made to achieve maximum survival with the use of currently standard methods in the control group, short of the use of resuscitative fluids, antibiotics, and drainage. With this model we could not confirm the efficacy of methylcyanoacrylate as a hemostatic agent on liver wounds in dogs, but we could demonstrate the efficacy of the more rapidly polymerizing, better spreading compounds.

However, a disturbing finding was the high incidence of local sepsis. Antibiotics were not used, making this a particularly severe test in this regard. Nevertheless, a contaminated field will be present in many suitable clinical situations. The incidence of local sepsis was high in this study (almost one-third), and examples of persisting sepsis were found up to 21 months after application. These polymer films are not homogeneous plastic masses, but rather they are lamellar or honeycombed structures which might be expected to contribute to persistent local sepsis. It should be pointed out that a similar incidence of local sepsis was found in the control group and in animals treated with the rapidly degrading methyl, but never after several months in groups in which the polymer was not present.

This points up the desirability of a compound which will degrade within a reasonable period of time. Methyl does this, but it is locally necrotizing, clearly less effective in topical hemostasis, and forms a weaker tissue-to-tissue bond than the longer alkoxy compounds. Branched alkoxy homologues are effective and may degrade more rapidly, but this was not very evident with the isobutyl used in this study. Mixtures of compounds have been studied in the hope of combining the best features of various groups, but with little success.

In the present study, even the most effective cyanoacrylates could not stop brisk bleeding by topical application alone. Clearly there were limitations to their use as hemostatic aids, but in low-pressure bleeding from an accessible surface which can be temporarily dried, they may be clinically useful when standard techniques have failed.—Collins, J. A. *et al.*: Cyanoacrylate adhesives as topical hemostatic aids. I. Experimental evaluation on liver wounds in dogs, *Surgery*, **65:** 256, 1969.

STRANGULATED OBTURATOR HERNIA PRESENTING AS SUBCUTANEOUS EMPHYSEMA OF THE THIGH*

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OBTURATOR hernia was first recognized by de Rensil who described such a case to France's Royal Academy of Sciences in 1724. Hilton did the first laparotomy for strangulated obturator hernia in 1848 and in 1851, Henriobre operated successfully on a patient with this type of hernia. It should be noted that this diagnosis is rarely made before intestinal symptoms develop or before emergency laparotomy is done.[1-3] It is unusual for a patient with strangulated obturator hernia to present with chronic complaints and subcutaneous emphysema of the upper thigh. Such a patient is the subject of this report.

CASE REPORT

G.D., a 60-year-old woman, was admitted to the Medical Institute Hospital, New Delhi, on September 21, 1967 with complaints of intermittent pain in the abdomen for 13 years, and pain in the front of the right thigh for the last 15 days. She was attending the orthopedic clinic of the Hospital for the pain in her thigh. On investigation, she was found to have cholelithiasis for which she was admitted to the surgical ward. She had no history of abdominal distension, vomiting or acute abdominal pain before this admission. Two years before, she had had a vaginal hysterectomy for prolapse of the uterus.

This woman had a pulse of 100/min., a temperature of 37.8° C. and a blood pressure of 140/98 mm. Hg. Her respiratory and cardiovascular systems were within normal limits. The abdomen was scaphoid with tenderness and mild rigidity in the right hypochondrium. The right hip had a fixed flexion deformity of 90° and any movements of it were painful. An abscess in the thigh just below the right greater trochanter was drained on September 22. Gas bubbles escaped when the deep fascia was incised. A draining sinus resulted. Antibiotics were administered and the hip movements returned to normal but the sinus persisted. Some pus and occasional gas bubbles were discharged from it. On vaginal examination, no mass or other abnormality was detected. Her hemoglobin was 10.2 g./100 ml.; total leukocyte count was 9800/c.mm.; erythrocyte sedimentation rate was 10 mm. in one hour (Westergren). The urine and liver function tests were normal. A plain film of the abdomen showed multiple gallstones and osteoarthritis of the left hip. A radiograph of the pelvis done on September 27 showed increased soft-tissue density ("cellulitis") with gas on the right side (Fig. 1). When opaque medium was injected, the sinus tract was seen to travel superiorly, anteriorly and medially (Fig. 2). It appeared to communicate with the rectum because the medium spilled into it (Fig. 3). The lower limit of the tract was the mid-shaft of the femur. An upper gastrointestinal series and barium enema revealed no abnormalities.

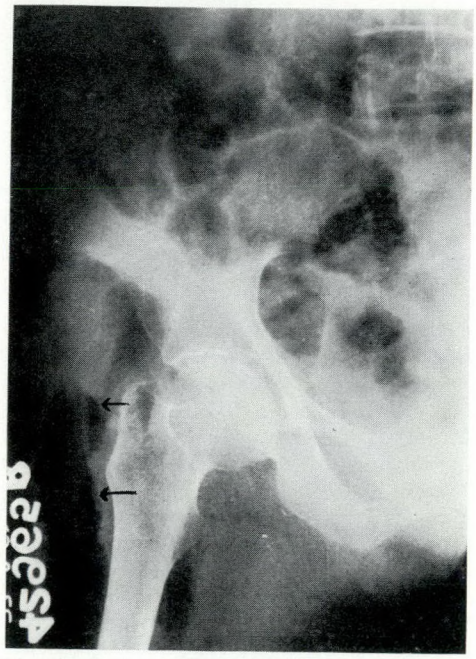

Fig. 1.—Radiograph of the pelvis and upper one-half of the right thigh showing soft-tissue cellulitis with gas.

We made a provisional diagnosis of Richter's hernia of the right obturator canal and did a laparotomy. The right lateral wall of the large bowel at the rectosigmoid junction had herniated into the right obturator canal. We

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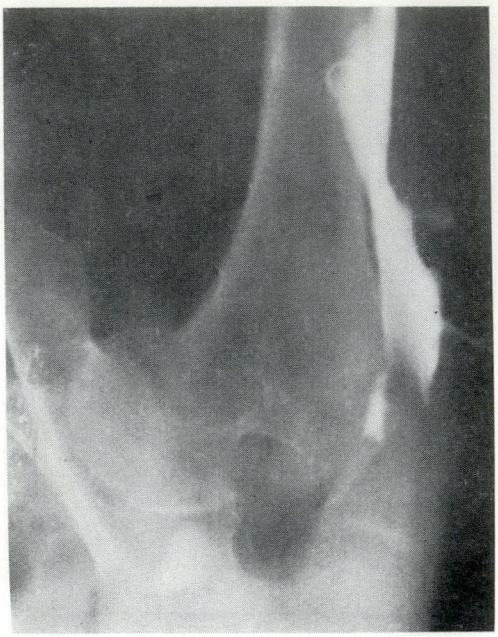

Fig. 2.—Sinogram showing the tract.

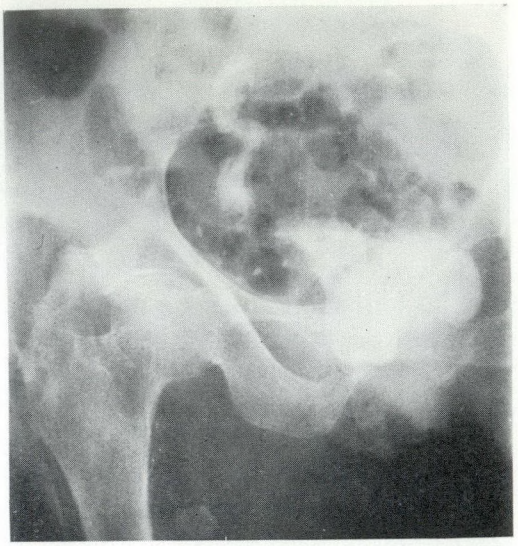

Fig. 3.—Sinogram showing dye spilling into the rectum.

could not reduce the hernia so we cut the bowel flush with the internal obturator opening. The hole in the right lateral wall of the rectosigmoid was stitched in two layers transversely. The mucous membrane left behind in the obturator canal was fulgurated, and the internal end of the canal was closed with two interrupted sutures. The pelvic peritoneum was mobilized and sutured over this area.

A corrugated drain was left outside the peritoneum. The postoperative period was uneventful and the sinus healed completely.

Discussion

Obturator hernia is uncommon in men; only 16% of those in Watson's series[4] were male. This hernia is usually seen in patients between the ages of 50 and 80. The diagnosis of reducible obturator hernia is seldom made and presenting symptoms are usually those of the acute intestinal obstruction that follows strangulation. Strangulation is common because the internal opening of the obturator canal is unyielding—made up as it is by the bony wall above, and the steep edge of the firm inelastic obturator membrane below. This condition can be diagnosed before operation if the physician thinks of obturator neuralgia (Howship-Romberg's sign). This sign is present in most of these patients,[5] but is overlooked because obturator hernia is so uncommon. The strangulated obturator hernia is usually of the Richter type and a tender mass is not palpable on rectal or vaginal examination.

In his large collected series, Watson[4] found that strangulation had occurred 1 to 30 days before the patient was operated upon or died. The patient described in this communication had a Richter's type of strangulation, which was misinterpreted as an episode of acute cholecystitis. She did not have complete intestinal obstruction and her bowels were moving. A small area over the apex of the affected loop sloughed off, allowing only gas to escape into the tissues. The gas tracked downwards, separated the tissue planes, and presented as an abscess on the lateral thigh just below the greater trochanter. When the abscess was incised and drained, and antibiotics were administered, the local inflammation subsided. The patient then was free of symptoms except for a sinus which occasionally discharged a few bubbles of gas.

It is difficult to reduce the contents in obturator hernia and excise the sac completely.[5] It is much easier and less shocking in patients with Richter's type of strangulation to cut the bowel flush with the internal obturator foramen and repair the defect. There is little chance of recurrence: first, the portion of the bowel left in the

obturator hernia will produce fibrosis; and second, the obturator foramen is now extraperitoneal.

Summary

A 60-year-old woman had an unusual strangulated obturator hernia which presented as subcutaneous emphysema of the thigh. The hernia, which was of the Richter type, was not reduced. The trapped bowel was resected, the defect repaired, and the internal end of the obturator canal closed. The patient made an uneventful recovery.

References

1. Adams, H. D. and Smith, D. C.: Obturator hernia; report of a successful operative case, *J. A. M. A.*, **137:** 948, 1948.
2. Desmond, A. M. and Hutter, F.: Strangulated obturator hernia, *Brit. J. Surg.*, **35:** 318, 1948.
3. Harper, J. R. and Holt, J. H.: Obturator hernia, *Amer. J. Surg.*, **92:** 562, 1956.
4. Watson, L. F.: Hernia; anatomy, etiology, symptoms, diagnosis, differential diagnosis, prognosis and treatment, 3rd ed., The C. V. Mosby Company, St. Louis, 1948, p. 457.
5. Kwong, K. H. and Ong, G. B.: Obturator hernia, *Brit. J. Surg.*, **53:** 23, 1966.

Résumé

Une femme de 60 ans présentait une déformation douloureuse de la cuisse droite qui la fixait en flexion à 90°; elle avait aussi un abcès de la cuisse droite juste en-dessous du grand trochanter. Lorsque fut incisée l'aponévrose profonde recouvrant l'abcès, des bulles gazeuses s'en échappèrent. Bientôt un canal se ferma, il était dirigé en haut, en avant et vers le milieu et communiquait avec le rectum. Le diagnostic pré-opératoire de hernie étranglée de Richter (au niveau du trou obturateur) fut confirmé à la laparotomie. La hernie ne fut pas réduite parce qu'il y avait de grosses adhérences. Le gros intestin, à la jonction recto-sigmoïdienne, fut reséqué au même niveau que le trou obturateur interne et les lésions intestinales furent réparées. L'extrémité interne du canal obturateur fut fermée et recouverte par le péritoine. Ce procédé opératoire est plus facile et cause moins de choc que la réduction forcée classique. Il y a peu de chance de récidive, d'abord parce que la portion d'intestin laissée dans la hernie du trou obturateur donnera du tissu fibreux, et ensuite, parce que le trou obturateur est maintenant extrapéritonéal.

FEMOROPOPLITEAL OCCLUSIVE DISEASE

An appreciation of the concept of critical stenosis is of great practical significance in the evaluation of patients for reconstructive arterial surgery. Aortoiliac stenosis that is associated with femoropopliteal occlusive disease should be promptly detected, and treated accordingly, before operation of the more distal lesion is undertaken. This may improve the long-term results of the reconstructive procedures and, at times, may even obviate their use.

It appears from the present study that more than 25% of the patients who have femoropopliteal disease also display aortoiliac lesions of hemodynamic significance. Serial arteriographic technique is a prerequisite for a comprehensive assessment of the arterial lesions. Detailed anatomic information of the arterial disease of the lower extremity is obviously essential for correct prognostic evaluation and appropriate surgical indications.

An analysis of 321 arteriograms of the lower extremity obtained by the transfemoral aorto-arteriographic method disclosed a variety of occlusive arterial patterns (tibial-peroneal, popliteal, popliteal-tibial, femoropopliteal, femoropopliteal-tibial, and aortoiliac).

The incidence of femoropopliteal disease as an isolated lesion was small (5.2%) in contrast to the combined femoropopliteal-tibial patterns (49.2%).

Three basic aortoiliac patterns were often associated with the above lesions: (1) aortoiliac stenosis, (2) unilateral iliac occlusion, and (3) aortoiliac aneurysm. In addition, aortoiliac tortuosity was frequently an accompanying feature.

The hemodynamic significance of the aortoiliac patterns was evaluated both clinically and by means of simultaneous intra-arterial pressure readings.

In this study, 27% of the aortoiliac lesions of hemodynamic significance were associated with femoropopliteal disease.

Appreciation of the concept of critical stenosis of the aortoiliac segment is of great practical significance in the evaluation and treatment of the femoropopliteal lesions.—Haimovici, H. and Steinman, C.: Aortoiliac angiographic patterns associated with femoropopliteal occlusive disease: significance in reconstructive arterial surgery, *Surgery*, **65:** 232, 1969.

EXPERIMENTAL SURGERY

COMPENSATORY RENAL HYPERPLASIA*

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THE increase in size of the remaining kidney following unilateral nephrectomy is referred to as "compensatory hypertrophy". The use of this term has tended to obscure the fact that hyperplasia is an integral part of this phenomenon. While some of the increase in size is due to an enlargement of nephrons, no new nephrons are formed.

Following unilateral nephrectomy, a peak in the hyperplastic response can be demonstrated in the remaining kidney. Most investigators have found that it occurs between 36 and 48 hours after nephrectomy.[1-4] The extent of the hyperplastic response is modified by many factors, such as the age, diet and sex of the animals. While there are conflicting reports on the effect of hypophysectomy on the hyperplastic response,[5, 6] it still occurs following thyroidectomy, adrenalectomy or orchiectomy.

