4-1-1959

Volume 02, issue 3

Canadian Medical Association

L'HISTOIRE DE LA CHIRURGIE CANADIENNE

SIR WILLIAM HALES HINGSTON
(1829 - 1907)

EDOUARD DESJARDINS, M.D., F.R.C.S.[C], F.A.C.S.,* *Montréal*

L'HISTOIRE de la chirurgie canadienne commence à Montréal avec William H. Hingston; il est un des premiers chirurgiens dont la renommée ait dépassé les limites du pays.

Né en 1829, l'année de la naissance de Theodor Billroth, de Meissner, de Lancereaux, de Lefort, de Setchenow, le père de la physiologie russe, de Jules Parrot, William Hingston a eu une carrière fructueuse et il a brillé dans tous les domaines: chirurgie, enseignement, direction universitaire, politique et haute finance.

Il vit le jour, le 29 juin 1829, sur une ferme du Bas-Canada. Son père, Samuel James Hingston, était un lieutenant à la retraite de l'armée impériale. Originaire du Comté de Cork, en Irlande, Samuel James était venu au Canada en août 1805, avec le 100e régiment d'infanterie. Las de la vie militaire, il quitta l'armée en 1818 et, quelques années plus tard, il obtint de la Couronne un lopin de terre situé sur les bords de la rivière Châteauguay, à proximité de la frontière américaine, en un endroit appelé Hinchinbrooke. Samuel James menait l'existence dorée du fermier-gentilhomme et il consacrait plus de temps à la chasse et à la pêche qu'à la conduite de ses affaires, si bien qu'à sa mort, survenue en 1831, il ne laissa à ses héritiers qu'un patrimoine amoindri et grevé de dettes.

Sa veuve, durement éprouvée, n'en réussit pas moins à élever dignement les cinq petits, privés prématurément de leur père. Elle parvint, par une sage administration, à récupérer une partie des biens engagés et à maintenir en équilibre instable le budget familial. Le courage de la mère modela l'esprit de ses enfants et les habitua aux actes de sacrifices et de renoncement.

William reçut à l'école rurale, ses premières leçons. Personne ne savait alors qu'il était l'élève d'un professeur qui devait devenir, en Angleterre, un grand financier et s'appeler Sir John Ross.

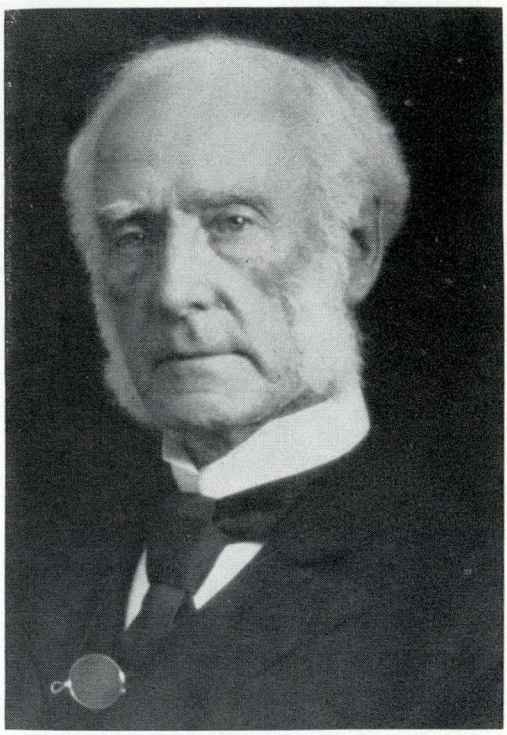


Fig. 1.—Sir William Hingston.

Cette rencontre imprévue d'un maître hors-pair et d'un élève réceptif et travailleur donna les résultats inévitables d'un succès scolaire sans précédent.

Ross, conscient de la valeur de William, insista auprès de Madame Hingston, pour qu'elle envoyât son fils poursuivre ses études au Collège de Montréal, ce qu'elle fit et qui s'avéra de bon aloi. Bien que l'enseignement fût au Collège donné en majeure partie en langue française, William arriva toujours au premier rang de ses classes et se mérita tous les prix.

La voie facile n'était pas celle que Dieu avait tracée pour William Hingston. Après deux années passées au Collège, les finances familiales redevinrent à la baisse et la mère, avec désespoir, apprit à son fils

*Professeur de chirurgie à l'Université de Montréal.

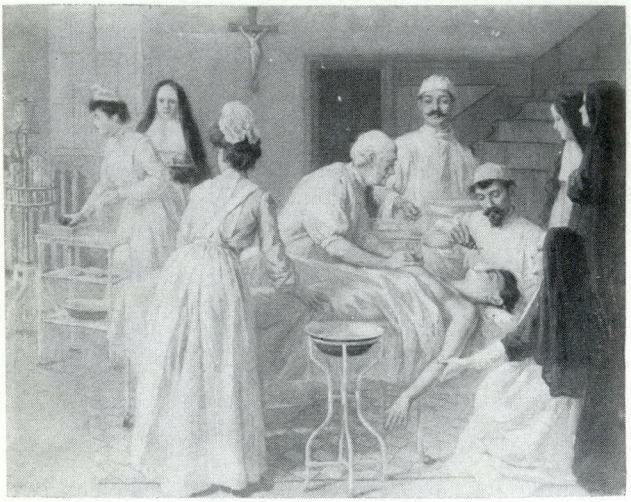


Fig. 2.—Le docteur Hingston à la salle d'opérations. Peinture de Charles Franchère, 1905. (Collection de l'Hôtel-Dieu de Montréal.)

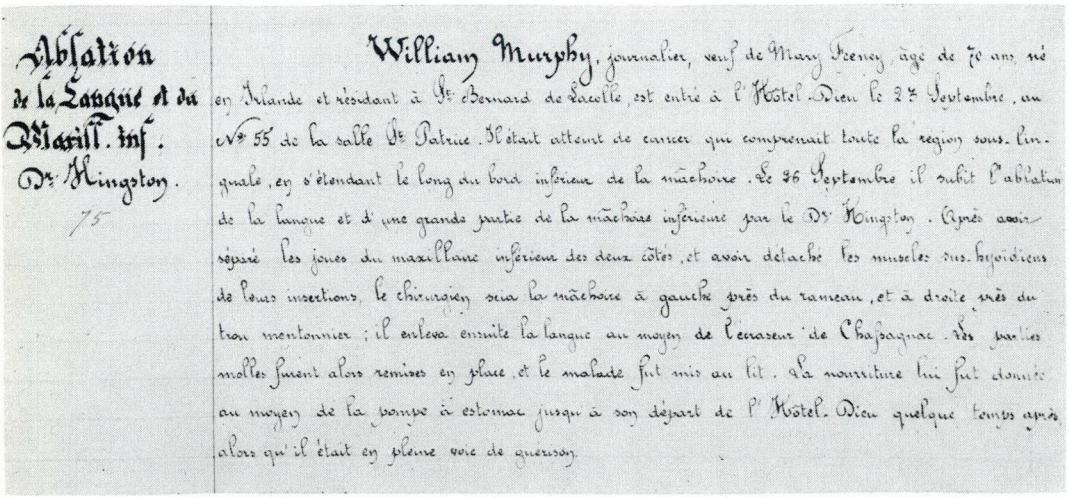
qu'il devrait quitter le Collège, à moins qu'il ne se destinât à la prêtrise. William lui répondit avec la droiture que l'on remarquera demain chez l'homme mûr: "Mother, I would certainly feel very happy and very honoured if God were to call me to the priesthood, but I am too young to make such a decision. If, therefore, you require a decisive answer, it will have to be no".

Le lendemain, il quitta le Collège de Montréal. Dévoré du feu de la connaissance, il se mit en quête d'un moyen pour parvenir à ses fins. Il trouva bientôt une place d'apprenti-pharmacien, où, moyennant un dur labeur quotidien, il obtenait gîte et nourriture. Le jour, il aidait l'apothicaire et, le soir, il se plongeait dans l'étude des textes que ses anciens maîtres de St-Sulpice lui fournissaient et que, gracieusement, ils corrigeaient.

Sous une direction aussi avisée, l'élève fit des progrès de géant et acquit rapidement le droit d'entrer à l'Université.

Il s'inscrivit à McGill, d'abord en pharmacie, puis en médecine. Il y fit des études sérieuses et réussit, en 1851, à obtenir son doctorat en médecine. Il était âgé de 22 ans; aussi, conscient de son insuffisance scientifique, il décida d'aller puiser aux sources de l'enseignement médical supérieur. Il s'embarqua donc pour l'Europe, où il vécut deux ans acharné au travail, vivant frugalement comme un ascète.

William Hingston faisait son apprentissage à la spartiate, prêt à tout renoncement pour arriver à acquérir la science nécessaire; quarante ans plus tard, soit en 1892, le doyen Hingston le faisait comprendre aux finissants de l'Ecole de Médecine et de Chirurgie de Montréal: "Souvenez-vous toujours et dans toutes les circonstances,


Ablation de la Langue et du Maxill. inf.
Dr Hingston.
75

William Murphy, journalier, veuf de Mary Feeney, âgé de 70 ans, né en Irlande et résidant à St. Bernard de Lacolle, est entré à l'Hôtel-Dieu le 24 Septembre, au N° 55 de la salle St Patrice. Il était atteint de cancer qui comprenait toute la région sous-linguale, en s'étendant le long du bord inférieur de la mâchoire. Le 26 Septembre il subit l'ablation de la langue et d'une grande partie de la mâchoire inférieure par le Dr Hingston. Après avoir séparé les joues du maxillaire inférieur des deux côtés, et avoir détaché les muscles sus-hyoïdiens de leurs insertions, le chirurgien scia la mâchoire à gauche près du rameau, et à droite près du trou mentonnier; il enleva ensuite la langue au moyen de l'écraseur de Chassaignac. Les parties molles furent alors remises en place, et le malade fut mis au lit. La nourriture lui fut donnée au moyen de la pompe à estomac jusqu'à son départ de l'Hôtel-Dieu quelque temps après alors qu'il était en pleine voie de guérison.

Fig. 3.—Reproduction d'un extrait du registre des opérations pour l'année 1872 de l'Hôtel-Dieu de Montréal. Protocole opératoire du premier cas d'ablation totale de la langue et du maxillaire inférieur pour cancer.

Fig. 4.—Leçon clinique du professeur Hingston à l'amphithéâtre de l'Hôtel-Dieu.

que la médecine est un sacerdoce. Pour l'exercer, il faut de la science, beaucoup de science; mais, quelque science que vous possédiez, vous ne serez jamais de bons médecins si vous n'y ajoutez l'autorité de qualités morales supérieures".

En ce milieu du XIX[e] siècle, il fallait se déplacer pour apprendre, car la science était sporadique. L'anatomie et la technique étaient le fort de l'Italie et de la France; la pathologie et l'histologie, de l'Allemagne et de l'Autriche. La chirurgie gastro-intestinale brillait en Autriche, l'antisepsie en Angleterre. William Hingston passa d'Irlande en Prusse, puis en Bavière, d'où il gagna, par la France, l'Autriche pour revenir en Angleterre et en Ecosse.* C'est là qu'il demeura le plus longtemps; il y fut un des assistants de Sir James Simpson, célèbre pour son emploi du chloroforme en obstétrique et en chirurgie.

Les grands chirurgiens d'Europe étaient passés maîtres dans l'art d'enseigner les ressources de la science chirurgicale; en plus d'être de brillants anatomistes, ils maniaient avec dextérité et précision ciseaux, pinces et bistouris.

"Il faut des mains", dit un jour Paul Valéry à des chirurgiens, "non seulement pour réaliser, mais pour concevoir l'invention la plus simple sous forme intuitive. Songez qu'il n'est peut-être pas, dans toute la série animale, un seul être autre que l'homme qui soit mécaniquement capable de faire un nœud de fil; et observez, d'autre part, que cet acte banal, tout banal et facile qu'il est, offre de telles difficultés à l'analyse intellectuelle que les ressources de la géométrie la plus raffinée doivent s'employer pour ne résoudre que très imparfaitement les problèmes qu'il peut suggérer".

En fin de stage, Sir James Simpson offrit à son élève Hingston une place permanente

*Durant ses stages, il recueillit des diplômes en Prusse et en Bavière; à Vienne, il fut admis à l'Académie Royale de Léopold; à Londres, il obtint une licence du Royal College of Physicians et il fut admis comme "fellow" du Collège Royal des Chirurgiens d'Edimbourg.

Fig. 5.—L'Ecole de Médecine et de Chirurgie de Montréal. Ses professeurs et son doyen, le professeur Hingston (circa 1884).

d'assistant dans son service. Flatteuse invitation déclinée avec émotion et qui ne rompit jamais les liens d'estime qui unissaient élève et patron, puisqu'en 1863, Sir James invita à opérer devant ses élèves William Hingston, de passage à Edimbourg.

De retour au Canada, précédé par la renommée de ses travaux et de ses titres acquis à l'étranger, Hingston n'eut pas à attendre la clientèle; elle accourut nombreuse et son magnétisme l'attirait; il en imposait et plaisait tout à la fois. "Grand, six pieds de taille, épaules larges, ceinture étroite, droit comme une flèche, athlète, lèvres fermes, visage sérieux et pensif".[1] Des traits saillants marquaient sa physionomie. "Front large d'intellectuel, favoris, nez au profil classique et pur, la bouche énergique nettement ciselée, des yeux bleus rêveurs d'une douceur infinie, la stature d'un

Fig. 6.—Groupe des professeurs titulaires réunis pour la première fois après la fusion entre Victoria et Laval (1892) autour du doyen Durocher.

militaire, les mains larges et fortes, l'allure d'un grand seigneur".[2] Sous l'emprise d'une telle figure, la communion de patient à médecin était instantanée. La personnalité morale était non moins frappante. Il possédait "une grande intelligence, une imagination très vive, une éducation parfaite, un tact et un jugement jamais en défaut, des manières d'une distinction exquise, une figure noble".[3]

La carrière de dévouement du docteur Hingston commença réellement en 1854, avec l'épidémie de choléra. Il se consacra nuit et jour au service de ces pauvres immigrants irlandais et "il était tellement fourbu par le manque de repos qu'il lui arrivait de tomber de sommeil sur son cheval ou de s'étendre sur le parquet". La même année survint l'accident du pont de Belœil; il y excella si bien auprès de la centaine de blessés que les officiers du Grand Trunk Railway lui offrirent, sur-le-champ, la place de médecin-chef à plein temps. Il refusa, car la voie qu'il s'était tracée dépassait la barrière du sédentarisme.

Attaché d'abord à l'hôpital St-Patrick, il est invité, le 20 juin 1860, à devenir chirurgien de l'Hôtel-Dieu. Il y a fait toute sa carrière et il en a été le chirurgien-en-chef pendant près de quarante ans.

Les gens de l'Hôtel-Dieu ont gardé de lui un souvenir impérissable; les religieuses lui vouaient un grand respect et il le leur rendait bien. Les anciennes religieuses

prennent plaisir à raconter certaines anecdotes qui caractérisent l'aspect humain de William Hingston. Il lui arriva, un jour, d'entrer dans la salle des hommes au moment du repas. "I smell this lovely soup", dit-il à celle qui l'accompagnait, selon la coutume. "Demandez aux Sœurs de m'en préparer une portion et de me la servir dans un bol comme celui des malades". L'ayant goûtée: "Quel régal!" Après une opération laborieuse, il répondit une fois à la pharmacienne qui lui offrait un verre de lait arrosé de cognac: "Ma Sœur, vous avez des vaches qui donnent un lait très distingué".

L'œuvre accomplie à l'Hôtel-Dieu par William Hingston fut considérable. Le registre des opérations mentionne son nom fréquemment et seul celui de son patron Peter Munro le devance en fréquence.

William Hingston aurait, vers 1863, innové en faisant une néphrectomie pour cancer du rein; mais, les historiens ne sont pas d'accord pour décerner au chirurgien de Montréal la priorité de cette opération. Les recherches entreprises en vue de la présente biographie n'ont pu confirmer, ni infirmer cette assertion.

Toutefois, un fait est acquis, puisqu'il est consigné au livre des protocoles opératoires, Hingston s'est avéré le pionnier de la grande chirurgie d'exérèse, alors qu'il fit, le 27 septembre 1872, l'ablation totale de la langue et du maxillaire inférieur.[4]

En plus d'être un chirurgien hardi et compétent, William Hingston fut aussi un excellent professeur; doué de sens clinique et d'un lucidité d'exposition remarquable, il ramassait d'un trait l'histoire du malade et il en extrayait la substance à retenir. Le Bishop's College of Medicine, fondé à Montréal en 1871, lui confia, dès le début, la chaire de chirurgie et, quelques mois plus tard, le désigna à la présidence de l'Ecole. Toutefois, la dualité de service était incompatible et il quitta Bishop pour rester fidèle à l'Hôtel-Dieu. En hommage de reconnaissance, l'Université de Lennoxville lui décerna un doctorat "honoris causa".

La littérature médicale a été enrichie par les nombreuses publications de William Hingston. En 1872, il faisait partie des vingt-huit médecins de Montréal qui présidèrent à la naissance de *l'Union Médicale du Canada* et qui en garantirent l'avenir de leurs deniers; bien plus, il en fut, de 1872 à 1879, un collaborateur fréquent.

L'Abeille Médicale et le *Journal de l'Association Médicale Canadienne* eurent aussi l'occasion de reproduire ses discours et ses communications aux congrès et aux réunions de sociétés. Les frères Dawson de Montréal publièrent, en 1884, le volume magistral de William Hingston: "The Climate of Canada". Ce livre valut à son auteur d'élogieuses appréciations et la lecture est encore agréable et instructive. L'auteur a fait œuvre originale, car il a ouvert large une croisée sur l'horizon scientifique. "I have only to add", lit-on dans la préface, "that one motive, and one only, have I had in placing the following paper before the public: that as little–very little –had been written on the subject . . . I felt I should contribute something".*

La profession médicale trouva en lui un défenseur ardent; il fut, en 1874, élu Gouverneur du Collège des Médecins de la Province de Québec, et, onze ans plus tard, il obtint la présidence du Collège.

Clinicien bénévole jusqu'alors, William Hingston fut nommé professeur titulaire de chirurgie en 1875; il enseignait à l'Ecole de Médecine et à l'Hôtel-Dieu.

La même année, trois évènements d'importance marquèrent la vie de William Hingston. Il épousa, le 10 septembre, Margaret Josephine MacDonald, fille du Colonel Donald Alexander MacDonald, lieutenant gouverneur de la province d'Ontario; il fut élu maire de Montréal et il devint directeur de la Banque d'Epargne de la Cité et du District de Montréal.

1875 marque le sommet de la carrière. Maire de Montréal, il transposa sur le plan social sa conception du devoir. Emu par la mortalité élevée, il entreprit une croisade

---

*Le docteur W.-H. Hingston publia également plusieurs brochures: "The Medical Institutions of Paris", The Medical Chronicle Office, Montreal, 1885. "Medical Evidence in the Wellington Street Murder Case", Montreal, 1860. "Myotomy and tenotomy in certain joint diseases and the sequelæ", Montreal, 1871. "Remarques sur la vaccination", Louis Perrault, Montréal, 1876. "Remarks on vaccination", Montréal, 1885. "Note-book for cases of ovarian and other abdominal tumours", Dawson Brothers, Montreal, 1887.

hygiénique. "Elsewhere, men and women die, here, they are killed", "tués par l'indifférence officielle, par l'insouciance des corps publics, par l'incompréhension de la masse", dit-il dans son discours inaugural. A peine en fonction, il fonda le premier bureau d'hygiène municipal.

William Hingston fut trois ans maire et accomplit ses fonctions officielles avec tact et dignité. Il y avait, à l'époque, beaucoup de chômage et les esprits s'échauffaient devant l'apathie des pouvoirs publics.

Un après-midi, les chômeurs organisèrent une marche de protestation sur l'hôtel de ville qu'ils entourèrent bientôt, criant menaces et injures. Le maire Hingston, mis au courant, se rendit sur le Champ de Mars, au-devant des manifestants. Calme, imperturbable, il réclama un instant de silence.

Ils s'attendaient à entendre la lecture de l'Acte d'émeute et ils se trompaient. "Ce ne sont pas des criminels, mais des affamés, et c'est le devoir de la Cité de leur fournir du travail", dit-il à son greffier. Et il leur parla sur le ton du père ému par le malheur des siens; il se montra compatissant et compréhensif. Il leur promit du travail et ils se retirèrent en silence. Un homme de cœur leur était apparu; il tint parole et, de ce moment, date l'origine des travaux d'embellissement du Parc de la Montagne. Le premier urbaniste de Montréal fut-il un chirurgien?

Le docteur Hingston resta toujours fidèle à l'Ecole de Médecine, affiliée depuis le 10 septembre 1866, à The University of Victoria College de Cobourg, Ontario.

L'Ecole était unie étroitement à l'Hôtel-Dieu, son hôpital Universitaire, d'autant plus que l'une était située avenue des Pins, en face de l'autre. Mais, de 1876 à 1891, il y eut des moments difficiles: trois écoles de médecine se faisaient concurrence à Montréal. Le professeur Hingston et ses collègues souffraient de cet état anormal. L'ancien maire Hingston, diplomate-né, s'ingénia à favoriser un rapprochement juste et équitable.

Un compromis fut suggéré en 1883. L'Ecole Victoria laisserait le champ libre à Laval et, en compensation, Laval offrait une chaire à trois titulaires de Victoria: William Hingston, Edouard Desjardins et Daniel Mignault, et l'honorariat aux autres professeurs de Victoria.

Les trois élus par Laval refusèrent de se désolidariser de leurs collègues et, peu après, William Hingston était nommé Doyen de l'Ecole, fonction qu'il conserva jusqu'à la fusion qui eut lieu le 1er juillet 1891.* Il continua, par la suite et jusqu'à sa mort, un enseignement chirurgical qui fut toujours très apprécié des élèves et qu'il faisait dans un français très élégant.

La carrière de William Hingston se déroula toujours aussi prestigieuse. Aucun mouvement médical n'était déclenché, où il ne fut parmi les fondateurs; l'Association Médicale Canadienne et le Conseil Médical du Canada lui durent en partie leur origine. Il était en grande demande comme conférencier et son nom était en tête de toutes les organisations sociales et philanthropiques.

Deux années, 1875 et 1895, ont marqué profondément le cours de son existence. En 1875: mariage, professorat, mairie; en 1895, il fut décoré par la reine Victoria du titre de Knight Bachelor et il accéda à la présidence de la Banque d'Epargne. Ce fut aussi l'année de ses débuts en politique fédérale. Candidat dans Montréal-Centre à l'élection du 27 décembre 1895, il a été battu, malgré sa popularité et peut-être à cause de celle-ci. Cette infortune lui valut, toutefois, d'être nommé sénateur en 1896.

Sir William avait une telle réputation comme chirurgien qu'il était un consultant très recherché et on a même écrit[5] qu'il aurait été appelé au chevet du Président Garfield, blessé gravement le 2 juillet 1881 et décédé le 19 septembre 1881.