The mechanism through which the hyperplasia is induced is not yet understood. Braun-Menendez[7] has summarized the evidence against functional overload as such a mechanism. Ogawa and Nowinski,[8] and Lowenstein and Stern,[9] using tissue culture techniques and *in vivo* studies, demonstrated a mitosis-stimulating humoral factor in the serum of animals after unilateral nephrectomy. However, Kurnick and Lindsay,[4] using similar techniques, could not demonstrate such a substance. Argyris and Trimble,[10] working with mice, showed that unilateral renal trauma produced hyperplasia in the contralateral organ. They concluded that the response was not due to loss of renal mass but to the release of a growth-stimulating substance from the traumatized kidney. However, it has been repeatedly demonstrated that the loss of renal mass in unilateral nephrectomy stimulates hyperplasia of the remaining kidney. It is possible that, following nephrectomy, an organ-specific, mitosis-stimulating factor is liberated[9] or that the level of a growth-inhibiting substance is decreased.[1] In any event, the remaining kidney increases in size.

In the following experiments, we examined some of the factors said to influence mitotic activity in the kidney.

MATERIALS AND METHODS

Ten-week-old, male, albino Sprague-Dawley rats were used throughout the experiments. The animals were kept on a standard diet, food and water were given *ad libitum*, and environmental conditions were controlled. Because of the well-known diurnal rhythm in mitotic activity of the kidney, we observed a rigid operating time schedule. The surgical procedures were carried out between 2 and 4 p.m. and, in most cases, the animals were sacrificed 42 hours after operation. In all procedures, the kidney was exposed through a subcostal incision. The initial procedure (nephrectomy, trauma or sham operation) was performed on the right kidney.

In the experiments done to study the effects of trauma, the mean depth of the renal cortex in 10-week-old rats was determined. Trauma was inflicted by puncturing the cortex with a 20-gauge needle, the extent of trauma being determined by the number of punctures.

In the experiments done to study the effects of homogenates and trauma on the level of mitosis, the homogenates were prepared by homogenizing 16 kidneys, weighing a total of 19 g. in a Virtis 45 homogenizer. The final volume of homogenate was made up to 64 ml. with Ringer's lactate solution. The homogenates, which were frozen until needed, were injected intraperitoneally 18 hours before the anticipated peak in hyperplastic response (42 or 48 hours).

In the experiments done to investigate the existence of a humoral factor, serum

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was obtained from sham-operated, and from nephrectomized rats 42 hours after operation. Blood was removed from the abdominal aorta and allowed to stand in a tube for 30 minutes. It was then centrifuged for 10 minutes at 2000 rpm, and the serum removed with a Pasteur pipette. Serum from the animals in each group was pooled, and injected intraperitoneally into intact animals in doses of 3 ml. of serum per 100 g. body weight. Tritiated thymidine was injected intraperitoneally in doses of 0.7 μc./g. body weight four hours before the animals were sacrificed. The removed kidneys were fixed in buffered formalin, sectioned at 5μ thickness, mounted and stained with hematoxylin and eosin. The slides were dipped in Kodak NTB-2 nuclear-track emulsion and exposed for three weeks. After developing, the mitotic response in the tubular cells of the renal cortex was determined by counting the number of labelled nuclei (that is, cells synthesizing DNA) per 50 high power fields. Autoradiographic techniques were used to determine the hyperplastic response in all experiments except the first, where mitotic figures were counted.

Results

Mitotic Response Following Unilateral Nephrectomy

Following unilateral nephrectomy, the extent of the mitotic response is found to vary greatly with time, and appears to reach a peak within the first 48 hours. Early in this study, we investigated the time at which such a peak occurs. Groups of animals were sacrificed at 24, 28, 36, 42, 48, 52 and 96 hours after unilateral nephrectomy, and the removed kidneys were used as controls. The number of mitotic figures in the tubules of the renal cortex was counted, and the mitotic activity expressed as number of mitoses per 1000 cells counted. The maximum hyperplastic response was found to occur at 42 hours (Fig. 1). The data were analyzed in the following manner: for each animal, the mitotic rate in the control kidney (removed at the time of the primary procedure) was subtracted from that of the remaining kidney. The mean of these differences for each time

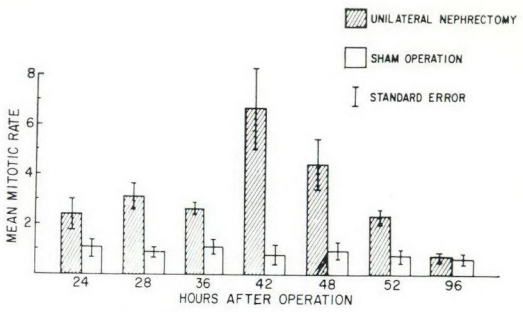

Fig. 1.—Mitosis in the kidney cortex at various times after operation.

period was determined, and the greatest mean difference was found to occur at 42 hours. The difference between the peak responses was found to be statistically significant (non-parametric tests on ranks, $P < 0.01$).

Using tritiated thymidine *in vivo* and autoradiographic techniques, the hyperplastic response in the remaining kidney was examined at 36, 42 and 48 hours after nephrectomy. This experiment was repeated to compare the results obtained using tritiated thymidine and autoradiography with those obtained by counting mitotic figures. Table I suggests that the greatest response occurred at 42 hours. However, statistical analyses suggest that the number of labelled cells at 42 hours was not significantly higher than at 36 hours (students t test—$P=0.20$). The sham values show no such maximum in the number of labelled cells at 42 hours, therefore the peak in labelling in the nephrectomized group appears to be due to kidney removal alone.

TABLE I.—DNA Synthesis in the Remaining Kidney After Unilateral Nephrectomy

Treatment	*Number of rats*	*Hours after treatment*	*Number of labels per 50 fields**
Unilateral nephrectomy	6	36	93.8 ± 14.5
	6	42	111.2 ± 12.7
	7	48	71.0 ± 7.5
Sham operation	2	36	17.5 ± 6.0
	2	42	14.0 ± 6.0
	2	48	14.0 ± 1.0

*Average ± standard error of the mean.

Influence of Renal Trauma on Mitosis in the Contralateral Organ

Two experiments were done to investigate the effects of renal trauma. In the first experiment, two degrees of trauma

TABLE II.—DNA Synthesis in the Intact Kidney Following Unilateral Trauma (Two Experiments)

Treatment	Number of rats	Number of labels per 50 fields*
(a) None	7	34.4 ± 5.4
Sham operation	7	32.0 ± 4.2
Double needle insertion	6	31.5 ± 3.6
Multiple (14) needle insertions	7	34.3 ± 1.5
Unilateral nephrectomy	7	162.9 ± 9.6
(b) None	7	16.6 ± 2.6
Sham operation	7	16.3 ± 3.3
Double needle insertion	5	17.8 ± 0.8
Multiple (20) needle insertions	7	22.9 ± 2.0
Unilateral nephrectomy	6	58.8 ± 10.0

*Average ± standard error of the mean.

TABLE III.—The Effect of Kidney Homogenate on DNA Synthesis in the Intact Kidney Following Unilateral Renal Trauma

Material injected	Amount (ml.)	Time of injection (hours post operation)	Number of labels per 50 fields of cortex*
Kidney homogenate	1	24	16, 17 (16.5)
	1	30	7, 7, 24, 49 (21.7)
	4	24	8, 22, 29 (19.7)
	4	30	1, 6, 15, 22 (11.0)
Suspending medium	1	24	35
	1	30	34
	4	24	46
	4	30	36

*In parenthesis, average value for each group.

were induced by puncturing the renal cortex with a 20-gauge needle 2 and 14 times respectively. In order to measure the hyperplastic response in this group, some animals in the group were subjected to unilateral nephrectomy. The data in Table IIa indicate that, following unilateral nephrectomy, there is a significant hyperplastic response. Analysis of variance among the means of the four remaining groups reveals no significant difference.

The design of the second experiment was similar to the first, except that the trauma was made more extensive by increasing the number of needle insertions from 14 to 20. As in the previous experiment, there is marked hyperplasia after unilateral nephrectomy (Table IIb). The magnitude of the hyperplastic response may vary with each group of animals studied. This is why it was determined as part of each experiment. However, despite the increased trauma, an analysis of variance again shows no significant difference among the remaining groups. Therefore, it is apparent that these amounts of trauma had little or no effect on mitosis in the intact kidney.

Influence of Renal Trauma and Homogenates on Mitosis in the Contralateral Organ

Saetren[1] demonstrated that kidney homogenates inhibited the compensatory hyperplastic response; however, when smaller amounts were given, these homogenates stimulated hyperplasia. In this part of the study we elected to examine the combined effect of trauma and homogenates. (This experiment was designed before the results of the trauma experiments were known.) The data (Table III) indicate that trauma and homogenates together have a depressing effect when compared with controls (trauma and suspending medium).

Experiment to Study the Effect of Post-Nephrectomy Serum

In this experiment, serum was obtained from Sprague-Dawley rats 42 hours after nephrectomy. It was pooled, and injected intraperitoneally into intact animals in doses of 3 ml./100 g. body weight. The animals were sacrificed 42 hours later. In addition, some animals in the group were subjected to unilateral nephrectomy, and the remaining kidneys examined at 42 hours. The data indicate that these animals were capable of a hyperplastic response (Table IV). However, the animals that received nephrectomy serum did not demonstrate any significant response when compared with animals receiving normal serum. Thus, we did not obtain any evidence of the existence of a humoral factor.

TABLE IV.—The Effect of Serum on DNA Synthesis in the Kidneys of Rats

Material injected	Condition of recipient	Number of rats	Number of labels per 50 fields*
Nephrectomy serum	Intact	7	14.3 ± 3.4
Normal serum	Intact	5	14.2 ± 4.2
Physiological saline	Unilateral nephrectomy	6	58.7 ± 7.2

*Average ± standard error of the mean.

Discussion

After unilateral nephrectomy, the time at which the peak in the hyperplastic response occurs is variable. In some studies it was found at 48 hours.[1, 11] Williams[3] found a peak at 40 hours, whereas Phillips and Leong[12] reported that a peak was reached at 36 hours. Our own data suggest that, in 10-week-old Sprague-Dawley rats, a higher incidence of mitosis occurs at 42 hours than

at either 36 or 48 hours. Some of the difference observed may be explained by the different time intervals studied because, in some experiments, the time intervals examined were as long as 24 hours.[2] The magnitude of the response under these circumstances is less variable. Goss and Rankin[2] found that the mitotic activity at 48 hours was six times greater than that obained in sham-operated controls. Reiter and McCreight[13] reported a 4.5-fold increase in 3-week-old animals, and a 5.4-fold increase in 4-month-old animals. In our investigations, the response was six times greater in nephrectomized animals than in sham-operated controls. It should be noted that peaks, as measured by both labelling indices and mitotic indices, occurred at 42 hours. This is in agreement with the work of Phillips and Leong.[12]

Our results concerning the effects of renal trauma on the hyperplastic response conflict with those of Argyris and Trimble.[10] While we inflicted considerable trauma, no evidence of a stimulating effect was found. In these experiments, the trauma was confined to the renal cortex, whereas Argyris and Trimble may have been traumatizing the medulla as well. In addition, their work was done on mice.

Many have searched for a kidney-specific, mitosis-stimulating humoral factor in the serum after unilateral nephrectomy. Ogawa and Nowinski,[8] using kidney tissue culture, concluded that 42 hours after nephrectomy the serum of rats contained such a factor. Lowenstein and Stern[9] repeatedly injected nephrectomy serum and concluded that it contained a mitosis-stimulating factor. However, Williams,[5] Reiter and McCreight,[13] and Kurnick and Lindsay[4] all failed to demonstrate a mitosis-stimulating, humoral factor in post-nephrectomy serum. In our studies, although relatively large amounts of nephrectomy serum were injected (3 ml. of serum per 100 g. body weight), we did not demonstrate a significant mitotic response in the kidneys of animals receiving this serum.

Summary

The literature on compensatory renal hyperplasia has been reviewed briefly. Some of the factors said to influence the response were re-examined. This study revealed that renal trauma did not stimulate mitosis in the contralateral organ. We were unable to demonstrate a kidney-specific, mitosis-stimulating factor in the serum of animals following unilateral nephrectomy.

References

1. Saetren, H.: A principle of auto-regulation of growth; production of organ specific mitose-inhibitors in kidney and liver, *Exp. Cell Res.*, **11:** 229, 1956.
2. Goss, R. J. and Rankin, M.: Physiological factors affecting compensatory renal hyperplasia in the rat, *J. Exp. Zool.*, **145:** 209, 1960.
3. Williams, G. E. G.: Some aspects of compensatory hyperplasia of the kidney, *Brit. J. Exp. Path.*, **42:** 386, 1961.
4. Kurnick, N. B. and Lindsay, P. A.: Mechanism of compensatory renal hypertrophy. Possible role of serum factor, *Lab. Invest.*, **17:** 211, 1967.
5. Williams, G. E. G.: Studies on the control of compensatory hyperplasia of the kidney in the rat, *Lab. Invest.*, **11:** 1295, 1962.
6. McCreight, C. E. and Reiter, R. J.: Renal compensatory hyperplastic response in hypophysectomized rats receiving various hormone supplements, *Anat. Rec.*, **151:** 384, 1965 (abstract).
7. Braun-Menendez, E.: Hypertension and the relation between body weight and kidney weight, *Acta Physiol. Lat. Amer.*, **2:** 2, 1952.
8. Ogawa, K. and Nowinski, W. W.: Mitosis stimulating factor in serum of unilaterally nephrectomized rats, *Proc. Soc. Exp. Biol. Med.*, **99:** 350, 1958.
9. Lowenstein, L. M. and Stern, A.: Serum factor in renal compensatory hyperplasia, *Science*, **142:** 1479, 1963.
10. Argyris, T. S. and Trimble, M. E.: The growth promoting effects of damage in the damaged and contralateral kidneys of the mouse, *Anat. Rec.*, **150:** 1, 1964.
11. Rollason, H. D.: Compensatory hypertrophy of the kidney of the young rat with special emphasis on the role of cellular hyperplasia, *Anat. Rec.*, **104:** 263, 1949.
12. Phillips, T. L. and Leong, G. F.: Kidney cell proliferation after unilateral nephrectomy as related to age, *Cancer Res.*, **27:** 286, 1967.
13. Reiter, R. J. and McCreight, C. E.: Failure to demonstrate a humoral factor controlling compensatory renal hyperplasia, *Anat. Rec.*, **148:** 396, 1964 (abstract).

Résumé

L'augmentation du volume du rein restant après néphrectomie unilatérale est en partie attribuable à l'hyperplasie. Ceci a été démontré en comptant les éléments mitotiques des cellules des tubules du cortex rénal. On a noté un sommet 42 heures après la néphrectomie unilatérale. Les auteurs essayent de savoir si cette réaction est provoquée par un facteur humoral spécifique qui stimulerait la mitose des cellules rénales. De larges doses de sérum (3 ml/100 g de poids) provenant de

rats néphrectomisés et de rats témoins chez qui on avait fait un simulacre de néphrectomie, ont été injectées dans le péritoine de rats intacts. De la thymidine marquée au tritium (0.7μc/g de poids) a été injectée dans le péritoine de ces bêtes quatre heures avant qu'elles eurent été sacrifiées (soit 42 heures après l'injection de sérum). Les tissus ont été préparés d'après les techniques autoradiographiques classiques et les cellules marquées ont été comptées. Dans les reins excisés, rien n'a démontré l'existence d'un facteur humoral. Chez d'autres animaux, le cortex a été l'objet de multiples lésions artificielles créées par de nombreuses piqûres pratiquées à l'aide d'une aiguille de calibre 20. Cette expérience n'a pas accéléré la mitose.

Les auteurs concluent en disant qu'il faudrait établir de longues périodes de circulation croisée entre le rat néphrectomisé et un receveur intact pour savoir si l'hyperplasie rénale de compensation relève d'un facteur humoral.

SERUM CALCIUM AND TUMOUR METASTASES

Abnormalities of calcium and phosphate metabolism in patients with advanced malignant disease have been recognized for the past 30 years. In 1942, Farrow and Woodard clearly delineated hypercalcemia as part of a syndrome in five women with osteolytic metastases of breast cancer. During the 1950's, many additional reports of hypercalcemia associated with malignant disease appeared and it is now conceded that hypercalcemia may occur with carcinoma or sarcoma when it appears almost anywhere in the body. In addition, recent reports have demonstrated the occurrence of this abnormality associated with lymphomas and leukemia.

Since 99% of the body's calcium is located in bone, it would be natural to assume that hypercalcemia could be attributed to involvement of the bone by tumour. In 1956, Connor, Thomas and Howard reported two patients with carcinoma of the lung and associated hypercalcemia who responded to resection of their tumour with a fall in serum calcium levels to normal. Regrowth of the tumour was associated with elevation of the serum calcium levels. Plimpton and Gellhorn subsequently reported a number of instances of hypercalcemia unassociated with evidence of bone destruction. As early as 1889, Paget noted that some areas of osteolysis in patients with breast cancer contained no tumour deposits at autopsy. Despite this evidence, it is possible that the hypercalcemia is produced by bone destruction because the presence of metastases may be occult. An analysis of patients with advanced breast cancer at the University of California revealed that, of those followed with repeated measurements of serum calcium every six weeks, 43% would at one time or another demonstrate hypercalcemia.

Hypercalcemia associated with breast cancer is now a frequent observation. Since there is a preponderance of metastases to bone in this disease, it is natural to assume that the breakdown of the bone by tumour releases sufficient calcium into the blood stream to be responsible for this. Calcium is cleared readily by the kidneys, and measurement of the urinary calcium excretion has been used by a number of investigators as an indication of the degree of bone breakdown and, therefore, progress of the disease.

Evaluation of the patient's response to therapy may be difficult by clinical and roentgenographic means. Progression of disease may take many weeks to become evident on roentgenograms of the bones, for example. Where average longevity is measured in months, this may represent a significant portion of the patient's remaining life expectancy. Certainly if rapid means were available to determine the response to therapy, then ineffective drugs could be assessed and discontinued within a short period of time. It seemed logical that an increase in urinary calcium excretion might then represent an ideal test for progression of disease. Contrary to this opinion, however, it was found that the mean urinary calcium excretion of untreated patients without osseous involvement was not significantly different from such excretion in patients with progressive osteolytic disease. Furthermore, patients evaluated at different stages of their disease showed that those undergoing androgen therapy had similar mean urinary calcium excretions regardless of whether their disease was progressing without osseous involvement, regressing without osseous involvement, progressing osteolytic, regressing osteolytic, or static disease. In addition, there was no change in the urinary calcium excretion before and during osteolytic progression, osteolytic regression, and progression without osteolytic disease in individual patients without change in the mode of therapy.—Gardner, B.: The relation between serum calcium and tumour metastases, *Surg. Gynec. Obstet.*, **128**: 369, 1969.

HORMONAL STIMULATORY MECHANISM PRODUCING GASTRIC HYPERSECRETION FOLLOWING MASSIVE SMALL-INTESTINAL RESECTION*

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STASSOFF[1] in 1914 showed that gastric hypersecretion followed extensive small-intestinal resection. Landor and Baker[2] showed that Heidenhain pouch hypersecretion in dogs occurred immediately after the small intestine was either resected or bypassed and that this Heidenhain pouch secretion returned to normal levels as soon as normal intestinal continuity was restored in these dogs. Ivy and Bachrach[3] showed that, in dogs, gastric-pouch secretion increased considerably after the Mann-Williamson operation. It appears that gastric hypersecretion does not necessarily depend upon resection of the small intestine. This may lead one to speculate that a gastric inhibitor is removed because the food has very limited contact with the intestinal mucosa, or that a gastric secretogogue is produced by the terminal ileum or colon when these portions of the intestinal tract come in contact with partly digested food or chyme which may still have an acid pH. Because the hypersecretion that follows intestinal resection in experimental animals can be quantitated in dogs with Heidenhain (denervated) pouches, we postulate that a hormone or hormones are involved. We have shown earlier[4] that thoracic-duct lymph plays an important part in the transport of gastric secretogogues. The present study was undertaken to see if thoracic-duct lymph played any role in the gastric hypersecretion that followed enterectomy.

METHOD

Six mongrel dogs weighing 20 to 25 kg. were used. However, in the last phase of this experiment, two dogs died following anesthesia; hence, this report concerns our studies on four dogs. In these dogs Heidenhain pouches, constructed using an aseptic technique, were drained through stainless steel cannulas into rubber balloons. After operation, the dogs were maintained on intravenous fluids for two days, and on a milk and Pablum diet for four to six days. Subsequently, they were maintained on a normal vivarium diet of water, commercial dog food and bread. After a minimal recovery period of two weeks, the first phase of the experiment began (Table I).

TABLE I.—THE SEQUENCE OF SECRETORY STUDIES DONE TO DETERMINE THE EFFECT OF THORACIC-DUCT-LYMPH DIVERSION AND REINFUSION

Phase I.	Dog awake. 24-Hour study
	Part 1. Heidenhain pouch
	Part 2. Massive enterectomy
Phase II.	Dog anesthetized. 8-Hour study
	Part 1. Massive enterectomy
	Part 2. Thoracic-duct-lymph diversion and reinfusion

In the first phase, 24-hour Heidenhain pouch secretions were studied in the conscious dogs. After these studies were completed in dogs fasted for 24 hours and dogs fed normally over a 24-hour period, the small bowel was resected, leaving two feet of proximal jejunum and one foot of distal ileum. Intestinal continuity was re-established using a one-layer, end-to-end anastomosis. During the recovery period (a minimum of two weeks), some of the dogs developed diarrhea which was readily controlled by giving 20 g. $CaCO_3$ daily for several days. No studies were done until the dogs recovered and the diarrhea had been controlled for at least one week. Studies of 24-hour preprandial and postprandial Heidenhain pouch secretions were repeated in these conscious, enterectomized dogs.

In the second phase of the experiment, the hourly Heidenhain pouch secretions were measured for eight hours in these

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four pentobarbital (Nembutal) anesthetized, enterectomized dogs. This phase was divided into two parts: an enterectomy control and an enterectomy lymph-diversion study.

Part 1 (the control).—The pouch secretions were collected hourly during a two-hour fasting base-line period, and a six-hour postprandial period. Through an orogastric tube, the dogs were fed 120 g. of commercial dog food (Tops Dog Food) homogenized in 60 ml. of distilled water.

Part 2 (the lymph-diversion study).—The pouch secretions were collected hourly for eight hours after Nembutal anesthesia and a right lower thoracotomy. After the two-hour fasting base-line period was over, the dogs were given an intragastric infusion of homogenized dog food. Four hours after starting the gastric secretory studies, the supradiaphragmatic portion of the thoracic duct was cannulated through the thoracotomy and, for the duration of the experiment, the lymph collected into a sterile cylinder to which 2.5 mg. of heparin had been added. Six hours after the experiment started, the lymph that had been collected during the previous two hours was reinfused, under sterile conditions, into a systemic vein over the remaining two hours. Two of the dogs were sacrificed immediately after the lymph-diversion study was completed, and two were kept alive with chronic thoracic-duct-lymph fistulas.

Results

Fig. 1 shows the mean, total, 24-hour Heidenhain pouch secretion in four conscious, fasted and meat-fed dogs before and after enterectomy.

During the 24-hour fast, the mean total secretory volume increased from 50.1 ml. in the intact dogs to 88.6 ml. after enterectomy. In dogs fed a normal laboratory diet, the mean total 24-hour secretory volume increased from 56.7 ml. in intact dogs, to 206.7 ml. in dogs with massive small-bowel resection. These findings confirm that an 80% enterectomy increased gastric secretion when the dogs were fasted, and when they were fed a normal laboratory diet.

The mean secretory volumes during the

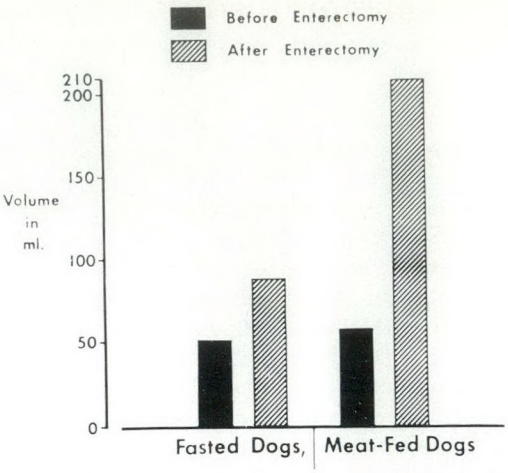

Fig. 1.—Twenty-four-hour Heidenhain pouch secretions in conscious, fasted and fed dogs, before and after 80% enterectomy.

fasting and postprandial periods in Nembutal anesthetized dogs are summarized in Fig. 2. In dogs with 80% of the small

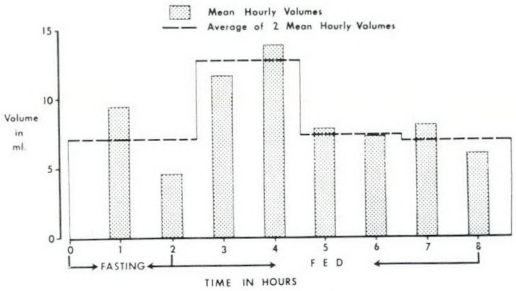

Fig. 2.—Mean hourly Heidenhain pouch secretions in anesthetized, enterectomized dogs, before and after feeding.

bowel removed, the average of the mean secretory volumes during the two-hour fasting base-line period was 7.05 ml.; this

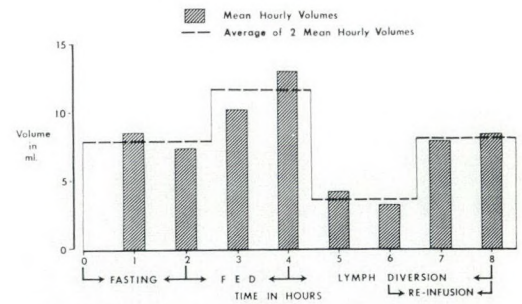

Fig. 3.—The effect of thoracic-duct-lymph diversion and lymph reinfusion on Heidenhain pouch secretion in meat-fed, enterectomized, anesthetized dogs.

rose to an average two-hour postprandial volume of 12.75 ml. The pouch secretion fell markedly over the next two hours to an average (two-hour) volume of 7.4 ml. This average decreased to 7.15 ml. over the last two hours of the experiment.

During lymph diversion (Fig. 3), the same procedure was repeated as in the antecedent control except that thoracic-duct lymph was diverted two hours after feeding and was continued throughout the experiment. The mean, total secretory volume during the two-hour base-line fasting period was 7.9 ml.; that is, slightly higher than in the control group. During the subsequent two-hour postprandial period, gastric secretion rose to 11.6 ml.—1.15 ml. less than during the corresponding period in the control group. In the two-hour period after thoracic-duct-lymph diversion, the

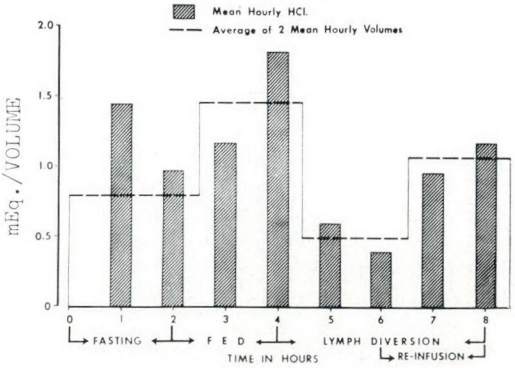

Fig. 4.—The effect of thoracic-duct-lymph diversion and lymph reinfusion on total acid production in meat-fed, enterectomized, anesthetized dogs with Heidenhain pouches.

pouch secretion averaged 3.6 ml., compared to 7.4 ml. in the control group. In the two-hour period during lymph reinfusion, the pouch secretion rose to 8.1 ml., compared to 7.15 ml. in the control group.

Fig. 4 shows the total acid, in mEq./volume, produced by the enterectomized dogs during the lymph-diversion study. In these dogs, acid production paralleled the volume of gastric juice secreted.

Discussion

Our findings confirm the results of other workers in this field: that a massive enterectomy causes gastric hypersecretion. In conscious dogs following an 80% enterectomy, there was a modest increase of gastric secretion in fasted animals and a marked increase in fed dogs. The differences in gastric secretion between fasted and fed dogs suggest that enterectomy stimulates an increased production of some gastric stimulant or, possibly, that it removes an intestinal gastric inhibitor.

In enterectomized dogs, thoracic-duct-lymph diversion decreased Heidenhain pouch secretion by more than one-half. Reinfusion of lymph increased this secretion by 125% during comparable two-hour periods; that is, to levels that were slightly higher than obtained in these dogs before lymph diversion. This finding suggests that some gastric secretogogue is being removed in the lymph. It is possible that this secretogogue is produced by the small intestine and that it acts like gastrin.

Summary

Massive enterectomy increases Heidenhain pouch secretion in fasting dogs and markedly increases postprandial pouch secretion. Complete diversion of thoracic-duct lymph in meat-fed dogs removes this postenterectomy hypersecretion. Reinfusion of diverted lymph increases pouch secretion. Extensive enterectomy may stimulate the production of a gastric secretogogue.