Cependant, un article récent ne mentionne pas le nom Hingston parmi les consultants.[6]

Dès 1895, la vie publique et la Banque prirent le pas sur la médecine. Sir William devait se rendre fréquemment à Ottawa et il était, par ses responsabilités financières, largement accaparé. Les honneurs lui arrivèrent de partout, sans qu'il eut besoin de les solliciter et ils se succédaient à l'année longue; le dernier qui lui fut offert

---

*Le bill qui réunissait Victoria et Laval fut sanctionné le 30 décembre 1890; il conservait à l'Ecole de Médecine son entité comme corporation distincte.

le toucha profondément: celui de la présidence d'honneur du Congrès International de Chirurgie qui se tint à Paris en 1906.

Sir William eut la fin qu'il avait souhaitée: mourir à la tâche, être atteint au devoir. En pleine consultation, un après-midi, il se sentit mal, s'excusa auprès de ses malades et prit le lit. Moins de vingt-quatre heures plus tard, il n'était plus.

Le souvenir d'un grand destin s'attachera au nom de Sir William Hingston et sa vie s'inscrira dans l'histoire à la page des belles existences.

La plus vive reconnaissance est due aux autorités religieuses de l'Hôtel-Dieu, au Révérend Père William H. Hingston, s.j., de Toronto, et au docteur Paul Dumas, chargé du cours d'histoire de la médecine à l'Université de Montréal, qui ont fourni une large part de la documentation.

BIBLIOGRAPHIE

1. HINGSTON, W. H.: *Canadian Catholic Historical Association Reports*, 109, 1951.
2. KENNEDY, E. J.: *Union méd.*, **37:** 178, 1907
3. HERVIEUX, H.: *Union méd.*, **37:** 634, 1907.
4. HINGSTON, W. H.: *Union méd.*, **2:** 100, 1873.
5. ST-JACQUES, E.: *Union méd.*, **49:** 129, 1921.
6. MILLER, J. M.: The death of James Abram Garfield, *Surg. Gynec. & Obst.*, **107:** 113, 1958.

MEDICINE AND THE NAVY 1200-1900. Volume II—1649-1714. J. J. Keevil. 332 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1958. $6.75.

In the second volume of this authoritative textbook on the history of medicine in the Royal Navy, the late Surgeon-Commander Keevil takes up his tale with the beginning of the Commonwealth in England and ends it with the death of Queen Anne. Throughout this period, the place of the medical man in the Navy was becoming more secure, though his status was still low. But at least society had at last recognized that the sailor was a human being with a right to treatment when he was wounded or injured, though his right to suffer from internal diseases was not so readily conceded and the surgeon was still the only important type of practitioner aboard.

Through most of the book there is a constant struggle going on between those responsible for procuring treatment for sick and wounded seamen, and civilians reluctant to lodge these heroes either in their seaside homes or in the great hospitals of London, St. Bartholomew's and St. Thomas's. The hospital ship appears in mid-century, and the first naval shore hospital at Greenwich at the end of the eighteenth century—a much overdue institution.

Meanwhile the lordly academic physicians remain as divorced from common man and his needs as ever, and the Barber-Surgeons' Company of London carries on a running fight with government, much as the B.M.A. has later done. As now, the surgeon serving his country was miserably paid in comparison with his civilian brother, yet great figures such as James Pearse, the first Surgeon-General, appear in the service.

Keevil throws much light on surgical methods at sea, noting how the haste and overwork associated with cockpit surgery probably contributed to a high amputation rate, as it did in the German army in its recent Russian campaigns.

There are biographical sketches of many entertaining characters, including a forerunner of Florence Nightingale, a tough-minded woman, Mrs. Elizabeth Alkin who rode around England in 1653, procuring nursing and other comforts for the sick and wounded. Her fate was to die in poverty, neglected by the government she had served so well.

This is a book to keep, to read and to re-read, and to draw lessons from. It is to be hoped that some worthy successor to this admirable historian will come forward to complete the work.

ORIGINAL ARTICLES

AN EVALUATION OF ASEPTIC AND ANTISEPTIC TECHNIQUES AS PRACTISED IN A MODERN HOSPITAL
I: Operating Theatres*

M. W. NIMECK, M.Sc.,† G. E. MYERS, Ph.D.† and
W. C. MACKENZIE, M.D., F.R.C.S.[C], F.A.C.S.,‡ *Edmonton, Alta.*

INTRODUCTION

MANY SOURCES of contamination for surgical wounds have been suggested in the past. Aerial contamination has been studied by Hart,[9-11] Robertson and Doyle,[23] Anderson,[1] Bourdillon *et al.*,[4] Goodman *et al.*,[8] and Girdlestone and Bourdillon.[7] The latter workers included studies of skin contamination in their investigations. Poppe[19] studied wash basins, Propst[22] investigated instruments and drapes. Surgeons' aprons were studied by Corry[6] and anæsthesia equipment was examined by Zeigler and Jacoby[26] and Joseph.[12]

The recent increase in postoperative infection particularly by *Micrococcus pyogenes var. aureus* and the ubiquitous nature of this micro-organism indicates a need for a searching appraisal of the aseptic and antiseptic techniques practised in modern hospitals. The report presented at this time constitutes the first part of such an investigation.

EXPERIMENTAL DESIGN

The operating theatres of a large teaching hospital with a capacity of approximately 1000 beds were investigated. Situated on the fifth floor of the hospital, all the operating theatres are connected by a common corridor which is separated from the main corridor of the hospital by swinging doors. The doors of the operating theatres open directly into the corridor and are frequently left open throughout the day when the theatres are in use. Windows are equipped with screens and draught guards and are usually open in warm weather.

The following possible sources of contamination were studied under normal working conditions: (1) air, (2) masks, (3) splash basins, (4) operating table mats and sheets, (5) floors, (6) scrub water for floors, (7) mops used in scrubbing floors, (8) anæsthesia masks, (9) endotracheal tubes, (10) stretcher blankets, (11) depots, (12) surgeons' hands, (13) patients' skin, (14) respiratory tract of personnel. All factors were investigated simultaneously and continuously throughout the entire operating day. The investigation was conducted intermittently over a period of two years, in the absence of any indication of an outbreak of infection.

MATERIALS AND METHODS

1. *Air sampling.*—The General Electric duplex bacterial air-sampler was used for all tests for micro-organisms suspended in the air.[13-16] The sampler was located in the operating theatre at approximately the same height as the operating table and three to four feet (90-120 cm.) away from it. Samples were collected on pairs of blood agar plates containing 5% sheep's red blood cells. Samples were obtained from the unoccupied operating theatre (at 5:00 to 6:00 a.m.) and thereafter before the first operation, during each operation, between operations, during clean up and after the last operation of the day.

Organisms settling out of the air were tested for by exposing open culture plates during the same periods of time. Open culture plates were exposed overnight to determine the rate of "settling-out" when the room was unoccupied. All cultures were incubated aerobically at 37.5° C. for 48 hours before counting colonies.

*Supported in part by a grant from the University of Alberta Hospital.

†Department of Bacteriology, University of Alberta, Edmonton, Alberta.

‡Director of Surgical Services, University of Alberta Hospital.

2. *Masks.*—Curity gauze masks approximately 6 inches (15 cm.) square, consisting of two layers of gauze with a heavy layer of flannelette between, are worn in the operating theatres. These masks are routinely sterilized before use but are then placed unwrapped in an open receptacle in the corridor or in the surgeons' room. These masks were tested for contamination before use by cutting ¼ inch square areas from the inner and outer layers of gauze, and culturing.

To test the efficiency of the masks, cotton cloth discs ¼ inch in diameter were sewn to the inner and outer surfaces of the mask at a point corresponding with the position of the mouth when the mask is in use. The specially prepared masks were then wrapped, sterilized by autoclave and distributed to operating theatre staff. After use the masks were collected in individual sterile paper bags and transported to the laboratory. The cotton discs were aseptically removed and placed in 10 ml. of sterile water. The sterile water was shaken mechanically until the cloth disc disintegrated. Aliquots of the suspension were plated out on sheep's blood agar. All cultures were incubated for 48 hours at 37.5° C. before counting colonies.

3. *Splash basins.*—Stainless steel basins are used. The basins are sterilized by autoclave the evening before use. After cleaning of the operating theatre is completed and immediately before the operation commences, the basins are uncovered and filled with sterile distilled water or saline. Instruments, sponges and surgeons' gloves are rinsed in the solution during the operation. The solution may be used throughout an operation or may be changed periodically.

One ml. samples of the solution in the splash basin were removed aseptically at half-hour intervals during an operation. Each sample was immediately added to 9 ml. of Letheen broth (Stuart, Ortenzio and Friedl[25]) to inactivate the quaternary ammonium compound in which the instruments were soaked before use. In the laboratory a dilution series of each sample was prepared and pour plates were made with nutrient agar.

4. *Operating table mats and sheets.*—Stainless steel operating tables with foam rubber, plastic covered mats are used. The tables and mats are washed with an aqueous solution of benzalkonium chloride 1 in 1000 at the end of each operating day and after "dirty" cases or when considered grossly contaminated. Mats are covered with freshly laundered, non-sterile sheets which are changed after each operation.

Sterile aluminum templates with one square inch areas cut out were used to limit the sampling area for operating table mats and sheets. Samples were taken by swabbing the entire open area with a sterile swab moistened in nutrient broth. Swabs were immediately shaken in 3 ml. of sterile nutrient broth mechanically for 15 minutes. Aliquots of the broth were then plated out to sheep's blood agar. All cultures were incubated aerobically 48 hours at 37.5° C. before counting colonies.

5. *Operating theatre floors.*—All the operating theatres have marble chip floors. An aqueous solution of Germa Medica (which contains 1-2% hexachlorophene) with a small, undetermined and variable amount of Dettol added is used for scrubbing the floors. The mixture is usually renewed at the end of each day. Floors are mopped after each case and scrubbed more thoroughly at the end of the day. String mops are used and are not decontaminated in any way. After use the mops are stored in the buckets used for the cleaning mixture.

One square inch of floor surface near the base of the operating table was swabbed before and after each mopping. The swabs were shaken for 15 minutes in serum broth containing 5% sterile human blood serum to inactivate the hexachlorophene (Myers and Barth[17]). Aliquots of the broth were then plated on sheep's blood agar and incubated 48 hours aerobically at 37.5° C. before counting colonies.

6. *Scrub water.*—The mixture used for scrubbing the floors, has been described. Serial dilutions of the mixture were prepared and pour plates made of nutrient agar. All pour plates were incubated 48 hours at 37.5° C. before counting colonies.

7. *Mops.*—One inch samples of string from the mops used for scrubbing floors were obtained aseptically. Each sample was immediately immersed in serum broth and

mechanically shaken for 15 minutes. Serial dilutions of the suspension were prepared in serum broth and aliquots of each dilution plated to sheep's blood agar. All cultures were incubated 48 hours at 37.5° C. before counting colonies.

8. *Anæsthesia masks.* — Rubber and plastic masks are used. Masks are not routinely decontaminated except for an occasional washing with Germa Medica. They are kept in a closed metal cabinet until required for use.

Masks were tested for contamination by swabbing the entire inner surface with swabs moistened in sterile nutrient broth. The swabs were then placed in nutrient broth and mechanically shaken for 15 minutes. Aliquots of the suspension were then plated out to sheep's blood agar, and incubated as previously indicated.

9. *Endotracheal tubes.*—These are routinely washed in Germa Medica or pHisohex solutions, soaked in a solution of benzalkonium chloride 1 in 1000, rinsed in tap water and hung in an open cupboard until required for use.

Tubes were removed from the cupboard aseptically and placed in Letheen broth. They were then shaken mechanically for 15 minutes. Aliquots of the suspension were plated to sheep's blood agar, and incubated in the indicated manner.

10. *Stretcher blankets.*—Cotton blankets and sheets are used for covering patients, except infants, in transit to and from the operating theatre. Before entering the theatre the blankets are removed, folded and placed at the bottom of the stretcher. After the operation the blankets are unfolded and the patient is blanketed in the operating theatre. The blankets receive no special treatment except an occasional laundering.

Blood agar plates were used to culture the blankets by the sweep plate technique.[3] Blankets were sampled immediately after the patient had entered the operating theatre. All culture plates were incubated, as indicated above, before counting colonies.

11. *Depots.* — Cupboards, sinks, lamps, operating tables and other fixtures are washed at the end of each day or after dirty (infected) cases, using cotton sponges soaked in aqueous benzalkonium chloride 1 in 1000. The lower parts of the walls are washed once weekly with a solution containing Germa Medica or tincture of green soap. The upper parts of the walls are washed infrequently by the janitorial staff, who are also responsible for cleaning the radiators. Areas inaccessible or hard to reach, such as radiators, high cupboards and the lower parts of furniture, may miss cleaning and are sometimes covered with dust. No dust-laying methods are used. Floor areas beneath immovable furniture frequently escape cleaning.

Swabbing and culturing methods for the above mentioned depot areas were the same as those described for operating theatre floors.

12. *Surgeons' hands.* — Germa Medica (containing 1-2% hexachlorophene) or pHisohex (pHisoderm containing 3% hexachlorophene) are used for all preoperative scrubs. The length of the scrub is determined by the individual surgeon, and may vary from one to 10 minutes depending on the time lapse between scrubs. Nurses follow predetermined scrub routines lasting either three or 10 minutes. Hands and arms are rinsed in tap water, nails cleaned and hands rinsed again, hands and arms are scrubbed using sterile brushes, rinsed in tap water and then in the soap solution. The hands and arms are then dried with sterile towels, gowns are donned, talcum is applied to hands and sterile gloves are donned. Brushes and nail cleaners are sterilized and placed in the operating theatre at the beginning of the day. Tap water is used for all scrubs. Taps have gauze filters and are controlled by knee levers.

Swabs moistened in serum broth were used to take cultures from the surgeons' hands before the preoperative scrub, after the preoperative scrub and after the operation. A moistened swab was streaked three times across the back of the hand and once down each finger and the thumb. The procedure was repeated on the palm of the hand. Both hands were sampled. Swabs were immersed in serum broth. After mechanically shaking for 15 minutes ali-

quots of the suspension were plated to blood agar. All plates were incubated as described before counting colonies.

13. *Patients' skin: preoperative preparation.*—Preoperative preparation of skin in the wards consists of washing and shaving the operative site if necessary the night before the operative procedure.

Preoperative preparation in the operating room routinely consists of swabbing with ether by a "non-sterile" nurse using sterile gauze sponges and forceps. Sterile towels are then placed around the area which is painted with tincture of benzalkonium chloride 1 in 1000 by a "sterile" nurse or surgeon using sterile gauze sponges and forceps. Care is taken to treat all of the operative site. The operative site is draped immediately and the first incision is made. The time lapse between the skin treatment and the incision varies from one to 10 minutes depending on the time required for draping. No effort is made to control this time lapse. The same skin preparation is used before the administration of spinal anæsthetics.

In orthopædic cases an alcohol-iodine treatment is routinely employed. The operative site is swabbed with 50% alcohol before the towels are placed around the site. The area is then swabbed with 2% tincture of iodine in 50% alcohol. After the operation, the iodine is removed with 50% alcohol.

Germa Medica is used for obstetrical cases. The agent is swabbed on in full strength before draping.

Areas of skin approximately one square inch in size were swabbed before skin preparation, after preparation and after operation. Swabs were immediately immersed in broth contained in screw-capped vials, the ends of the swabs were clipped off with sterile scissors and the vials sealed. Aliquots of the broths were plated on sheep's blood agar after the broths had been shaken for 15 minutes. Letheen broth was used to swab skin prepared with tincture of Zephiran, while sodium thiosulphate broth was used to swab skin prepared with tincture of iodine. All cultures were incubated aerobically for 48 hours at 37.5° C. before counting colonies.

14. *Respiratory tract flora of personnel.*—Noses and throats of operating room personnel were sampled with swabs moistened in serum broth. Swabs were streaked on blood agar plates. Colonies of *M. pyogenes var. aureus* were isolated for determination of antibiotic sensitivity, coagulase reactions and bacteriophage typing. Coagulase reactions were determined by the addition of a loopful of culture from a nutrient agar plate or 0.5 ml. of a 24 hour broth culture to 0.5 ml. sterile human plasma. The mixture was examined for clotting after one hour's and three hours' incubation at 37.5° C. All coagulase positive strains were bacteriophage typed.

Results

1. *Air samples.*—Three hundred and five sets of samples were obtained during 105 operations on 35 separate operating days. The results are summarized in Table I. *Micrococcus pyogenes var. aureus** was recovered from 67% of the samples. All strains were bacteriophage typed (see Table II for a list of the bacteriophage pools used). Approximately one-half of the strains were non-typable. The remainder were distributed fairly evenly over a large number of phage types and patterns. *Streptococcus viridans* was recovered from 88% of the samples and *Streptococcus pyogenes* was recovered from 2.5%. No relationship could be established between amount of aerial contamination and either indoor or outdoor temperature and relative humidity. Traffic in and out of the theatre during the operation was excessive in many cases.

2. *Masks.*—One hundred and fifty masks prepared with sterile cotton discs as previously described were sampled *after use.* An average of 250,000 micro-organisms per square inch was found on the inside and 25,000 micro-organisms per square inch on the outside. Eleven strains of *M. pyogenes var. aureus* were isolated from the inside surface of used masks. Eight of these were non-typable, one was lysed by phage type

**Micrococcus pyogenes var. aureus* as defined in *Bergey's Manual of Determinative Bacteriology*, The Williams and Wilkins Company, Baltimore, 6th Edition, 1948.

TABLE I.—Bacterial Contamination of Air in the Operating Theatre

Sampling period	Duration of sampling in minutes	Number of samples taken	Micro-organism/cu. ft. G.E. sampler	Micro-organisms settling out/hour
Theatre unoccupied				
5.00 to 6.00 a.m.	60	34	0.9	
Overnight				12.5
Before first case	56	35	19.5	64.1
During first case	87	35	16.5	49.8
Before second case	48	34	21.2	80.7
During second case	61	34	13.8	48.7
Before third case	49	26	21.2	85.8
During third case	55	26	16.8	47.7
Before fourth case	46	13	19.7	68.8
During fourth case	32	13	14.4	49.5
Before fifth case	43	5	14.9	77.6
During fifth case	35	5	15.6	119.1
After last case	66	35	14.3	74.0

81 and two were lysed by phages 3A/pool B. One strain of *M. pyogenes var. aureus* was isolated from the outside of a used mask and was found to be non-typable.

Forty-five masks taken from the supply in the surgeons' room and tested *before use* had an average of 30,000 micro-organisms per square inch on the inside and 53,000 per square inch on the outside surface.

3. *Splash basins.*—One hundred and twenty-two basins were sampled. One hundred and three remained sterile or the number of micro-organisms present was too small for accurate enumeration. Fourteen of the remaining 19 basins showed slight contamination with an average of 146 organisms per ml., three had over 20,000 per ml. and two had over 30,000 per ml. The following micro-organisms were recovered: aerobic spore-bearers, diphtheroids, coliforms, Actinomycetes, moulds, Gaffkya and Micrococcus. *M. pyogenes var. aureus* was recovered from 11 of the 19 contaminated basins. Eight strains were non-typable, two were lysed by phage pools C1 and C2 and one was lysed by phage pattern 29/Pool C1.

4. *Operating table mats and sheets.*—Average counts for operating table mats were 140 per square inch before the operative procedure and 342 per square inch after the operation. Average counts for sheets were 637 per square inch before the operation and 181 per square inch after. *M. pyogenes var. aureus* was found in five cultures from mats and four from sheets. Each of the following bacteriophage patterns lysed one of the strains: 81/52/52A, 28/52/47/Pool C1/C2, 47/Pool C1/C2. The remainder of the strains was non-typable.

TABLE II.—Bacteriophage Pools Used in this Investigation

Pool A	Bacteriophages	29/52/52A/79
Pool B	"	3B/3C/55
Pool C	"	7/42E/53/54/70/73/75/77
Pool C1	"	6/7/53/54
Pool C2	"	42E/70/73/75/77

5. *Operating theatre floors.*—Thirty-eight cultures of floors *before mopping* had an average of 1265 micro-organisms per square inch of floor surface, while 31 cultures obtained *after mopping* had an average of 10,880 micro-organisms per square inch. Although the number of micro-organisms obtained from the floor before mopping decreased progressively throughout the day, each time the floor was mopped the number of micro-organisms per square inch increased to more than 10,000 per square inch. The majority of the organisms were coliforms. *M. pyogenes var. aureus* was present in two of the 38 cultures obtained before mopping and in four of the 31 cultures obtained after mopping. Two of the latter strains were coagulase positive, one of these was non-typable and the other was lysed by bacteriophage 3A.

6. *Scrub water.*—Sixteen samples of scrub water showed an average contamination of over 74,000,000 micro-organisms per ml. The counts decreased progressively throughout the day. The majority of the micro-organisms recovered were coliforms, although Actinomycetes, Micrococcus and aerobic spore bearers were also present in small numbers in some samples. *M. pyogenes var. aureus* was recovered from five

of the 16 samples. Two of the strains were coagulase positive and both were non-typable with bacteriophage.

7. *Mops.*—Twenty-one samples of mop string gave an average count of over 5 x 10^9 micro-organisms per square inch of fibre. The bacterial count of mops decreased progressively throughout the day. *M. pyogenes var. aureus* (coagulase negative) was found in one of the 21 samples.

8. *Anæsthesia masks.*—Twenty-four masks sampled from the storage cupboard had an average of 1700 micro-organisms per mask. One mask sampled before use had a count of 910 micro-organisms, while another mask sampled after use had 37,600. Micro-organisms recovered from the masks included diphtheroids, moulds, Actinomycetes, Neisseria, Micrococcus and *Streptococcus viridans. Pseudomonas pyocyanea* was recovered from one of the masks from the storage cupboard. Coagulase positive *M. pyogenes var. aureus* was recovered from six of the 24 masks sampled. One strain was weakly lysed by bacteriophage 52 and the other five strains were non-typable.

9. *Endotracheal tubes.* — Sixteen endotracheal tubes taken from the storage cupboard had an average of 6000 micro-organisms per tube. After 24 hours' incubation in Letheen broth, two tubes yielded coagulase positive *M. pyogenes var. aureus*, two contained *Ps. pyocanea* and two contained coliforms.

10. *Stretcher blankets.*—A total of 72 stretcher blankets had an average of 132 micro-organisms per square foot of blanket surface. The time of sampling had no apparent effect on the degree of bacterial contamination. Micro - organisms which grew in the cultures included moulds, aerobic spore bearers, diphtheroids, yeast, Actinomycetes, Gaffkya, Micrococcus, Pseudomonas and Streptococcus. *Ps. pyocyanea* was recovered from one blanket, *Streptococcus viridans* was recovered from 36 blankets, and *M. pyogenes var. aureus* was recovered from 51 of the 72 blankets. Coagulase tests were done on 32 of the *M. pyogenes* strains; of these 22 were coagulase positive. Thirty-eight of the *M. pyogenes* strains were tested for bacteriophage type; of these 14 were non-typable. Four strains were lysed by phage type 81, two by phage type 3A and six by phage pattern 3A/Pool B. The remainder of *M. pyogenes* strains were lysed by a variety of phage patterns.