References

1. Stassoff, B.: Experimentelle Untersuchungen über die kompensatorischen Vorgänge bei Darmresektionen, *Bruns' Beitr. Klin. Chir.*, **89:** 527, 1914.
2. Landor, J. H. and Baker, W. K.: Gastric hypersecretion produced by massive small bowel resection in dogs, *J. Surg. Res.*, **4:** 518, 1964.
3. Ivy, A. C. and Bachrach, W. H.: An abnormal mechanism for the excitation of gastric secretion in the dog, *Amer. J. Dig. Dis.*, **7:** 76, 1940.
4. Yakimets, W. W. and Bondar, G. F.: The effect of complete thoracic-lymph diversion on gastric secretion in dogs, *Canad. J. Surg.*, **10:** 218, 1967.

Résumé

On savait déjà qu'une hypersécrétion gastrique suit une entérectomie massive. La présente étude a pour objet de préciser le point de savoir si la lymphe du canal thoracique joue un rôle dans la régulation de l'hypersécrétion gastrique qui survient après résection massive du grêle. Nous avons employé, à cet effet, quatre chiens bâtards. La phase I de notre étude consistait, après

un jeûne de 24 heures, à étudier la sécrétion gastrique post-prandiale sur un petit estomac de Heidenhain; ces mesures ont été reprises après entérectomie massive. Ceci a permis de confirmer l'existence d'une hypersécrétion gastrique après résection intestinale.

Dans la phase II de l'étude, nous avons utilisé les mêmes chiens entérectomisés, mais nous avons mesuré la sécrétion à intervalle d'une heure sous anesthésie au pentobarbital (Nembutal) pendant une période de huit heures, et dans les conditions du jeûne et de l'alimentation. Une période de stabilisation de deux heures (période de contrôle) précédait le repas. Dans la seconde partie de la phase II, nous avons recommencé l'expérience de la première partie et, en outre, à la fin de la période de stabilisation de deux heures, nous avons introduit une canule dans le canal thoracique par voie de thoracotomie. Nous avons recueilli pendant quatre heures la lymphe du canal thoracique et l'avons réinjectée par voie I.V. pendant les deux dernières heures de l'expérience. L'hypersécrétion gastrique a été supprimée par la diversion de la lymphe et a réapparu quand nous avons réinfusé la lymphe. Nous croyons pouvoir conclure de ces expériences qu'une entérectomie massive est capable de stimuler la production d'un sécrétagogue gastrique.

NEEDLE ASPIRATION OF BREAST CYSTS

Microcysts are present, at one time or another, in most women, from menarche to menopause. Dominant macrocysts of the breast, single or multiple, occur in many women and lead them with fear to a physician. Several choices remain open to the physician. He may reassure the patient that these masses are cystic, and follow-up examination for a period of time would be desirable to see what, if any, changes occur. Mammography and thermography may be included as adjunctive measures. On the other hand, he may insist on immediate biopsy and removal of the cyst, or cysts, to rule out adenocarcinoma. In our opinion, the best choice lies with needle aspiration performed as an office procedure. If the cyst completely disappears, he can assure his patient that this was not cancerous and that biopsy is not required. Follow-up examinations are mandatory, and the patient is allowed to go home with a considerably improved outlook and to return for re-evaluation in two weeks. She is then seen every six months for routine breast examination.

One objection to needle aspiration has been the fear of spread of malignant cells into the needle tract and into the blood stream and lymphatics. In analyzing 67 patients, in whom needle aspiration was unsuccessful and in whom subsequent biopsy of the lesions proved malignant, it was found that the five-year survival rate was 89%. This result would seem to indicate that no harm was done to the tumour by this technique.

The longest interval between aspiration and operation was 34 days. The average delay was 12 days. When aspiration is unsuccessful, we prefer that definitive operation be delayed no more than two weeks; however, we have no evidence that a somewhat longer delay has affected the survival rate in our series. The needle tract was excised at the time of mastectomy in each case. No instance of implantation of tumour cells was found in the needle tract.

It is our belief that there is no proved relationship between the presence of fibrocystic disease and cancer of the breast. That is not to say, however, that a carcinoma cannot be concurrent in the breast near the cyst or in an entirely different location, in the opposite breast, or subsequently develop.

According to Vanden Berg, "Anyone not trained, skilled, and experienced to do radical breast surgery should do neither a simple puncture nor a biopsy." It is preferable that the physician who performs the aspiration be equipped to handle the situation, when fluid is not returned. However, if the alternative to aspiration is watching a mass several months to see if it is cystic or solid, then we would rather that aspiration attempts be made by the family physician, who most often sees these patients initially. With the issue then settled, arrangements for referral could be made promptly, if necessary. It would seem reasonable that any physician, with experience in breast examination, could undertake this procedure, if he abides by the simple rules set up to avoid errors with its use.—Rosemond, G. P., Maieo, W. P. and Brobyn, T. J.: Needle aspiration of breast cysts, *Surg. Gynec. Obstet.*, **168**: 351, 1969.

SURGICAL TECHNIQUE

KNEE-CHEST SUPPORT FOR LUMBOSACRAL OPERATIONS

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THE clinical investigation described in this paper was undertaken: (1) to determine the advantages and dangers of the knee-chest position during operation, and (2) to develop a simple operating room support that would render the knee-chest position perfectly safe.

THE KNEE-CHEST SUPPORT

The support used during this investigation was conceived in the Hotel-Dieu Hospital, Montreal and is recommended because of its great simplicity and adaptability to any operating room table. The support is described in detail because it may be fabricated easily in any tool shop and need not be purchased (Fig. 1).

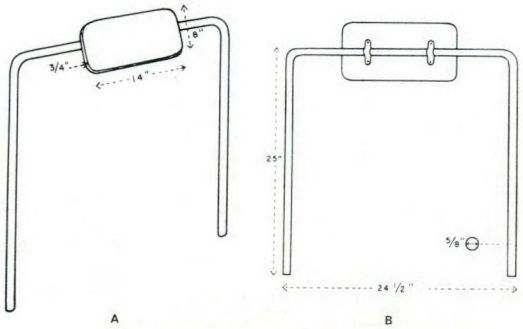

Fig. 1.—The knee-chest support.

A solid metal rod (⅝ of an inch in diameter) is bent in the form of the letter "U" (the vertical sections should be 25 inches long and the transverse bar 24½ inches). The buttock support, a padded wooden slab measuring 8 x 14 inches, is fixed to the horizontal bar by two metal brackets. The diameter of the metal rod (⅝ of an inch) is that of the common drape support used by anesthetists to shield the head and neck area during operation. Thus, the vertical bars of the support can be fixed to the operating room table using the sockets that normally receive the drape support or the kidney support.

POSITIONING OF THE PATIENT

The vertical bars of the support are first inserted in the sockets on either side of the operating table and the support is then laid flat at the foot end of the table (Fig. 2A and E). The patient is anesthetized on the stretcher, intubated and then rolled on to the operating room table, face down with the legs over the knee-chest support (Fig. 2B and F). The patient is then lifted into the knee-chest position; the support is swung under the buttocks and locked in place (Fig. 2C). It may be necessary to put a pillow under the chest to prevent cervical hyperextension (Fig. 2G). The table is then tilted in a reverse Trendelenburg position so that the patient is almost sitting on the buttock support (Fig 2D and G). Two transverse adhesive tapes (Fig. 2H) are then applied over the knees and buttocks of the patient and fixed to the operating room table to insure a perfectly safe and stable position: a two-inch adhesive tape is first applied over the buttocks, down either thigh and then to the table to prevent the surgeon, or the assistant, from pushing the patient laterally off the support; a second tape is applied over the anterior aspect of the flexed knees and on to the table to prevent hip abduction. With these two tapes in place, the patient's position is secure; final adjustments should confirm that the calves and the abdomen are completely free. It will be shown that these two points (the free calves and abdomen) are crucial to the use of this position.

DANGERS OF THE KNEE-CHEST POSITION

It is important to avoid excessive hip

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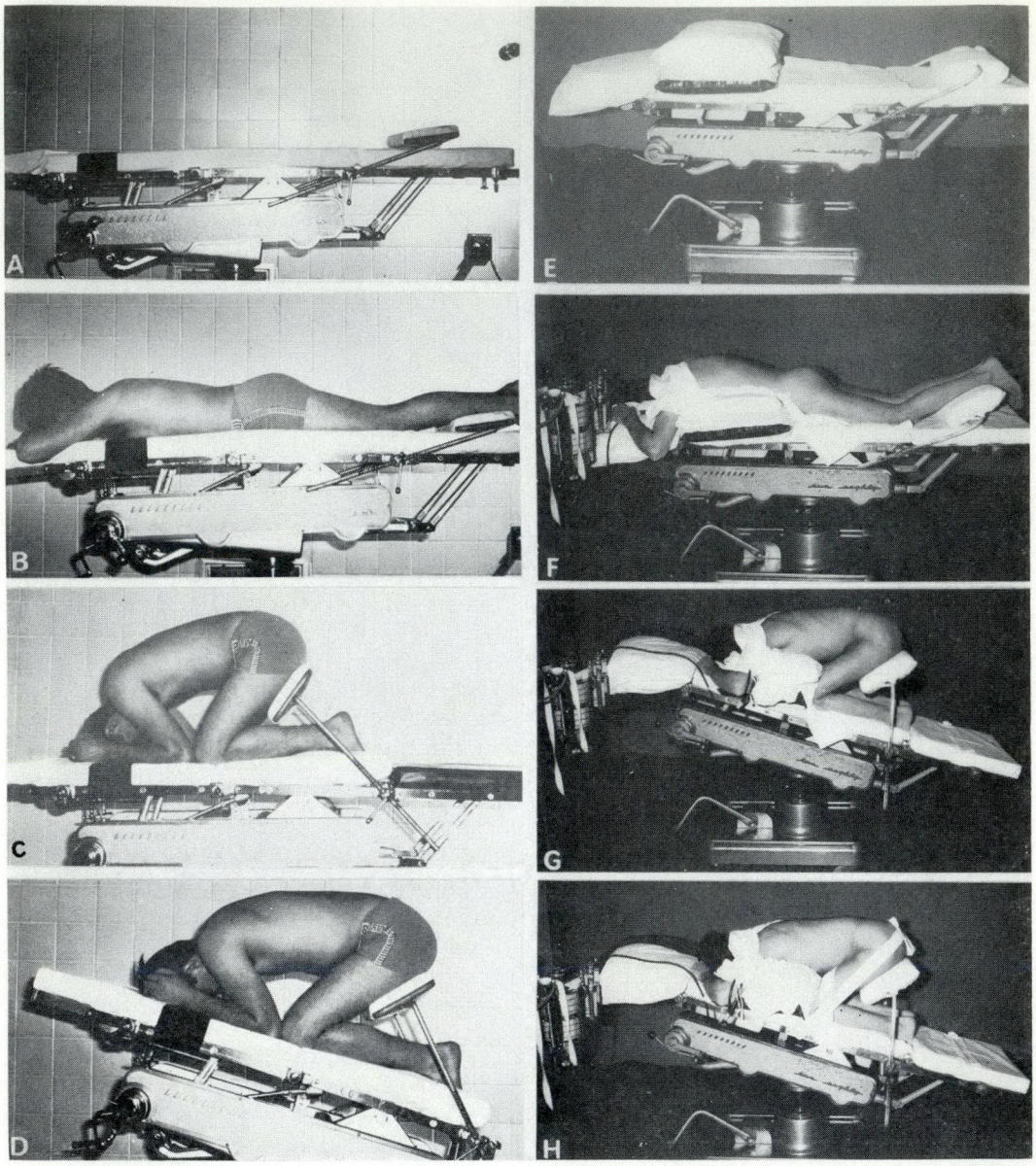

Fig. 2.—Positioning of the patient. (1) After intubation the patient is rolled on to the operating table, over the pelvic support (B and F). (2) The knee-chest position is achieved by lifting the pelvis and flexing the hips and knees (C). (3) The pelvic support is rotated under the pelvis, lifting the buttocks away from the calves; note that the curvature of the lower lumbar spine is not excessive (compare Figs. 2C, 3A and 3B). (4) The table is tilted gently so that the patient is almost in a sitting position (D and G); it is better to keep the chest slightly lower than the buttocks so that gravity favours the drainage of the inferior vena cava into the left auricle (see Fig. 4). (5) Two adhesive tapes are applied over the knees and buttocks to insure complete stability (H).

and knee flexion; prolonged and marked hip flexion may cause chronic and persistent postoperative pain in the hip and quadriceps area. If hip hyperflexion is avoided, knee hyperflexion, a far greater danger, will never occur. The great danger of the knee-chest position is vascular impairment distal to the knees, either due to vascular kinking in the popliteal space or to calf compression. This possible complication has been a most understandable and justifiable reason for the relative unpopularity of the knee-chest position. Gordon and Newman[1] described a patient who

died following an operation done in the knee-chest position. Death was due to lower nephron nephrosis secondary to bilateral calf-muscle ischemia. The ischemia was due to prolonged compression during a three-hour laminectomy, which had been performed with the patient literally sitting on his calves with the hips and knees in hyperflexion (Fig. 3B and C).

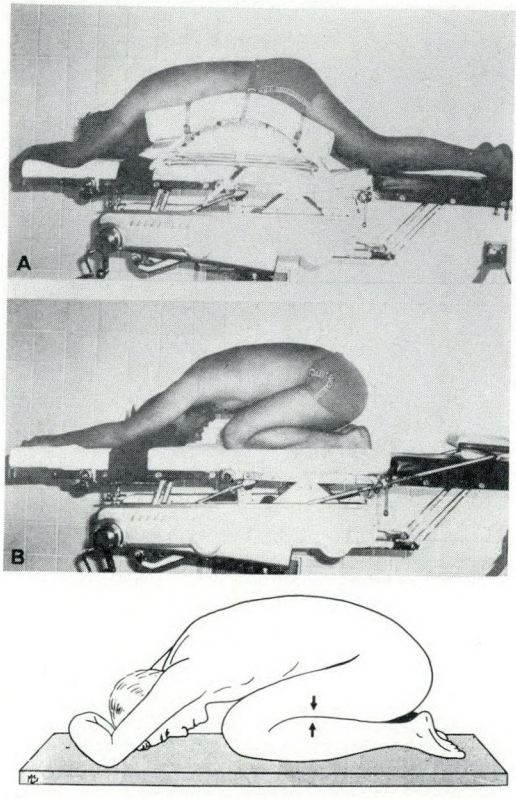

Fig. 3.—Comparable lumbar flexion with the commercial frame (A) and the knee-chest support (Fig. 2C). The tuck position (B and C) or the unsupported knee-chest position permits greater lumbar flexion but it is not recommended since the excessive knee and hip flexions are dangerous.

The tuck position recently reported by Wayne[2] shares some of the physiological advantages of the knee-chest position but is not recommended because of possible lower-limb vascular impairment. With the support described here, vascular embarrassment is almost impossible. Once the patient is installed on the support, it is easy to insert a hand into the popliteal space of the flexed knees to verify that the position is perfectly safe. The temperature of the feet should, nonetheless, be verified at regular intervals during the operation.

Advantages of the Knee-Chest Position

The great advantage of the knee-chest position is that the patient's abdomen is completely free. Once the patient is on the knee-chest support, it is easy to confirm that the abdominal wall is relaxed, free of any contact or pressure, and gently convex. The benefits of this unharassed abdomen are both respiratory and vascular; respiratory function is at its maximum and there is no pressure on either the viscera or the inferior vena cava. The combined effect of these two advantages is that venous drainage is so efficient that, at the time of laminectomy, the epidural veins are flattened and almost empty. Hence, visibility is considerably enhanced and any bleeding is easily controlled, because it is always minimal and never under any pressure. Indeed, the convex abdominal wall exerts a negative pressure on the inferior vena cava thus emptying the epidural veins which, in the knee-chest position, drain almost by gravity into the inferior vena cava. The improved drainage from the epidural veins was obvious at the time of operation. It was again confirmed with venograms of the inferior vena cava and by measuring the venous pressure within the inferior vena cava in unanesthetized patients in the laboratory (Table I).

The basis of the vastly improved epidural venous drainage is found in Batson's work. He demonstrated a valve-free communication between the epidural veins and the inferior vena cava; the direction of flow in this communication depends on the intra-abdominal pressure. The epidural veins drain normally into the inferior vena cava but the flow can be reversed temporarily (for example during coughing or forceful voiding), or longer by raising the intra-abdominal pressure.

In order to visualize the epidural veins during epidural venography, the intra-abdominal pressure must be raised by direct abdominal-wall compression; the venous drainage of the entire vertebral sys-

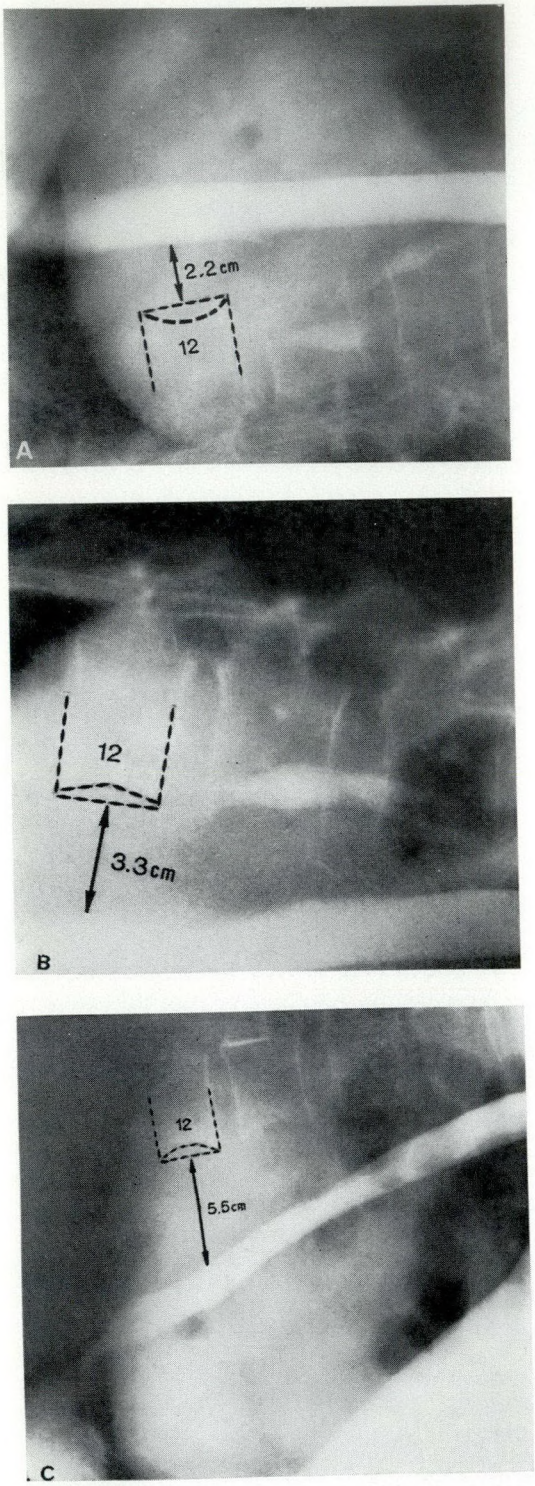

Fig. 4.—Venograms of the inferior vena cava in various positions: (A) with the patient supine; (B) using the lumbosacral frame; (C) using the knee-chest support. No direct pressure was exerted

tem is then reversed along the epidural veins and no dye enters the inferior vena cava. Similarly, Batson had reversed the drainage of the epidural veins by direct pressure on the abdominal wall; dye injected in the epidural vein of the penis could be seen to reach the cerebral venous system entirely via the epidural system.

For these reasons, the slightest rise in the intra-abdominal pressure or in the inferior vena caval system will increase the pressure in the epidural veins and, conversely, any decrease in the inferior vena caval pressure, or any measure that will relax the abdominal wall, will facilitate epidural drainage and collapse the epidural veins (Table I).

TABLE I.—Inferior Vena Caval Pressures in Various Positions

Position	*Inferior vena caval pressure (mm. Hg)*	*Valsalva's test (mm. Hg)*
Supine	10	—
Prone	11	74
Prone and longitudinal cushions	18	85
Knee-chest position without support	5	70
Knee-chest position with pelvic support	2-0	68
Knee-chest position with pelvic support and pillow under chest	2-0	78

The Valsalva test was performed in every position to verify the patency and function of the catheter in the inferior vena cava. Note that the venous pressure is lowest in the knee-chest position when the pelvis is supported and the abdominal wall is entirely free; a slight elevation of the chest wall did not modify the pressure. A negative intrathoracic pressure (using an artificial respirator) would be most beneficial with such a low pressure in the inferior vena cava.

A venogram (Fig. 4) of the inferior vena cava was performed with the patient held in the knee-chest position on our new support (Fig. 4B) and then repeated with the patient on a regular frame used for lumbosacral operations, i.e. lateral supports on

on the abdominal wall and the venous flow was never reversed; hence, the epidural veins were never coloured by the injection of dye in the iliac veins. The diameter of the inferior vena cava is decreased in the knee-chest position and reflects the much lower venous pressure; another factor which contributed to a more rapid emptying into the left auricle is gravity; it can be seen that the left auricle is much lower than the epidural veins in the knee-chest position, as the vein falls away from the lumbodorsal spine.

the chest and abdomen (Fig. 4C). Although commercially available frames (Fig. 3A) are useful, they exert enough lateral pressure on the abdomen to affect the venogram and the inferior vena caval pressure (Fig. 4 and Table I).

Another benefit of the free abdominal wall in the knee-chest position is its influence on respiration and air exchange (the decreased intrathoracic pressure has obvious and immediate effects on venous drainage, contributing, by yet another mechanism, to the emptying of the epidural veins). The volume of air exchange was compared with an artificial respirator at a fixed pressure when the patient was (a) flat on his back, (b) on a lumbosacral frame and (c) in a knee-chest position (Table II). Blood samples were taken simultaneously to verify the influence of these various positions. Table II shows that, in order of efficiency, the optimal position was the knee-chest position, even better than when a patient is on his back, when the diaphragm must work against the weight of the abdominal viscera.

TABLE II.—Influence of Position on Ventilation

Patient	*Air exchange (l./5 min.)*	*P_{CO_2} (mm. Hg)*	*pH*	*HCO_3 (mEq./l.)*	*Arterial oxygen saturation (%)*
B.S.—44 years—228 lb.					
Supine	56.02	29.6	7.41	22.1	92.8
Prone	54.48	28.8	7.43	21.0	93.2
Supported knee-chest position	83.54	24.2	7.52	22.6	97.0
N.L.—38 years—169 lb.					
Supine	75.0	26.0	7.55	24.6	98.0
Prone	65.0	27.0	7.50	24.0	95.0
Supported knee-chest position	78.0	24.0	7.58	23.0	98.8

An artificial respirator was used and the machine adjustments were not modified during anesthesia; the changes in ventilation were due to the altered position of the patient. Note the increased air exchange and the improved oxygen saturation in the supported knee-chest position. The beneficial influence of the supported knee-chest position on respiration is more obvious in obese patients.[3]

Another advantage of the knee-chest position is that the viscera and the major vessels fall forward, away from the intervertebral discs, and hence away from any surgical instrument which may inadvertently pierce the anterior longitudinal ligament and damage an intra-abdominal organ or vessel.

The Knee-Chest Position for Lumbar Laminectomies and Lumbar Fusions

It had been hoped that the knee-chest position would produce sufficient lumbar-spine flexion to distract the posterior laminae and thus facilitate laminectomy. Lateral radiographs of the lumbar spine (Fig. 5) were taken with the patient prone on a table, on a lumbosacral frame, and finally in the knee-chest position. Radiographs confirmed what could be observed at the time of operation, namely, that compared to a commercial lumbosacral frame, the knee-chest position does not significantly increase lumbar-spine flexion. The knee-chest position works because of further hip flexion rather than increased lumbar flexion; lumbar flexion was mildly increased at the L4-5 level, but never at the L5-S1 level. Hence, the lateral muscular mass is not under undue tension in the knee-chest position and sufficient muscular retraction is possible to permit bilateral-lateral fusion.

Because of the considerable vascular and respiratory advantages of the knee-chest position, this position and the frame have been used, without any complication, for all lumbar operations over the past nine years.

Wound closure is performed in the knee-chest position with great advantage; since the muscular, fascial and cutaneous structures are then sutured in their original anatomical relationships, postoperative lumbar mobility appears to be restored more rapidly and more completely.

Summary

A simple, adaptable, home-made knee-chest support has been described.

The advantages and the physiology of the knee-chest position have been investigated using inferior vena caval venograms,

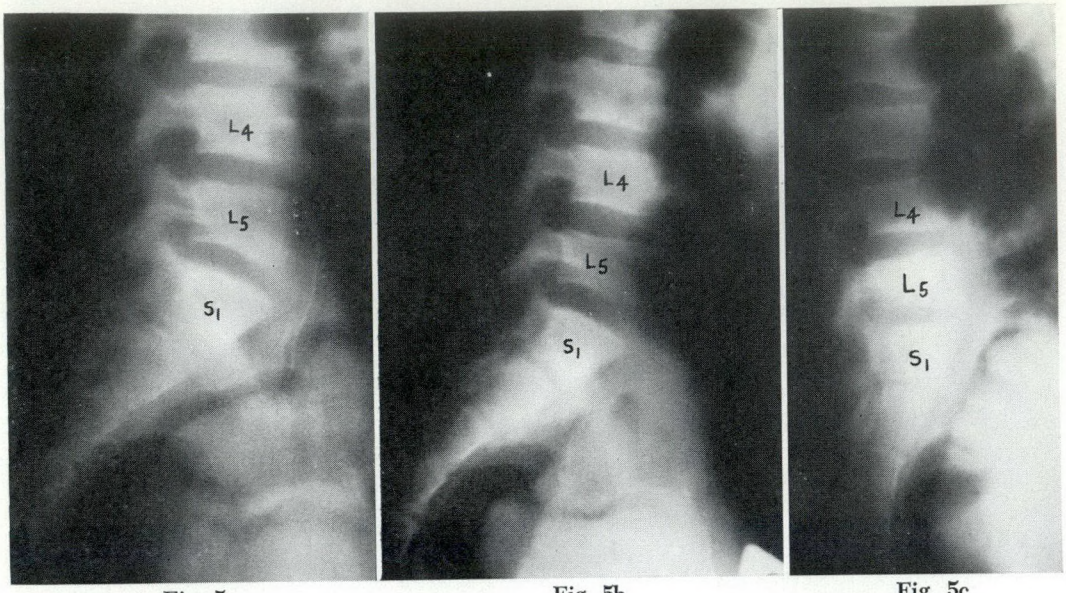

Fig. 5a Fig. 5b Fig. 5c

Fig. 5.—Lateral radiographs of the lumbar spine with the patient in the following positions: (a) supine; (b) on the lumbosacral frame; (c) on the knee-chest support. The knee-chest position does not provide much more lumbar flexion than the commercial lumbosacral frame (Figs. 2C and 3A); the L5-S1 space is unaltered. Lumbar fusions and wound closure can be achieved with ease in the supported knee-chest position.

inferior vena caval pressures and air-exchange measurements. The knee-chest position is recommended for lumbar laminectomies and fusions.

The authors are pleased to acknowledge the excellent work of Misses J. Valentine and Madeleine Gagnon and Mr. Jacques Doyon.

References

1. Gordon, B. S. and Newman, W.: Lower nephron syndrome following prolonged knee-chest position, *J. Bone Joint Surg.* [*Amer.*], **35A:** 764, 1953.
2. Wayne, S. J.: The tuck position for lumbar disc surgery, *J. Bone Joint Surg.* [*Amer.*], **49A:** 1195, 1967.
3. Tarlov, I. M.: The knee-chest position for lower spinal operations, *J. Bone Joint Surg.* [*Amer.*], **49A:** 1193, 1967.

Résumé

Les auteurs ont étudié les avantages physiologiques et les dangers de la position genu-pectorale. Les dangers peuvent être évités si l'on emploie un support spécial minutieusement décrit de façon que d'autres chirurgiens puissent le faire copier par leurs hôpitaux respectifs.

Avec des veinogrammes de la veine cave inférieure, les lectures de la pression à l'intérieur de la veine cave inférieure et différentes études d'échanges gazeux à l'appui, les auteurs prouvent que la position genu-pectorale est physiologiquement sans danger et avantageuse. Elle peut être employée pour toutes sortes d'opérations lombo-sacrées.

A SIMPLE FRAME FOR OPERATIONS ON THE LUMBAR SPINE

D. E. HASTINGS, M.D., F.R.C.S.[C],* *Toronto, Ont.*

In operations on the lumbar spine the patient is placed in one of three conventional positions: prone, lateral recumbent and knee-chest.[1-4] Each has advantages and disadvantages.

When the patient is in the prone position over bolsters or a tubular frame, the degree of flexion or extension of the lumbar spine is relatively well controlled. Often, however, the associated abdominal compression interferes with respiration. This abdominal compression may produce a complete block of the inferior vena cava.[5] Then the venous return, diverted through Batson's plexus of veins,[6] may cause engorgement of the epidural veins. The troublesome local hemorrhage that may result may prevent an adequate exploration of the involved spinal level.

In the lateral recumbent position, the patient respires freely and venous return is not blocked. However, in this position wide laminectomy and, especially, spinal fusion to the tips of the transverse processes are difficult.