11. *Depots.*—One hundred and ninety-two cultures taken from 15 different locations in the operating theatre averaged 1000 micro-organisms per square inch of depot surface. Floors, radiators, the bases of operating tables, lamps and walls were found to be heavily contaminated with average counts higher than 1000 per square inch. Cupboards for anæsthesia equipment, sinks, soap dishes, windows and x-ray viewers were less heavily contaminated with counts between 500 and 1000 micro-organisms per square inch. Anæsthesia cabinets, air conditioning units, supply cupboards and sponge racks had the least degree of contamination with counts between 100 and 500 per square inch. Sixteen of the cultures contained *M. pyogenes var. aureus* and 12 contained *Streptococcus viridans.*

12. *Surgeons' hands.*—Of 196 cultures obtained from surgeons' hands after the scrub 39 contained *M. pyogenes var. aureus.* The average count on surgeons' hands before the scrub was 3000 per swab; after the scrub the count was 1700 per swab and after the operation 1300 per swab. In seven instances where a second preoperative scrub was done, samples taken from the surgeons' hands after this scrub averaged 5000 micro-organisms per swab. Of these seven samples three contained *M. pyogenes var. aureus.* Thirty-three strains of *M. pyogenes var. aureus* taken from surgeons' hands were phage typed; of these 24 were non-typable and the remainder were lysed by a variety of phage patterns. Diphtheroids, aerobic spore bearers, Neisseria, Gaffkya, Micrococcus and Actinomycetes were found on surgeons' hands.

13. *Patients' skin.*—A limited number of cultures were obtained from the patients' skin before and after the operative procedure. Cultures showed that the skin was not sterilized by the preoperative preparation but that the number of micro-organisms present was reduced. No increase in

TABLE III.—Incidence of Carriers of *Micrococcus pyogenes var. aureus** Among Operating Theatre Personnel

Personnel	No. of subjects	No. positive for *Micrococcus pyogenes var. aureus*		Total carriers	
		Coagulase positive	Coagulase negative	No.	%
Nurses	38	10	3	13	39.5
Interns	20	6	1	7	35.0
Surgeons	31	12	3	15	48.4
Anæsthetists	9	2	2	4	44.4
Orderlies	3	2	0	2	66.7
Secretaries	3	2	1	3	100.0
Students	7	3	0	3	42.9
Total	111	37	12	47	
% carriers		33.3	10.7		42.0

**Micrococcus pyogenes var. aureus* as defined in *Bergey's Manual of Determinative Bacteriology.*

degree of skin contamination was detected during the operation.

14. *Respiratory tract flora of personnel.*—Nose and throat swabs from 111 operating theatre personnel showed an average carrier rate of 42% for *M. pyogenes var. aureus.* Thirty-three per cent of the personnel were carriers of coagulase positive strains of *M. pyogenes.* The percentage of respiratory carriers of pathogens among the staff increased with the duration of service in the hospital. *Streptococcus pyogenes* (Lancefield Group A) was isolated only once and then from the throat of an anæsthetist. Micro-organisms of the coliform group were isolated from the nose cultures of 10 subjects and the throat cultures of three subjects. Yeast was isolated from the throat culture of one surgeon. The results of these examinations are given in Table III.

Discussion

Air samples taken in the operating theatres showed a degree of bacterial contamination approximately double the maximum suggested by Girdlestone and Bourdillon[7] for major operations. More rigid control of traffic in and out of the operating theatre was indicated, and restrictions are now enforced. Air conditioning units installed late in the investigation increased bacterial contamination of the air, probably because particulate matter disturbed during cleaning of the theatre was prevented from "settling out" and the air was recirculated without filtration.

No fault was found in the construction of face masks.

Contamination of the contents of splash basins was evident. Obviously the basins should not be exposed until the last moment before use and should be replaced at frequent intervals. These suggestions are now in effect.

Special care should be taken in decontaminating the operating table mats since Beck and Collette[2] and Colbeck[5] have demonstrated that micro-organisms can easily pass through sheets contaminated with blood or exudates, and our results show contamination of the mats with pathogenic micro-organisms.

The revision of cleaning techniques is clearly indicated. In some instances the procedures used constitute a direct source of contamination, e.g. scrub water and mops used for floors, depots which escape cleaning.

Anæsthesia masks and endotracheal tubes showed contamination with pathogenic bacteria including *M. pyogenes var. aureus.* Obviously the sanitizing procedures employed were inadequate. The method of decontamination for anæsthesia masks and endotracheal tubes has now been revised.

The presence of pathogenic bacteria emphasizes the necessity for sterilization of blankets. Blankets are now sterilized by autoclave.

With regard to the preoperative scrub by surgeons and the preparation of the patients' skin, it should be noted that hexachlorophene soaps are effective only if used exclusively and repeatedly[20] and that thorough scrubbing is still essential.[21] The inactivating effect of human serum on hexa-

chlorophene[17] throws doubt on the validity of the use of this antiseptic in the operating theatre. Since this investigation, the surgeons' preoperative scrub has been lengthened to 10 minutes.

Starkey[24] and McDermott[18] emphasized the ubiquitous character of the micro-organisms currently involved in hospital infections. In the absence of any evidence of an outbreak of infection and under normal working conditions we have isolated pathogenic micro-organisms, including *Micrococcus pyogenes var. aureus*, from the following sources in the operating theatre: the air, face masks, splash basins, operating table mats and sheets, floors, scrub water used for floors, mops, anæsthesia masks, endotracheal tubes, stretcher blankets, a variety of depot surfaces, surgeons' hands, the patients' skin and the respiratory tract of the personnel of the operating theatre.

Our results indicate that the aseptic and antiseptic techniques currently practised in the operating theatres of modern hospitals are frequently inadequate. Reassessment of techniques and more rigid application of the established principles of asepsis and antisepsis are required by the entire operating theatre personnel from the janitorial staff scrubbing the floor to the surgeon performing the preoperative scrub.

Summary

The following possible sources of contamination in the operating theatre have been bacteriologically investigated: air, face masks, splash basins, operating table mats and sheets, floors, scrub water for floors, mops, anæsthesia masks, endotracheal tubes, stretcher blankets, depots, surgeons' hands, patients' skin, respiratory tract flora of personnel.

A high degree of contamination was found in the air, floors, scrub water for floors, mops, certain depots, and surgeons' hands. Occasionally, samples from splash basins, endotracheal tubes and anæsthesia masks also showed a high degree of contamination.

Pathogenic micro-organisms including *M. pyogenes var. aureus* were among the contaminators found in the air, masks, splash basins, operating table mats and sheets, floors, scrub water for floors, mops, anæsthesia masks, endotracheal tubes, stretcher blankets, depots, surgeons' hands, patients' skin and respiratory tract flora of personnel.

The carrier rate for *M. pyogenes var. aureus* increases with duration of employment of surgeons and other operating room personnel.

Aerial contamination increased markedly during individual operations and throughout the day.

Aseptic and antiseptic techniques employed were shown to be inadequate, and as a result these procedures have been revised and more rigid control established.

Acknowledgments

The authors gratefully acknowledge the valuable assistance given by the technical staff of the Alberta Public Health Laboratory who performed the bacteriophage typing of organisms mentioned in this paper.

References

1. Anderson, W. T.: Ultraviolet air disinfection, *Arch. Phys. Med.*, **28:** 705, 1947.
2. Beck, W. C. and Collette, T. S.: False faith in the surgeon's gown and surgical drape, *Am. J. Surg.*, **83:** 125, 1952.
3. Blowers, R. and Wallace, K. R.: The sterilization of blankets with cetyl trimethylamine bromide, *Lancet*, **1:** 1250, 1955.
4. Bourdillon, R. B., McFarlan, A. M. and Thomas, J. C.: Air-borne bacteria in operating theatres. *In*: Studies in Air Hygiene, Medical Research Council Special Report Series No. 262, His Majesty's Stationery Office, London, 1948, p. 241.
5. Colbeck, J. C.: Studies in hospital infections. 1: The importance of fomites in the spread of staphylococcal infections with particular references to mattresses and washing facilities, *Canad. Serv. M. J.*, **12:** 563, 1956.
6. Corry, D. C.: Cleanliness of surgeons' aprons, *Lancet*, **1:** 909, 1950.
7. Girdlestone, G. R. and Bourdillon, R. B.: Infection in "clean" surgical wounds by the surgeon and from the air, *Lancet*, **1:** 597, 1951.
8. Goodman, J. M. *et al.*: The effect of ultraviolet radiation and air conditioning upon the air sterility in a closed surgery, *Surgery*, **25:** 284, 1949.
9. Hart, D.: Sterilization of the air in the operating room by bactericidal radiant energy, *Arch. Surg.*, **37:** 956, 1938.
10. Hart, D.: Pathogenic bacteria in the air of operating rooms, *Arch. Surg.*, **37:** 521, 1938.
11. Hart, D.: The importance of air-borne pathogenic bacteria in the operating room, *J. A. M. A.*, **117:** 1610, 1941.

12. JOSEPH, J. M.: Disease transmission by inefficiently sanitized anesthetizing apparatus, *J. A. M. A.*, **149:** 1196, 1952.
13. LUCKIESH, M., TAYLOR, A. H. AND HOLLADAY, L. L.: Sampling devices for air-borne bacteria, *J. Bact.*, **52:** 55, 1946.
14. LUCKIESH, M., HOLLADAY, L. L. AND TAYLOR, A. H.: Sampling air for bacterial content, *General Electric Review*, **49:** 8, 1946.
15. LUCKIESH, M. AND TAYLOR, A. H.: Determining and reducing the concentration of air-borne micro-organisms, *Heating, Piping and Air-conditioning*, **19:** 113, 1947.
16. LUCKIESH, M., TAYLOR, A. H. AND KNOWLES, T.: Sampling devices for determining the bacterial content of air, *Rev. Scient. Instruments*, **20:** 73, 1949.
17. MYERS, G. E. AND BARTH, N. K.: Inactivation of hexachlorophene by human serum and dead bacteria, *Canad. Pharm. J.*, **91:** 63, 1958.
18. MCDERMOTT, W.: The problem of staphylococcal infection, *Brit. M. J.*, **2:** 837, 1956.
19. POPPE, J. K.: Wash basin contamination in operating rooms, *Arch. Surg.*, **44:** 103, 1942.
20. PRICE, P. B. AND BONNETT, A.: The antibacterial effects of G-5, G-11 and A-151 with special reference to their use in the production of a germicidal soap, *Surgery*, **24:** 542, 1948.
21. PRICE, P. B.: Fallacy of a current surgical fad—the three-minute preoperative scrub with hexachlorophene soap, *Ann. Surg.*, **134:** 476, 1951.
22. PROPST, H. D.: Effect of bacterial agents on the sterility of surgical linen, *Am. J. Surg.*, **86:** 301, 1953.
23. ROBERTSON, E. C. AND DOYLE, M. E.: On the control of air-borne bacteria in operating rooms and hospital wards, *Ann. Surg.*, **111:** 491, 1940.
24. STARKEY, H.: Control of staphylococcal infections in hospital, *Canad. M. A. J.*, **75:** 371, 1956.
25. STUART, L. S., ORTENZIO, L. F. AND FRIEDL, J. L.: Use of dilution confirmation tests for results obtained by phenol coefficient methods, *Assoc. Off. Agric. Chem.*, **36:** 466, 1953.
26. ZEIGLER, C. AND JACOBY, J.: Anesthetic equipment as a source of infection, *Anes. & Analg.*, **451:** 35, 1956.

RÉSUMÉ

Une récente augmentation des infections post-opératoires à *Micrococcus pyogenes var. aureus* a déterminé l'accomplissement de l'enquête dont les résultats sont donnés ici.

Dans le service opératoire d'un hôpital de 1000 lits, les facteurs suivants furent successivement étudiés:

1. *L'air.*—Des échantillons d'air ont été prélevés dans les salles d'opération avant ou pendant les interventions, et mis en culture sur agar au sang, pendant 48 heures; les colonies étaient alors comptées.

2. *Les masques opératoires.*—Des échantillons y furent découpés avant et après usage et mis en culture.