The major advantage of the knee-chest position is that the lumbar spine is flexed and the interlaminar spaces are opened widely. The patient's respiration is satisfactory and no pressure is exerted on the inferior vena cava. Hyperflexion, however, tends to increase tension on nerve roots which may already be quite tight due to disc herniation. Full flexion at both hips and knees is required and may impede venous return from the legs, produce stasis and predispose to deep-vein thrombosis.[7] Finally, in spite of careful positioning, the patient is often unstable. This paper describes a simple frame to stabilize and improve the knee-chest position.

The Frame

In an attempt to combine the advantages of the knee-chest position, with increased stability and better control of flexion and extension, the frame described in this paper was devised (Fig. 1). This wooden frame has an adjustable seat and a removable cross-bar to add stability. It can be attached to a standard operating room table. The degree of flexion or extension of the lumbar spine is controlled by changing the height of the rest that supports the patient's chest.

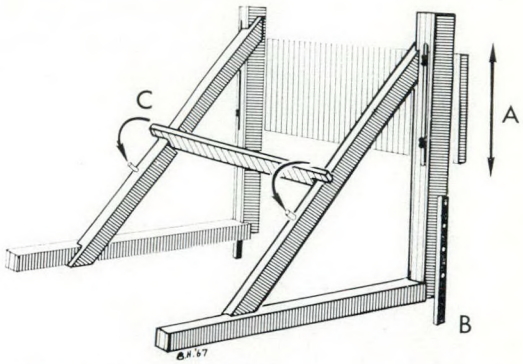

Fig. 1.—The frame. The seat is adjusted by wing nuts (A). The frame is fastened to the table by a standard metal mount (B). The adjustable cross piece (C), which adds stability, is inserted about patient's feet.

Technique

The patient is anesthetized and intubated on a stretcher in the supine position. The height of the chest rest is then determined. For routine laminectomy, a box four inches high covered with a pillow supports the patient in a relatively neutral position. If the patient is to be fused in extension, a higher box is selected—up to 18 inches in height. The patient is placed in the knee-chest position with the table horizontal, hips and knees being flexed to

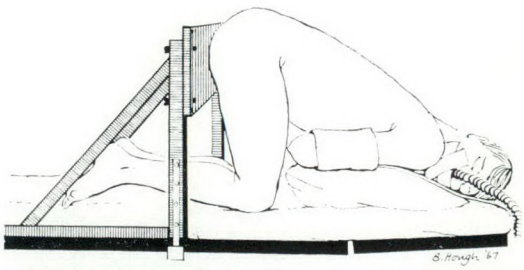

Fig. 2.—Patient in the knee-chest position with the table horizontal.

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approximately 100°. The frame is then placed approximately three inches behind the buttocks and the seat adjusted to the appropriate height (Fig. 2). The table is tilted into the reverse Trendelenburg position so that the buttocks come to rest firmly upon the seat, and the lumbar spine is then horizontal (Figs. 3 and 4).

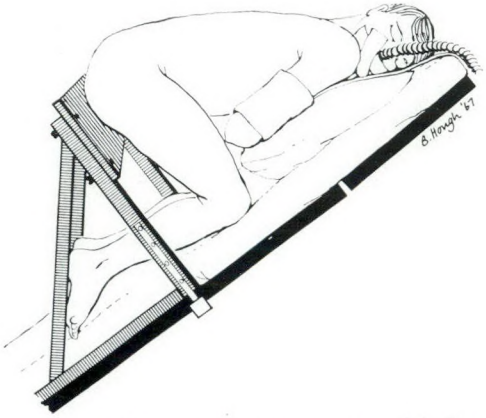

Fig. 3.—Table in the reverse Trendelenburg position brings the spine to the horizontal and stabilizes the patient against the seat.

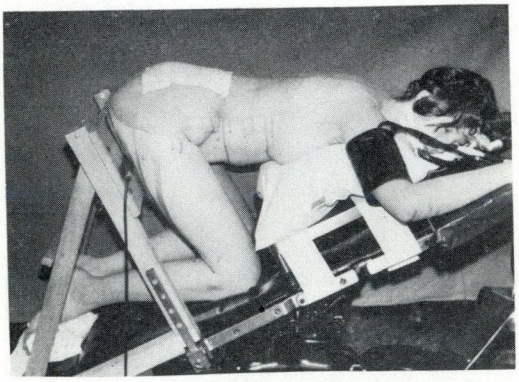

Fig. 4.—The frame in use. Note that the abdomen is not compressed.

Discussion

Since we adopted this frame, we have reduced to a minimum hemorrhage during spinal operations: 50 laminectomies and discotomies were done without significant blood loss. No transfusions were required and we did not encounter epidural bleeding. In 10 patients we carried out two-level spinal fusion extending to the tips of the transverse process; in only one of these was transfusion required.

The only complication we encountered with this frame was hypotension during and after the operation. This appeared to be due to pooling of blood in the dependent lower extremities and was most common in patients over 40 years of age. Wrapping the patient's legs snugly with tensor bandages before positioning appeared to control this hypotension.

Summary

A simple frame for operations on the lumbar spine is described. This frame improves the standard knee-chest position by increasing stability and improving control of flexion and extension. In this position, the abdomen hangs free; the diaphragmatic excursion is full and there is no troublesome epidural hemorrhage.

I would like to thank Dr. H. Foster Smith of the Department of Anesthesia for his invaluable help in the design of this frame and the carpenter's shop of the Wellesley Hospital, Toronto for its construction.

The illustrations were prepared with the aid of Miss M. Bliss of the Wellesley Hospital Photography Department.

References

1. Armstrong, J. R.: Lumbar disc lesions, pathogenesis and treatment of low back pain and sciatica, 3rd ed., E. & S. Livingstone Ltd., Edinburgh, 1965, p. 257.
2. Tarlov, I. M.: The knee-chest position for lower spinal operations, *J. Bone Joint Surg.* [*Amer.*], **49A:** 1193, 1967.
3. Taylor, A. R. *et al.*: Posture and anaesthesia for spinal operations with special reference to intervertebral disc surgery, *Brit. J. Anaesth.*, **28:** 213, 1956.
4. Wayne, S. J.: The tuck position for lumbar disc surgery, *J. Bone Joint Surg.* [*Amer.*], **49A:** 1195, 1967.
5. Relton, J. E. S. and Hall, J. E.: An operation frame for spinal fusion. A new apparatus designed to reduce haemorrhage during operation, *J. Bone Joint Surg.* [*Brit.*], **49B:** 327, 1967.
6. Batson, O. V.: The function of the vertebral veins and their role in the spread of metastases, *Ann. Surg.*, **112:** 138, 1940.
7. Holscher, E. C.: Vascular complication of disc surgery, *J. Bone Joint Surg.* [*Amer.*], **30A:** 968, 1948.

Résumé

L'auteur a conçu un support très simple pour maintenir en position genu-pectorale les candidats à l'intervention. Il emploie ce support depuis trois ans à l'hôpital Wellesley de Toronto, et lui a trouvé de nombreux avantages. Ce support empêche la compression de l'abdomen et par conséquent n'entraîne pas la ventilation pulmonaire. La veine cave n'est pas obstruée et les veines épidurales sont généralement vides; l'hémorragie

qu'elles causent est minime et n'assombrit pas la pathologie locale.

Ce support est simple, est fait de bois et comprend un siège ajustable. Le patient est placé en position genu-pectorale et la table d'opération est inclinée en sens inverse du Trendelenburg, si bien que l'opéré s'appuie contre le siège.

L'auteur a fait 40 laminectomies en se servant de ce support, chaque fois la perte de sang a été inférieure à 150 cm^3; aucune transfusion n'a été nécessaire. Il n'y a pas eu d'hémorragie épidurale. Pour cinq cas de fusions des extrémités des apophyses transverses, un patient seulement a eu besoin d'une transfusion.

POSTOPERATIVE COMPLICATIONS IN ARTHRODESIS OF THE FOOT

The author has reviewed the postoperative follow-up studies of 246 patients, from 13 to 56 years of age, after arthrodesis of the foot carried out between 1946 and 1963. The arthrodesis was done for deformity of the foot after poliomyelitis, hemiplegia, spina bifida occulta, syringomyelia and polyneuritis, club feet, spastic flat feet, deformities resulting from fractures of the malleolae and tarsal bones, and rheumatoid arthritis.

After operation, in many patients the hematoma became absorbed slowly and as a rule a drain was left *in situ* until the second postoperative day. In some, necrosis of the skin was found, but in most it was confined to the edges of the skin. Occasionally sensory nerves were cut, leaving an area of so-called anesthesia which was only of minor clinical significance. Infection presented a real danger in these operations, but in most instances the objective of arthrodesis was obtained even in patients with infection within the bones of the foot. As a precautionary measure, penicillin was given during the first seven days after operation. In some, amputation of part of the extremity was necessary after an acute postoperative infection causing severe osteomyelitis.

Poor results after arthrodesis of the foot included absence of fusion, unstable feet, and persistent pain. Aseptic necrosis of the astragalus was observed in a number of patients. Pseudarthrosis was also observed and in several patients the preoperative deformity recurred. The lack of fusion was caused by aseptic necrosis, trophic changes, error in operative technique, and ineffective immobilization of the plaster cast.

Occasionally, it was observed that the deformity was surgically overcorrected. In some instances, the bone graft broke, and in a few instances degenerative arthroses developed in the adjacent joints.—Soren, A.: Complications postopératoires et mauvais résultats dans les arthrodèses du pied, *Rev. Chir. Orthop.*, **54:** 249, 1968.

ASEPTIC NECROSIS OF FEMORAL HEAD AND NON-UNION OF FEMORAL NECK

The authors treated avascular necrosis of the femoral head with and without non-union of the femoral neck, and with non-union of the femoral neck with insertion of tibial bone grafts into the neck and into the head. Phemister reported satisfactory results in 41 of 53 patients so treated. Reports concerning the use of hip prostheses for salvage of hip fracture complications indicate that 71.4% to 87% of the patients followed had overall satisfactory results. The authors reviewed 112 patients followed for 2 to 15 years. Of these, 57 had been followed for more than 5 years and 15 for 10 years.

If the non-union of the femoral neck is displaced, closed manipulations generally were carried out, but a varus position of less than 30° was considered acceptable. In only seven patients was open reduction necessary. The authors tried to get the bone grafts into the anterior superior aspect of the head and additional fixation was achieved using 5/32-inch threaded pins. Three-point-crutch walking within a few days was permitted until roentgenograms showed union. The average time for union was 10 to 12 months. Of 31 patients with minimum to moderate aseptic necrosis of the head with non-union of the femoral neck fracture, 26 showed satisfactory results. Of 46 patients with aseptic necrosis and nonunion, 35 achieved satisfactory results. Of 39 operations performed on 35 patients with avascular necrosis without non-union of the femoral neck, 30 produced satisfactory results. The average period of non-weight bearing was 12 months.—Bonfiglio, M. and Voke, E. M.: Aseptic necrosis of the femoral head and nonunion of the femoral neck; effect of treatment by drilling and bone grafting (Phemister technique), *J. Bone Joint Surg.* [*Amer.*], **50A:** 48, 1968.

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BOOK REVIEWS

(See also page 268)

AN ATLAS OF PELVIC OPERATIONS. 2nd ed. Langdon Parsons and Howard Ulfelder. 448 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $25.95.

The second edition of this atlas first published in 1953, though large, is now of a more convenient and manageable size. Other changes include modest alterations in format and text, and the inclusion of some additional operations, notably exenteration. Although most of the procedures described here lie within the limits of gynecological surgery, operations for gastrointestinal and urinary tract disease and complications that might be encountered in the pelvis are also included. The book contains an introductory chapter on general principles of gynecological surgery and "helpful hints" on hysterectomy but indications and preoperative and postoperative care following specific operations have purposely been excluded.

The most satisfying feature of this excellent atlas is the thorough presentation of operative technique in a complete spectrum of minor and major operations by clear line drawings, which are accompanied by a lucid, concise, descriptive text. Rather than describing a number of probably equally useful techniques, the authors call upon their wide experience in offering a single proved method. The interest of the authors in malignant disease is reflected in a thorough consideration of the radical operations for carcinoma of the cervix and vulva.

This atlas is a valuable edition to any medical school or hospital library where the sound philosophy and clarity of the authors will guide residents and gynecological surgeons.

CLINICAL MANAGEMENT OF SHOCK. Surgical and Medical. Robert M. Hardaway, III. 599 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $26.50.

This practical text on shock, which is designed for reference at the bedside, acknowledges recent sophisticated advances but fortunately does not suggest that the problem is solved. This book is based on laboratory and clinical studies made by the Division of Surgery of the Walter Reed Army Institute of Research and its important hospital affiliations in the United States and South Vietnam. The considerable success of the modern treatment of shock has been of immense importance to the military.

The book is divided into two sections: the first, which is concerned with basic concepts, emphasizes the importance of disseminated intravascular coagulation (DIC) in all forms of experimental and clinical shock. While the arguments are convincing, even stimulating, this reviewer found it difficult to relate all forms of refractory shock to DIC.

The second section begins by describing the architecture, equipment and personnel at the Shock Study Unit of Walter Reed General Hospital. It is stated, correctly, that this is a research unit but the basis for monitoring should apply to all *viz.* "measuring dynamic changes rather than static parameters". The extension of the more practical and useful measurements derived from the laboratory to the field of combat, i.e. "research in the jungle" is admirably illustrated. One cannot help but feel that if it is possible in Vietnam, why not closer to home? It is clear that many more lives are being saved in Vietnam than were saved during the Korean War. A significant contribution to this improvement is the centralization of care which permits central venous pressure to be monitored, and arterial blood to be sampled for pH, Po_2, Pco_2 and lactate. This section contains many well-illustrated and carefully selected clinical examples of shock and its treatment. The book emphasizes total treatment of the patient and complete chapters are devoted to fluid and electrolyte balance, respiratory support and problems, nursing procedures and the work of cardiac arrest teams.

The authors favour the use of vasodilators (phenoxybenzamine) when the patient in refractory shock has a high peripheral resistance, a high central venous pressure and oliguria. They illustrate this treatment with many convincing case reports.

This excellent text on modern concepts in shock should be of great interest to the wide variety of physicians and surgeons who are called upon daily to treat patients with shock.

DAS EKG NACH OPERATIONEN AM HERZEN UND AN DEN GROSSEN GEFAESSEN. Ernst Kriehuber. 256 pp. Illust. Intercontinental Medical Book Corp., New York; Georg Thieme Verlag, Stuttgart, West Germany, 1968. DM 98,00. $26.65 (approx.).

This small monograph, which covers more than the title indicates, provides brief discussions of the pathophysiology of all cardiac lesions that can be treated surgically. The unspecific effects of cardiac surgery on the electrocardiogram are outlined, such as the changes due to incision into the myocardium, postoperative pericarditis and others. Then, the electrographic changes observed following each individual type of operative correction of a cardiac lesion are discussed and documented. The book is certainly up to date: it even includes the electrocardiograms of the first human cardiac transplant.