3. *Les bassins.*—Il s'agit ici des bassins en acier inoxydable, qui, après stérilisation à l'autoclave, sont utilisés pour le rinçage à la solution physiologique des instruments, des éponges et des gants du chirurgien. Des échantillons du contenu furent prélevés à divers temps pendant une opération et cultivés.

4. *Matelas et linges de table d'opération.*—Ces objets furent lavés sur certaines surfaces avec du milieu de culture: le liquide recueilli fut mis en culture sur agar au sang.

5. *Planchers des salles d'opération.*—Les planchers sont lavés après chaque opération et plus spécialement à la fin de la séance opératoire avec un liquide désinfectant; cependant, les serpillières utilisées à cette fin ne sont jamais stérilisées. On préleva des échantillons sur des surfaces d'un pouce carré, à proximité immédiate de la table d'opération et ceux-ci furent mis en culture.

6. *Le liquide de récurage des planchers*, précédemment décrit fut aussi testé: des solutions de plus en plus étendues furent préparées et mises en culture.

7. *Les serpillières* utilisées à la même fin furent examinées par prélèvements de fragments, lavage de ces fragments dans du sérum et mise en culture du liquide obtenu.

8. *Masques d'anesthésie.*—Ces masques en plastique ou en caoutchouc ne sont pas désinfectés de façon systématique. Ils furent examinés par lavage de leur surface interne dans du sérum, et mise en culture du liquide obtenu.

9. *Tubes endotrachéaux.*—Ceux-ci sont systématiquement nettoyés après usage par immersion dans une solution désinfectante, puis rincés à l'eau du robinet et gardés dans un placard. Les tubes furent lavés au bouillon de culture, et la suspension obtenue mise en culture.

10. *Couvertures.*—Ces couvertures, utilisées pour couvrir le malade lors des transports, ne sont pas désinfectées, mais simplement envoyées au lavage. Des échantillons y furent également prélevés et mis en culture.

11. *L'entretien général.*—Les armoires, placards, éviers, lampes, tables et l'ensemble du mobilier sont généralement lavés à la fin de chaque journée opératoire avec une solution désinfectante. Tout ceci fut contrôlé par prélèvement d'échantillons mis en culture.

12. *Les mains du chirurgien.*—Ce facteur fut examiné par frottage d'un tampon imbibé de milieu de culture sur la paume et la partie dorsale de chaque main, avant, pendant et après l'intervention. Le liquide obtenu fut mis en culture.

13. *La peau du patient.*—Des surfaces cutanées d'environ un pouce carré furent lavées au bouillon de culture avant, pendant et après l'intervention; comme précédemment le liquide obtenu fut mis en culture.

14. *La flore du tractus respiratoire* du personnel fut examinée par prélèvement d'échantillons par frottis dans les cavités nasales et la gorge.

Les résultats de toutes ces investigations montrent que le nombre de germes se situe aux environs du double du maximum de tolérance proposé par Girdlestone et Bourdillon. Une des plus importantes sources de contamination réside dans l'eau des bassins de rinçage et dans les matelas de table d'opération. D'autres sources, également importantes sont: l'air, les planchers, les serpillières, les mains du chirurgien et le tractus respiratoire du personnel.

En conclusion, un certain nombre de pratiques de désinfection sont à modifier ou à changer radicalement.

FAT EMBOLISM
AN EXPERIMENTAL STUDY USING QUARTZ ROD MICROSCOPY*

T. D. R. BRIANT, M.D. and W. ROBERT HARRIS, M.D., F.R.C.S.[C], *Toronto*

Introduction

A certain degree of fat embolism appears to be common after any injury to the skeleton.[1] The remarkable fact is that only rarely does the embolism produce clinically recognizable signs and symptoms. While no adequate explanation has been given for this, many observers have noted experimentally that the minimum lethal dose (MLD) of fatty acids is far less than that of neutral fats and have concluded that clinical fat embolism is caused by the breakdown of ordinarily neutral marrow fat with release of fatty acids. It has been suggested that the toxicity of the fatty acids is due to the direct chemical action of the acid on the tissue it comes in contact with.[2] Support for this view is gained from the well-documented observation that hæmorrhagic consolidation of the lungs is very marked after an intravenous injection of fatty acid, while after the administration of neutral fat the lungs remain relatively pale and dry[2, 3] (Fig. 1).

Unfortunately, study of the action of fat emboli in the body is made difficult because the standard frozen section technique for the examination of fat in tissue sections is cumbersome and may be grossly inaccurate. Further, it gives no information about the intravascular flow of fat which might explain the different behaviour of neutral fat and fatty acids. For this reason, we have adapted the quartz rod technique to the study of the pulmonary circulation *in vivo* and have used it to compare the fate of neutral fat and fatty acid injected into the blood stream.

Material and Methods

The method used was essentially that described by Irwin *et al.*[4] Adult rabbits weighing approximately two kilograms were used. Under Nembutal anæsthesia a tracheotomy was performed and a polyvinyl tube inserted into the trachea. Through the same incision one of the jugular veins was also cannulated with a polyvinyl tube. This was subsequently used both to maintain the intravenous Nembutal anæsthesia, and/or injections of fat. Oxygen was then introduced into the tracheotomy tube through a smaller polyvinyl tube. This provided a simple by-pass mechanism which prevented over-distension of the lungs. By adjusting the oxygen flow (to about three litres per minute in the average animal) the animals could be made apnœic. The resulting respiratory paralysis was complete, and allowed satisfactory microscopic observations of the lung to be made. The right side of the chest was then opened between the fourth and fifth ribs, care being taken to ligate the intercostal vessels both proximally and distally. This was an important step, as hæmorrhage from these vessels often produced shock phenomena with sludging of red cells in the pulmonary circulation. The anterior margin of the middle lobe of the lung was then carefully suspended over the tip of a cannulated quartz rod. The end of this rod was made as small and as flat as possible, so as to concentrate the light properly, but not so small as to tear the lung. The exposed surfaces of the lung were then packed off with gauze moistened with warm Ringer's solution, and that part of the lung being examined was kept moist with warm (37° C.) Ringer's solution introduced both from an overhead drip and from the cannula in the quartz rod (Fig. 2). This step was very important, as the lung proved to be very sensitive both to drying and to temperature change, either of which caused "sludging" of red cells and spoiled the experiment. Cardiac movements could be "damped" by packing off the heart with Saran wrap.

The pulmonary circulation was then studied with a binocular dissecting microscope, fitted with an attachment which allowed the observations to be recorded on cine film. Various animals were then injected with either neutral fat (human or rabbit marrow fat or corn oil) or fatty acid (oleic acid). The behaviour of the resulting fat emboli in the pulmonary vessels could then be studied with the microscope. Preliminary experiments showed that the LD 50 of neutral fat is 1.8 ml. per kg., while that of fatty acid is 0.6 ml. per kg. By keeping the dosage below these

---

*From the Department of Anatomy, University of Toronto, and the Division of Orthopædic Surgery, Toronto General Hospital. Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Montreal, October 1957.

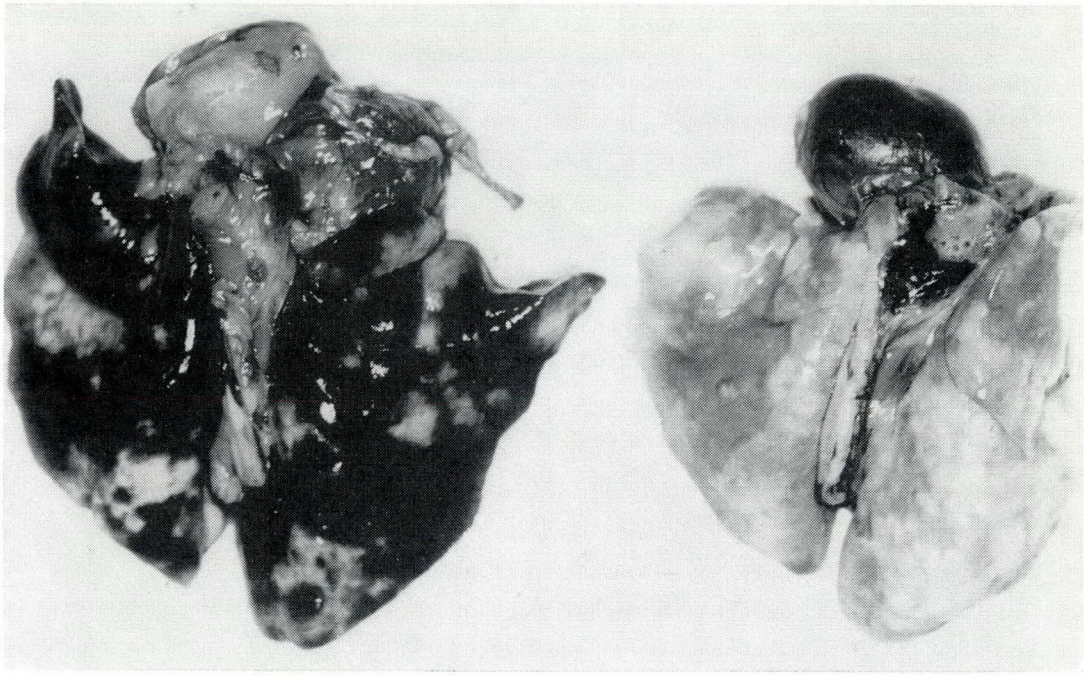


Fig. 1.—Comparison of the effects of fatty acid and neutral fat in the lungs of rabbits given lethal doses of each type. Fatty acid (left) produces severe hæmorrhagic consolidation while neutral fat (right) leaves the lungs relatively pale and dry.

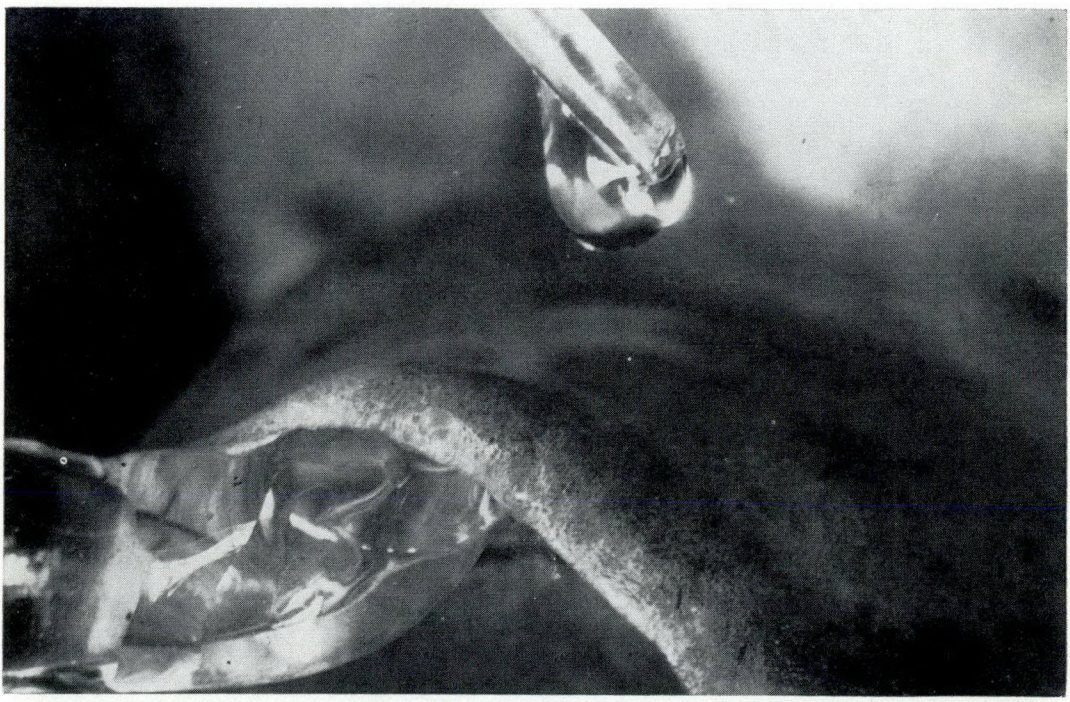


Fig. 2.—Close-up view of the edge of the lung suspended on the tip of the quartz-rod below. The overhead drip keeps the surface of the lung moist with warmed Ringer's solution.