LES ENTRETIENS DE BICHAT. Chirurgie et Spécialités, 1968. P.-L. Chigot et A. Bellin. 323 pp. Expansion Scientifique Française, Paris, 1968.

L'édition 1968 de "Les Entretiens de Bichat" vient tout juste d'être publiée. Comme à l'habitude, il s'agit d'une série d'articles traitant de sujets de chirurgie générale, de chirurgie spécialisée, de radiologie, d'oto-rhino-laryngologie et de gynécologie et obstétrique.

On y traite une soixantaine de sujets qui peuvent intéresser tous les chirurgiens en pratique active. Quelques sujets sont traités en entier tandis qu'ailleurs on ne fait que résumer un sujet particulier. En plus, il y a des comptes rendus de sujets discutés en table ronde. Ce volume paraît chaque année et vaut réellement la peine d'être consulté. Il y a plusieurs mises au point de certains sujets controversés de même que des articles originaux sur des nouveautés chirurgicales et médicales.

EVALUATION OF RESULTS OF CARDIAC SURGERY. Report of a Symposium of the Fifth World Congress of Cardiology, November 3, 1966, New Delhi, India. American Heart Association Monograph Number Twenty-Two. Edited by Lewis Dexter and Lars Werkö. 111 pp. Illust. American Heart Association, Inc., New York, 1968. $4.00 (U.S. funds). Paperbound.

The authors present their views of the results of surgery in congenital and acquired heart disease up to 1966. In the chapters on congenital heart disease, the information is valid and worth reading. The chapters on acquired heart disease are of much less value. Only 2 of the 26 authors are surgeons and this imbalance is reflected in the vagueness of the cardiologist's knowledge of what was and is possible in valvular repair. In this rapidly changing field, techniques and results in 1968 are quite different from those of 1966. The entire subject of myocardial revascularization is ignored.

The book is useful as a history of certain cardiologists' viewpoint two or three years ago but has little reference to modern cardiac surgical techniques or results.

FACE-LIFT OPERATION. John Conley. 122 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $12.50.

This beautifully set-up and printed little book of 122 pages cannot really be taken seriously as a scientific work. Many of its few pages are taken up completely with photographs which have little or nothing to do with the face-lift operation. The chapter on the technique of the face-lift operation, which one would expect to be quite complete from the title of the book, outlines a technique with one or two minor variations which cannot be considered in any way a complete review of the subject. Indeed, there is no bibliography nor are the names of the originators of this operation even mentioned.

This book should find its place in a public library where the lay public could gain a superficial idea of what the face-lift operation is meant to achieve and how it may be done.

HODGKIN'S DISEASE. Compiled and edited by David W. Molander and George T. Pack. 212 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $18.00.

This comprehensive, well-written 200-page book on Hodgkin's disease covers five areas of the subject: (1) Etiology and pathogenesis is a short chapter because there is no known cause of the disease. (2) Pathologic anatomy is well described and gives a reasonable classification of the disease, according to histologic type. (3) The chapter on immunologic considerations is interesting and discusses the susceptibility of Hodgkin's disease patients to infection, such as tuberculosis. (4) Considerable discussion is given to radiotherapy and radioactive isotope treatment. (5) From the Pack Clinic comes a review of 316 patients, which covers all the clinical manifestations of the disease and gives the results of treatment.

This is a good book for a complete review of this baffling disease. It would be useful in any pathology department, radiotherapy department and cancer clinic. It will be particularly valuable to postgraduate students interested in this disease.

Finally, this book is well produced, well illustrated and easy to read.

THE IDLE THOUGHTS OF A SURGICAL FELLOW. Being an Account of Experimental Surgical Studies 1956-1966. Stacey B. Day. 344 pp. Illust. Cultural and Educational Productions, Montreal, 1968. $12.90.

A number of more or less successful medical practitioners have given up their practices to become writers. However, Stacey Day is, I believe, the only more or less successful surgical fellow who has turned to writing. (Of course he was an unusual surgical fellow to begin with.) In this book he describes his important contributions to the treatment of ischemic heart disease, studies in renal physiology, several historical studies and a number of unsuccessful experiments. Over one-half of the book is devoted to studies of the coronary and intercoronary circulation in man and animals, with special emphasis on the surgical treatment of myocardial ischemia. The author's own contribution to the topic is the description of a method to improve intercoronary communications, by creating a left atrial-pulmonary artery shunt. He gives experimental and clinical details. These and other experiments have led him to express his "idle

thoughts", some of which probably have occurred to many who are engaged in experimental surgery. However, only the writer and poet can express them.

Everybody who has spent time in experimental laboratories, not only doing things, but thinking about the experiments, will enjoy reading this book, but I hope that its readers will not be limited to laboratory people, because "One learns that there is but one laboratory of the open mind and that is, life. All other laboratories and many institutions, I have found, are but universities of the closed mind." In the Foreword, a pediatrician reports "I found it easy to empathize with Stacey's pain at the unresponsiveness of fellows, colleagues, tutors and professors, to his creative notions . . . I was, however, born tough—perhaps far too tough—" The tough pediatrician and the (over-) sensitive surgeon—a most unusual pair.

Surgical research means different things to different people. For many surgical fellows who are assigned to a project for a year during a surgical training program, it is just another job to get over with. For some clinical surgeons, including the survey team of the Medical Research Council of Canada, a surgical researcher is a man who is unable to meet the competition of surgical practice. For our colleagues in the basic sciences, surgical research is an amateurish effort at applied science and developmental research. Thus, the surgeon who devotes time and energy to research is attacked from all sides. You have to be tough in experimental surgery, Stacey, perhaps tougher than your pediatrician friend.

Undoubtedly, Dr. Day will continue to write books related to medicine, the humanities, and poetry. I wonder whether he shouldn't also be encouraged to return to the surgical research laboratory. We need him there, with his humour, his insight and the facility to express "idle" thoughts. In the meantime, every dean and department head should read the chapter entitled "The Surgical Fellow and the Little Prince". A well-worn copy of the first edition of "Le Petit Prince" in my own library attests that others have come to conclusions similar to Dr. Day's.

INTRA-UTERINE DEVELOPMENT. A. C. Barnes. 530 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1968. $20.00.

This book, the work of 26 contributing authors, which is edited by Allan C. Barnes, is a valuable addition to the obstetrical literature. The editor has admirably realized his purpose as defined in the preface "translating the newly acquired investigative knowledge into the practical care of the intra-uterine patient". The text reveals the widening scope and sophistication of academic obstetrics and provides a comprehensive but readable review of recent advances in reproductive biology.

The current understanding of conception, placentation and the morphological and physiological considerations of the developing fetus are described in the first two-thirds of the book. The final third reviews clinically related factors in the fetal environment, including the prediction of fetal jeopardy.

This book is essential reading for the teacher and the graduate student of the growing science that is modern obstetrics.

AN INTRODUCTION TO CLINICAL ANATOMY BY DISSECTION OF THE HUMAN BODY. R. D. Laurenson. 522 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $10.30.

In the preface, the author states that his purpose in writing this dissection manual is twofold: first, to make certain that medical students learn the facts their clinical instructors apply by "clinically" orienting the book; second, to reduce the amount of homework a student must do by avoiding the "hours of tedious reading called for when laboratory work and text are apart".

The basic organization of the manual is similar to that found in "Anatomy of the Human Body" by R. D. Lockhart, to whom Laurenson dedicates this book. The text is arranged so that steps in dissection are separated from descriptive text and given item numbers. The descriptive text includes clinical material, which the author feels is relevant to the anatomy being dissected. References to many clinical points are provided in the text. The book is illustrated, in the main, by clear, concise, line-and-shade drawings. The index is complete and accurate.

Despite the heroic efforts Laurenson has made, it is open to question whether this book succeeds in making anatomical dissection more meaningful and less arduous than does, say, such a standard manual as J. C. B. Grant's "A Handbook for Dissectors". It is a commonplace that for too long anatomy was taught as was Greek—for its own sake. Today, integration of anatomical and clinical knowledge is fundamental to modern medical teaching. But it must be remembered that such integration depends on learning the anatomy *first*.

The time allotted to the teaching of anatomy is everywhere becoming shorter and shorter. Thus, it is more than ever important to stress basic anatomical facts *as such* to medical undergraduates. To introduce a welter of clinical information and opinion into the learning of anatomical fundamentals is inevitably to introduce controversy. Clinical opinions and interpretations vary greatly, while basic anatomy does not. Inherent in this approach is the danger that the controversy will be remembered and the anatomical fact forgotten. The author has an exasperating tendency to describe routine instructions and observations in terms of the immutable, even of the profound.

Those involved in teaching anatomy to medical undergraduates will certainly find Laurenson's approach stimulating and perhaps useful in planning changes in present curricula. It may also be, however, that they will find that Laurenson has produced a prolix manual of dissection at a time when a concise one is needed.

LUNG TRANSPLANTATION. Max J. Trummer and Paul Berg. 122 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $10.25.

This monograph proves to be an easy, readable, concise review of the present state of lung transplantation. The authors have accumulated an excellent bibliography and use their own experience to analyze the data.

Operative technique and postoperative animal care are adequately described and illustrated. Complications are discussed such as those related to anastomotic sites—stenosis or thrombosis—early pulmonary congestion and decreased ability to raise secretions. The authors describe physiological changes in the degenerated lung and supplement this with an evaluation of the deficiencies in our present knowledge and in some of the investigative techniques we utilize.

An elementary review of present methods of immunosuppression is supplemented by an assessment of these agents in long-term animal studies. It should be understood that these results are based upon animal experiments and are not supported by documented histocompatibility profiles. Finally, patients in whom lung transplantation has been done to date are described.

This monograph provides a useful introductory reference to lung transplantation for the undergraduate or postgraduate student. It gives a good general outline and excellent bibliography but falls short of being a definitive reference text.

MAY AND WORTH'S MANUAL OF DISEASES OF THE EYE. 13th ed. T. Keith Lyle, Alexander G. Cross and Charles A. G. Cook. 796 pp. Illust. Baillière, Tindall & Cassell Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1968. $11.25.

It is a pleasure to welcome the thirteenth edition of this old but popular textbook. The first edition appeared in 1906, and the original intention of the authors was to present a concise, practical manual for students and general practitioners. The thirteenth edition is hardly concise, having 796 pages but, considering the rapid development of knowledge and of new techniques in ophthalmology, the present authors are to be congratulated for adding only 48 pages to the twelfth edition.

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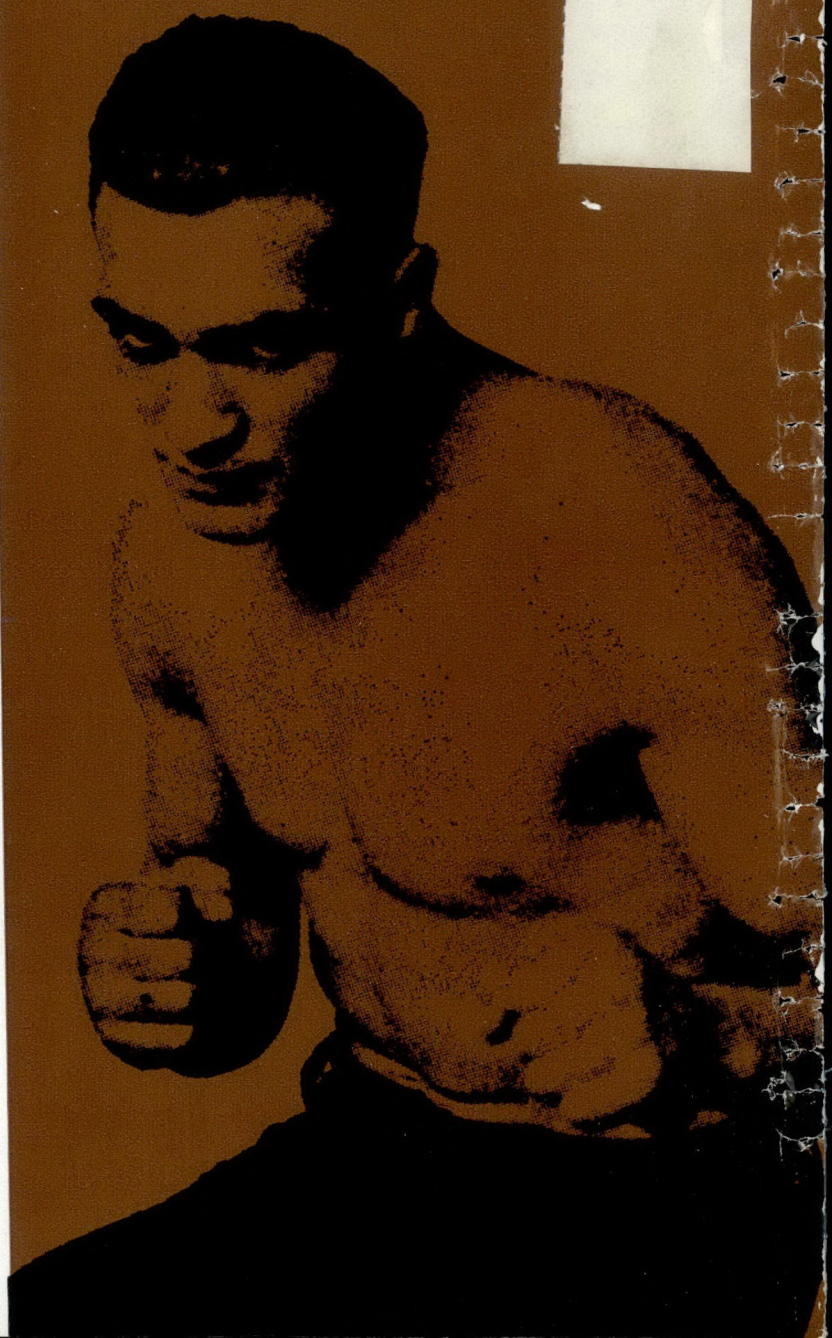

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material and outmoded methods. However, a little space could have been saved by the omission of external examination by oblique light focused by a +10Δ lens. Surely a focusing pen light is the modern equivalent. The same criticism could be made of the description of palpation as a method of estimating intraocular pressure. However, little space has been wasted in this way and all modern routine methods of examination are described, although the omission of fluorescein angiography of the retina is regretted.

The book is surprisingly up to date on new methods of treatment but makes no reference to the new penicillins, or to the use of mannitol and glycerol in glaucoma.

An entirely new section (122 pages) on general and visual optics by Montague Ruben is exceedingly well written, much better than its counterpart (71 pages) in the twelfth edition.