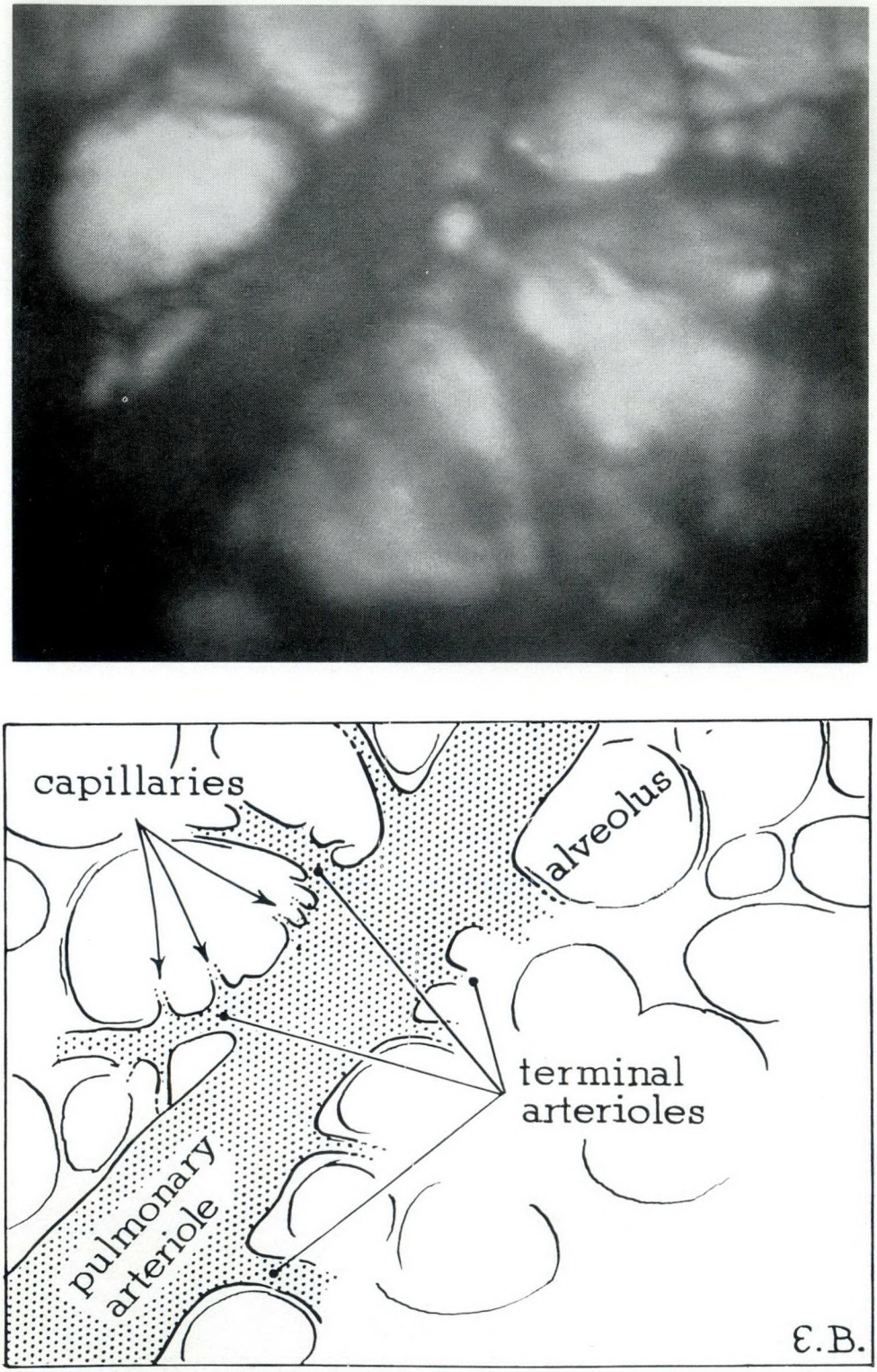


Fig. 3.—Diagram, based on a frame of cine film (above), to illustrate the anatomical features of the arterial circulation in the lung. Note the sudden diminution in calibre of the terminal arterioles in contrast to that of the pulmonary arterioles.

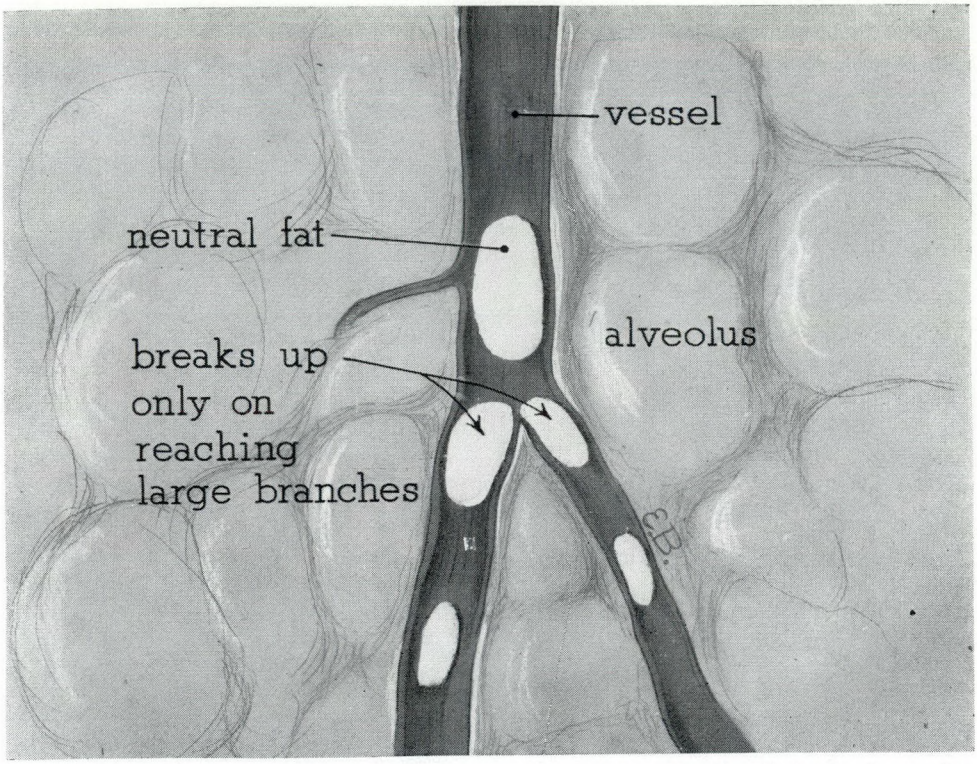


Fig. 4.—Behaviour of neutral fat in the lung (traced from the cine film). The fat persists in large droplets which occlude the pulmonary arterioles.

levels, and by injecting the fat slowly, the animals could be kept alive for a considerable period of time so that in many instances observations were made over a period of 10 or 12 hours.

Observations

A. *Anatomy of the Small Pulmonary Vessels*

The pulmonary artery terminates in a blunt pulmonary arteriole which ranges from 40 to 140 micra in diameter. The reason for the bluntness of these vessels is that their branches, the terminal arterioles, are very much smaller in diameter (Fig. 3). These terminal arterioles arise throughout the length of the pulmonary arterioles either singly or in pairs, and immediately enter the alveolar septa. They range from 15 to 40 micra in diameter. From them branch capillaries which are difficult to observe *in vivo*, but which appear to form a complicated anastomosing network over the surface of the alveolus.

Both the pulmonary and terminal arterioles were contractile and were often seen both dilating and contracting several times during a single experiment.

B. *Behaviour of Injected Fats*

A striking difference was found in the physical behaviour of neutral fat as compared to fatty acid. The neutral fat remained in rather large droplets, which broke up with difficulty. This was shown by the fact that the droplets were usually compressed into a sausage shape by the narrow calibre of the blood vessels, and that on reaching a branch or bifurcation in the vessel, all of the drop tended to pass down one or other branch, or if it did break up, it divided into two still relatively big drops (Fig. 4). Eventually the drop would lodge in a pulmonary arteriole approximately 70 micra in diameter. Finally most of the vessel proximal to the occlusion would be plugged with fat which retained a pulsatile movement for one or more hours. Little if any fat was observed breaking off from this main mass and entering the terminal arterioles. The adjacent lung bed

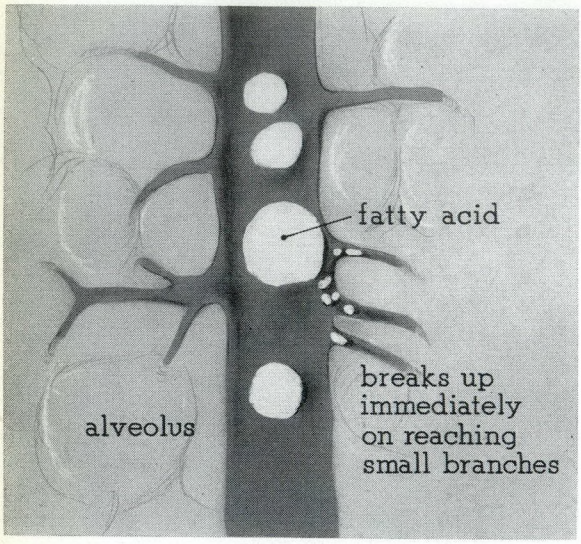


Fig. 5.—Behaviour of fatty acid in the lung (traced from the cine film). The fat breaks up into small droplets, which occlude the smaller (terminal) arterioles.

became ischæmic, and no flow was seen in the alveolar capillaries. Flow did continue, however, through those branches of the pulmonary arteriole proximal to the occluding column of fat.

By contrast, fatty acid appeared in smaller droplets which retained a spherical shape within the pulmonary arterioles. On reaching the branches (i.e. terminal arterioles) they fragmented into a multitude of smaller droplets all of which passed readily into the terminal arterioles and subsequently into the capillaries (Fig. 5). Quite a large quantity of fatty acid could thus be injected before fat began to back up in the pulmonary arteriole. Following embolization of the terminal arteriole, the segment supplied by it (usually two or three alveoli) became deep red as the result of hæmorrhage into the alveolar spaces.

Discussion

We feel that the key to the explanation of the difference in behaviour of the two kinds of fat lies in the sudden diminution in calibre between the pulmonary and terminal arterioles. This anatomical peculiarity was first observed by Knisely,[5] who referred to the pulmonary arterioles as the "vascular traps" of the lung. Particles of large size are trapped in the larger pulmonary arterioles, producing ischæmia of the adjacent alveolar bed. But as the proximal branches of the pulmonary arteriole are not necessarily occluded, reduced circulation to the alveolar bed may continue with survival of the animal. Conversely, smaller particles pass through the terminal arteriole and lodge in the alveolar capillaries with far more serious effects. Not only is oxygenation interfered with, but also the obstruction appears to produce enough backpressure to rupture the capillary walls with hæmorrhage into the alveoli.

It appears, therefore, that the different behaviour of neutral fat and fatty acid may not be entirely due to difference in their chemical activity, but to difference in their particle size. It is likely that difference in viscosity between the two types of fat accounts for this difference in their physical behaviour. Contrary to the theory advanced by Peltier[1] we saw no evidence of neutral fat being broken down into smaller particles during the 10 to 12 hour periods of observation.

Another observation that can be made from this experiment relates to the possibility of treating fat embolism by systematically administering fat solvents or emulsifiers. The fact that there is a stagnant column of fat proximal to the site of embolization makes it highly improbable that the obstructing fat could be broken up or dissolved by some material in the blood stream. We are, therefore in agreement with Peltier[1] in feeling that attempts to treat fat embolism by this means would be futile.