The addition of the excellent section on optics raises a question which is apparent through much of the book. Who is this book intended for? The optics section is beyond the scope of the medical student or general practitioner and is not even useful reference material for them. The rest of the book is too detailed as a routine textbook for medical students, but is a good reference book, particularly because the index is complete. Perhaps the book's main use will be as an introductory manual for the ophthalmology resident.

DIE OPERATIVE BEHANDLUNG DER PROGRESSIV CHRONISCHEN POLYARTHRITIS. Norbert Gschwend. 219 pp. Illust. Intercontinental Medical Book Corp., New York; Georg Thieme Verlag, Stuttgart, West Germany, 1968. DM 98,00. $26.65 (approx.).

In the now-fashionable, systems-oriented organization of medicine, a new specialty seems to be evolving—that devoted to the treatment of progressive chronic polyarthritis. One of the main themes of this book is the definition of this new subspecialty, which depends on the co-operation between the rheumatologist and the orthopedic surgeon. The book consists of two parts: first, the general part, which gives a brief historical survey of joint diseases, and a survey of the social importance of this crippling disease. The pathogenesis of progressive chronic polyarthritis is described in detail and is well illustrated. The author then outlines the aims and the limitations of various surgical procedures and gives a brief discussion of preoperative and postoperative treatment. In the second, special part of the book, much longer than the first part, the author discusses surgical procedures such as synovectomy, arthrodesis, and various arthroplasties according to anatomical regions. The illustrations are excellent and partly in colour. To the specialist, this book can be recommended as an up-to-date review of the present situation. In addition, the book can be recommended to the general practitioner or the general surgeon as a review of a new treatment approach, which is not presented in detail during undergraduate medical training.

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.

This is a small but useful book. It reflects the philosophy of those physicians who are involved in childbirth training and labour as practised in natural birth clinics. It is simply written and easily understood. The exercises are well detailed and chosen for their simplicity as well as for their functional value. This book can be recommended to nurses and all others involved in obstetrics.

PROCEEDINGS OF THE SYMPOSIUM ON COMBINED INJURIES AND SHOCK, Uppsala, June 3-6, 1967. Intermedes 1967, vol. 1. Edited by Bo Schildt and Lars Thorén. 311 pp. Illust. Försvarets Forskningsanstalt, Stockholm, 1968. Price not stated.

This book contains 35 papers presented at a symposium on combined injuries. "Combined injuries" are those that result from two or more types of energy and thus differ from "multiple injuries". The papers are collected into five major sections: combined injury, post-traumatic metabolism, reticuloendothelial system, capillary permeability and therapeutic aspects of shock. They constitute brief reports of current laboratory investigations with a few comments on observations in man under various conditions of wounding or surgery.

The first section, combined injury, describes a series of experiments which indicate that the combination of irradiation and mechanical or thermal trauma enhances the lethality of either wound. No basic information is given on the pathology of irradiation or of the associated experimental injury. The general relationship of incidence to survival suggests that, if one form of trauma is radiation, the timing of the different types of trauma is important when multiple energy sources are involved.

The section on post-traumatic metabolism presents, with unusual clarity, the complicated interrelationships of the pituitary-adrenal axis and its activation in trauma. There are good reports of the depression of energy metabolism in various experimental circumstances and several attempts to trace hormones and isoenzyme variations following different types of experimental injury.

The section on reticuloendothelial system is complex and is obviously intended only for the research worker. It suggests that methodologies are still primitive and that we cannot yet make firm conclusions concerning clinical management. Studies of capillary permeabilities in various laboratory circumstances demon-

strate the variability of capillaries and the difficulty of establishing precise reference points, and again it is obvious that new methods are needed before significant results may be obtained. The therapeutic aspects discussed here are derived from observations on clinical trauma management, but these papers are too few to constitute a comprehensive view of patient care. On the whole, the book is an attempt to provide a basic understanding of many of the current problems in shock. However, it lacks cohesiveness and an integration of purpose and design. It is not suitable for the casual reader but will probably be of considerable value to those whose clinical or laboratory investigations deal precisely with shock. It is a modern compendium of surgical research thought and, as such, demonstrates the magnitude of the problem and the need for continuing effort.

RECONSTRUCTIVE ANATOMY. A Method for the Study of Human Structure. Maurice Arnold. 529 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $11.65.

This new textbook of anatomy presents standard anatomical facts by using what the author calls a "reconstructional" technique. The technique consists of building up each section of human anatomy in orderly fashion from the skeletal substructure to the external surfaces.

The text is set out clearly in two columns per page, and many line-and-shade drawings are used throughout. In each section, the illustrations begin with simple geometrical representations of the skeleton of the area. Next, actual bone structures are shown and, in sequence from deep to superficial, the soft tissue layers are built up upon them. Many of the drawings are excellent but, unfortunately, some are too cluttered or include too great an area. Surface landmarks are stressed throughout "as a constant reminder of the deeper structures". The index, which is complete, is set out plainly in clear, readable type.

The author's most original contribution, a section at the back of the book called "Principles of Anatomy", consists of explanatory notes, opinions and theories on such subjects as joint structure and motion, muscle action, relation of structure to function, etc. It is only here, as Arnold himself had foreseen, that any serious demur can be entered against the content of the book. For example, the note on joint lubrication should have been left out because it gives no hint of current substantiated knowledge of this mechanism. Again, he retains the term "close-packed position" to describe joint congruity: this is to be deplored. Modern engineering methods have shown that the hip joint, for example, is congruous in *all* positions, thus exposing the "close-packed position" theory for the nonsense it is. Barring these objections, the book unquestionably gives a clear, accurate, sequential description of human anatomy. This text would appear to be most useful to medical people, either undergraduate or postgraduate, who want to review, expand or integrate their knowledge of anatomy.

SHEFTS' INITIAL MANAGEMENT OF THORACIC AND THORACO-ABDOMINAL TRAUMA. 2nd ed. Thomas H. Hewlett. 130 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $13.25.

The original edition, based on World War II experience, has been revised and brought up to date by Colonel T. W. Hewlett, United States Army Medical Corps (retired), now of Stanford University. This short monograph—the text and illustrations fill only 114 pages—includes illustrated reports on 12 typical patients. While it still has a battlefield flavour, the injuries produced by heavy industry and motor traffic have replaced, for us in Canada at present, those of the missiles of modern warfare.

The initial chapter on resuscitation, a model of clarity, was written by a surgeon of wisdom and experience. It could be studied with profit by any surgeon or casualty officer who is called upon to treat trauma, or by any practitioner who lives near a highspeed highway.

The subsequent chapters — Thoracic

Wounds, Tracheobronchial Injuries and Thoraco-Abdominal Wounds call for more careful study by any surgeon who is not experienced in the thoracic field. An enormous amount of material has been packed in a small space, so much in fact that unless it is closely read, important points can easily be missed. However, if the reader studies the text along with the illustrative cases he can gain a good understanding of the management of these injuries.

This monograph can be highly recommended.

SURGERY IN THE HEMOPHILIAC. T. J. Tarnay. 131 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $14.00.

This slim monograph offers easy access to the accumulated world experience in surgical procedures in the hemophiliac. There are detailed tabulations dealing separately with appendectomy, splenectomy, gastric procedures, other procedures for gastrointestinal bleeding, for hemophilic cyst and intervention for intracranial bleeding.

The general aspects of hemophilia are dealt with fairly comprehensively but, in some instances, brevity results in obscure condensations. The section dealing with sources of antihemophilic factor for systemic replacement is well handled and gives a good explanation of the basic differences in methods of obtaining these fractions. In discussing animal concentrates, no clear distinction is made between resistance and reaction to the products.

Local hemostatic procedures are dealt with briefly but the complete bibliography allows easy reference to the original experience.

This is a useful book for those managing patients with hemophilia.

A TEXTBOOK FOR MIDWIVES. 6th ed. Margaret F. Myles. 792 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1968. $7.95.

A book designed primarily for the instruction of student midwives will have a limited North American market. However, this book has gone through six editions since its initial publication in 1953, which suggests that it has found general acceptance elsewhere.

The coverage is comprehensive, encompassing a midwife's depth of interest in the full spectrum of normal and abnormal obstetrics. Further chapters deal with the newborn, home confinement, infertility, and a history of midwifery. The style is concise and dogmatic. The illustrations are generally of good quality. In this 792-page volume there is much sound practical obstetrical knowledge and philosophy, but it cannot be considered a valuable text for the physician-obstetrician nor superior to standard North American texts for obstetrical nurses.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Biopsy Procedures in Clinical Medicine. Edited by A. E. Read. 193 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1968. $6.50.

Blood Flow Through Organs and Tissues. Proceedings of an International Conference, Glasgow, March 1967. Edited by William H. Bain and A. Murray Harper. 515 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1968. $13.50.

Cervical Spondylosis and its Neurological Complications. Bernhard H. Smith. 231 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $14.50.

Clinical Management of Shock. Surgical and Medical. Robert M. Hardaway, III. 599 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $26.50.

Clinical Surgery. Genito-Urinary System. Edited by J. D. Fergusson. General editors: Charles Rob and Rodney Smith. 546 pp. Illust. Butterworth & Co. (Publishers) Ltd., London; Butterworth & Co. (Canada) Ltd., Toronto, 1965. $31.00.

Cryosurgery. Edited by Robert W. Rand, Arthur P. Rinfret and Hans von Leden. 428 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $31.75.

Fractures of the Facial Skeleton. 2nd ed. N. L. Rowe and H. C. Killey. 896 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1968. $33.50.

Fundamental Techniques of Plastic Surgery and Their Surgical Applications. Ian A. McGregor. 298 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1968. $5.60.

Historical Aspects of Abdominal Injuries. Frank L. Loria. 213 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $12.50.

The Idle Thoughts of a Surgical Fellow. Being an Account of Experimental Surgical Studies 1956-1966. Stacey B. Day. 344 pp. Illust. Cultural and Educational Productions, Montreal, 1968. $12.90.

Inhalation Therapy Procedure Manual. Thomas J. DeKornfeld and Don E. Gilbert. 114 pp. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $8.00.

Intestinal Antisepsis. Isidore Cohn, Jr. 245 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1969. $21.00.

An Introduction to the History of General Surgery. Richard Hardaway Meade. 403 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $18.40.

Logan Turner's Diseases of the Nose, Throat, and Ear. 7th ed. Edited by John P. Stewart assisted by J. F. Birrell. 584 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1968. $11.25.

Malabsorption Syndromes. William W. Shingleton and William O. Dobbins, III. 165 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $9.00.

Nerves and Nerve Injuries. Sydney Sunderland. 1161 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1968. $39.75.

Nouveau traité de technique chirurgicale. Tome XII. Fasc. 1. Foie et voies biliaires intra-hépatiques. René Bourgeon and Marcel Guntz. 324 pp. Illust. Masson et Cie, Paris, 1968. 120 F. $26.50 (approx.).

Operative Neurosurgery. Vol. 1. Cranial, Cerebral, and Intracranial Vascular Disease. Ludwig G. Kempe. 269 pp. Illust. Springer-Verlag, Berlin; Springer-Verlag New York Inc., New York, 1968. $39.50.

Pediatric Head Injuries. John Mealey, Jr. 243 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $12.50.

Planning of Surgical Centers. Basic Studies. Ervin Pütsep. 122 pp. Illust. Lloyd-Luke (Medical Books) Ltd., London; Natur och Kultur, Stockholm, 1969. 80/–. $10.00 (approx.). Paperbound.

Postoperative Frühkomplikationen. Grundlagen der Krankenbehandlung auf der Wachstation. Kurt Wiemers, Ernst Kern, Maria Günther and Hilmar Burchardi. 248 pp. Illust. Intercontinental Medical Book Corp., New York; Georg Thieme Verlag, Stuttgart, West Germany, 1969. DM 49,00. $13.30 (approx.).

Proceedings of the Symposium on Traumatic Arterial Lesions. Intermedes 1967. Edited by Tor Hiertonn and Bo Rybeck. 165 pp. Illust. Försvarets Forskningsanstalt, Stockholm, 1968. Price not stated.

Scientific Writing. Lester S. King and Charles G. Roland. 132 pp. American Medical Association, Chicago, 1968. $1.00. Paperbound.

Solitary Metastases. Philip Rubin and Jerold Green. 251 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $17.75.

Spinal Cord Injury. Vladimir Benes. 202 pp. Illust. Baillière, Tindall & Cassell Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1968. $7.25.

The Surgeon's Responsibility. John R. Derrick. 121 pp. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $6.00.

Surgery for Cerebrovascular Insufficiency (Stroke). With Special Emphasis on Carotid Endarterectomy. Jesse E. Thompson. 96 pp. Illust. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $11.50.

Surgery for Thoracic Disease. An Outline. Robert R. Shaw. 141 pp. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $8.50.

Surgical and Medical Emergencies. 2nd ed. Edited by John H. Schneewind. 318 pp. Illust. Year Book Medical Publishers, Inc., Chicago, 1968. $5.50. Paperbound.

Symposia on Reconstructive Plastic Surgery and on Surgery of the Hand. Reprinted from the Surgical Clinics of North America. Edited by John Marquis Converse and Martin A. Entin. 1183 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $16.20.

The Training of Surgeons in the Future. Sponsored by the Allen O. Whipple Surgical Society. Various contributors. 188 pp. Charles C Thomas, Publisher, Springfield, Ill.; The Ryerson Press, Toronto, 1968. $10.50.

The Treatment of Burns. 2nd ed. Curtis P. Artz and John A. Moncrief. 393 pp. Illust. W. B. Saunders Company, Philadelphia; W. B. Saunders Company Canada Limited, Toronto, 1968. $15.70.

SURGICAL PRINCIPLES. James Moroney and Francis E. Stock. 371 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1968. $10.50.

In this relatively small text the authors define and describe some of the principles underlying surgical diseases on a broad and somewhat unusual basis. Their categorization of lesions is somewhat artificial and will not help the average student; for example, as stated in the preface, "Conditions as dissimilar as hemorrhage, ascites, surgical emphysema, fistulas and dehydration of diarrhea are all examples of abnormal movements of fluid in the body and are thus considered together." An example of other conditions that are grouped together is the tetralogy of Fallot, patent urachus, ductus arteriosus, Meckel's diverticulum and oblique hernias. These are considered as "failures of obliteration of fetal pathways", thus are grouped together as examples of a similar pathological process.

This arrangement of surgical lesions is of little value to a student or to a practising doctor. The book might possibly be of value to a student preparing for examinations, if the questions were also unusual.

The text is well illustrated with pictures and line drawings. The content is interesting; for example, one reads that intracerebral hemorrhage and peritonitis with fluid exudate, are considered together as "fluid leakage into the body cavities".

The book is well written, the concepts are new and interesting, but it cannot be recommended for the student or graduate intern, only as an interesting and readable ancillary text for the graduate surgeon.