Summary

Using the quartz rod transillumination method, a comparison has been made of the effects of neutral fat and fatty acid on the pulmonary circulation. Neutral fat remains in large drops that occlude the larger (pulmonary) arterioles. By contrast, fatty acid breaks up readily into smaller droplets which immediately enter the capillaries in the alveolar bed with much more harmful effects. It was felt that this difference in behaviour was a physical effect rather than a chemical one.

The manner in which either type of fat occludes the vessels makes it highly un-

likely that fat embolism could be successfully treated by intravenously administered fat solvents or emulsifiers.

ACKNOWLEDGMENTS

The authors wish to express their gratitude to Dr. John W. Irwin, Director of the Microcirculatory Laboratory, Massachusetts General Hospital, who extended to us the hospitality of his laboratory in order that we might learn his technique for the study of the pulmonary circulation *in vivo*. We also wish to thank Dr. Sylvia H. Bensley, of the Department of Anatomy, University of Toronto, whose long experience with the quartz rod technique was of invaluable assistance to us.

This research was supported by a grant from the National Research Council of Canada.

REFERENCES

1. PELTIER, L. F.: An appraisal of the problem of fat embolism, *Internat. Abstr. Surg.*, **104**: 313, 1957.
2. PELTIER, L. F.: The toxic properties of neutral fat and free fatty acids, *Surgery*, **40**: 665, 1956.
3. HARRIS, R. I., PERRETT, T. S., AND MACLACHLIN, A.: Fat embolism, *Ann. Surg.*, **110**: 1095, 1939.
4. IRWIN, J. W. *et al.*: Microscopical observations of the pulmonary arterioles, capillaries and venules of living guinea pigs and rabbits, *Anat. Rec.*, **119**: 391, 1954.
5. KNISELY, W.: Mammalian lung vascular anatomy, Ph.D. Thesis, Duke University, Durham, North Carolina.

RÉSUMÉ

Les embolies graisseuses apparaissent comme une complication banale de tout traumatisme squelettique; pour des raisons évidentes, l'étude précise de ces lésions est très difficile chez l'homme.

Les auteurs ont repris ce problème du point de vue expérimental avec une nouvelle méthode d'observation. Une thoracotomie droite pratiquée chez un lapin anesthesié et convenablement préparé permet l'examen d'une languette de poumon assujettie à l'extrémité d'une baguette de quartz; cette baguette donne une transillumination de l'organe suffisante pour permettre l'observation microscopique du tissu vivant. On pratique ensuite des injections intraveineuses de graisse neutre et d'acides gras. Des enregistrements cinématographiques furent faits.

Une différence notable se montre dans le devenir des deux types de graisses utilisées: les graisses neutres forment des gouttelettes de suspension relativement grosses et ont peu de tendance à se fractionner, et on les voit obstruer des vaisseaux assez gros. Les acides gras au contraire s'émulsionnent beaucoup plus finement et leurs gouttelettes arrivent jusqu'au niveau des capillaires intra-alvéolaires. On conçoit alors que la gravité des lésions obtenues sera beaucoup plus grande dans le second cas. De toute façon cette action est liée à la structure physique des substances et non à leurs propriétés chimiques.

FAT EMBOLISM*

"Recent work on fat embolism has removed it from the limbo of long neglect. The existence of the syndrome is no longer subject to debate. It has now been defined, its incidence established, and its mechanism reasonably well demonstrated; means of confirming its diagnosis are available; and principles of treatment are emerging.

"Peltier defines fat embolism as probable when fat globules of 10 to 15 micra can be demonstrated in the circulating blood. Recently, Musselman and his co-workers demonstrated that there was enough fat in the femur of an animal to kill it; that fat embolism might be expected in about one-half of all persons who were injured moderately or severely; and that significant symptoms would develop in at least one-third of patients with fat embolism.

"Since the percentage of cases called 'fat embolism' by clinicians is much lower than this, there must be some discrepancy between points of view. There is. The research chemist is now able to diagnose morbidity due to abnormal circulating fat within minutes after injury occurs. Clinicians have been waiting until patients died to establish this diagnosis. Differences in standards lead to such contradictory statements as those from Kuntscher, 'Fat embolism is the most common complication of medullary nailing', and from the Seminar on Medullary Nailing of the American Academy of Orthopedic Surgeons, 'Fat embolism did not occur'. In the latter report, the very next paragraph states, 'Nearly all medullary nailings develop an unexplained postoperative fever . . . Thirty-two other complications occurred'. This convenient catchall undoubtedly includes some of the results of fat embolism such as cardiac failure, pneumonia, cerebral apoplexy, suspected tetany, subdural hematomas not found by trephining, convulsions and deaths for which an adequate postmortem was not performed."

*PIPKIN, G.: The early diagnosis and treatment of fat embolism. *In*: Clinical Orthopedics. No. 12—Rehabilitation. Edited by Anthony F. DePalma. J. B. Lippincott Company, Philadelphia and Montreal, 1958.

LES CYLINDROMES DES GLANDES SALIVAIRES PRESENTATION DE CINQ CAS

SABIN PLOURDE, M.D.* et JEAN-LOUIS BONENFANT, F.R.C.P.[C], *Québec, P.Q.*

BILLROTH[4] (1859) fut le premier à faire mention du cylindrome. Il appliqua en effet ce nom à une tumeur du sinus paranasal, envahissant l'orbite, qu'il décrivit comme étant constituée de cylindres remplis de cellules épithéliales et de corps ovoïdes lesquels sont entourés par des cordons de tissu conjonctif hyalin. D'autres noms ont été par la suite suggérés pour désigner ces tumeurs, tels que: adéno-épithéliome, tumeur mixte cellulaire, épithélioma basocellulaire à stroma hyalin, adénocarcinome malin et "adenoid cystic carcinoma". Le terme de cylindrome, qui s'inspire de l'aspect morphologique, est resté cependant d'emploi le plus courant.

Cliniquement, les cylindromes diffèrent des tumeurs franchement bénignes par leurs nombreuses récidives, et des tumeurs mixtes, par leurs métastases à distance, principalement pulmonaires. Ces caractères évolutifs du cylindrome n'ont pas empêché Bauer et Fox[2] en 1945, de le considérer comme une véritable tumeur bénigne et Kramer et Som,[9] en 1939, comme une variété de tumeurs mixtes. Plus récemment, Kirklin,[8] (1951) de la Clinique Mayo, préférait considérer comme un véritable cylindrome, toute tumeur mixte qui contient des plages vraiment cylindromateuses, étant donné que ces tumeurs avaient une évolution clinique superposable à celles qui étaient entièrement cylindromateuses.

Avec Wawro et McAdams[14] (1954), nous préférons considérer les cylindromes comme des tumeurs nettement malignes qui se rapprochent des adéno-carcinomes caractérisés "par la lenteur de leur envahissement local, des métastases tardives aux ganglions et à distance, et par leur résistance à la radiothérapie".

OBSERVATIONS

1er cas.—J.U.T. âgé de 50 ans, consulta au début de février 1949, pour une tuméfaction, à la région sous-maxillaire et cervicale droite, qui avait débuté trois ans plus tôt. Cette tuméfaction, qui avait augmenté progressivement de volume, mesurait 4 x 3 cm., était dure, bosselée et adhérente à la peau et aux plans profonds. Elle fut complètement excisée et, par suite du diagnostic histologique de cylindrome salivaire, on compléta l'intervention par un évidement ganglionnaire cervical droit. Aucun ganglion lymphatique ne contenait des foyers métastatiques.

Six mois plus tard, on réséqua un nodule tumoral de 1.5 cm. dans la cicatrice cervicale. Cette tumeur récidiva par la suite, à trois autres reprises et chaque fois, le traitement fut chirurgical. En 1954, la tumeur cylindromateuse envahit le pôle supérieur du corps thyroïdien et on retrouva des nodules sous-cutanés à la fourchette sternale. L'examen radiologique pulmonaire montra de nombreux foyers métastatiques disséminés à travers les deux plages pulmonaires.

Le patient mourut en 1957 à la suite de nouvelles récidives traitées par la rœntgenthérapie. Cette tumeur salivaire avait évolué durant onze ans.

2ième cas.—C.H.D., homme de 36 ans, fut admis à l'hôpital pour une tumeur de la glande sous-maxillaire droite de la grosseur d'une olive. L'excision chirurgicale de la glande et de la tumeur fut suivie de radiothérapie par suite du diagnostic histologique de cylindrome salivaire avec envahissement des gaines périneurales. Neuf mois plus tard, on fit l'ablation de deux ganglions cervicaux droits en partie détruits par des foyers métastatiques. Le patient reçut de nouveau de la radiothérapie. Il décéda presque subitement à 41 ans, soit cinq ans après le début de la tumeur, d'une métastase cérébrale confirmée à l'autopsie.

3ième cas.—A l'âge de 33 ans (février 1951), A.B. fut admis à l'hôpital pour une tuméfaction rétro-auriculaire droite, dure, adhérente aux plans profonds et dont le début remontait à deux ans. Des douleurs aiguës et lancinantes rendaient la mastication très difficile.

L'excision complète de la glande parotide droite et de la tumeur fut faite, avec un diagnostic histologique de cylindrome de la glande parotide. Des récidives apparurent alors à cinq reprises différentes et furent traitées par la chirurgie et la radiothérapie. Le néoplasme

*Hôpital Hôtel-Dieu de Québec, Québec, P.Q.

TABLEAU I.

Cas	*Sexe*	*Age*	*Localisation*	*Evolution pré-opératoire*	*Volume de la tumeur*
J.U.T.	M	50	Glande sous-maxillaire droite	3½ ans	4 x 3 cm.
C.H.D.	M	36	Glande sous-maxillaire droite		2.5. cm.
A.B.	M	33	Glande parotide droite	2 ans	
F.B.	M	76	Lèvre supérieure gauche	1 an	1 cm.
L.D.	M	46	Lèvre supérieure droite		1.5 cm.

finalement envahit la mastoïde, le conduit auditif externe, le nerf facial et le pavillon de l'oreille.

Le patient mourut en octobre 1954, 4½ ans après l'apparition du néoplasme. On ne put cependant déceler de métastases à distance.

4ième cas.—F.B., vieillard de 76 ans, consulta en 1955, pour une tuméfaction de 1 cm. de diamètre qui s'était développée à la face interne et latérale gauche de la lèvre supérieure. Cette tumeur fut largement enlevée et le diagnostic histologique fut celui de cylindrome de type salivaire.

Ce patient se porte encore très bien et aucune récidive n'a été constatée jusqu'à présent.

5ième cas.—Un homme de 46 ans, (L.D.) consulta récemment pour une petite tuméfaction à la face interne latérale droite de la lèvre supérieure. A la suite de l'ablation chirurgicale, un diagnostic de cylindrome salivaire fut porté.

Sexe et Age

Les cinq cas de cylindrome rapportés ici se sont développés chez des hommes de 33 à 76 ans, donnant ainsi une moyenne d'âge de 48 ans. Wawro et McAdams,[14] dans une série plus importante (18 cas) ont constaté que cette tumeur était plus fréquente chez les femmes que chez les hommes, dans une proportion de 13 à 5. Cependant, il semble que le cylindrome des glandes salivaires n'ait pas plus de prédisposition pour un sexe que pour l'autre. L'âge moyen est près de la cinquantaine.

Localisation et Fréquence

Les cylindromes peuvent se développer partout où il y a du tissu glandulaire d'origine épiblastique. On les trouve surtout aux glandes salivaires majeures et mineures de la tête et du cou.

Les glandes lacrymales, la muqueuse du nez, du sinus nasal, de la trachée et des bronches sont des localisations rapportées dans la littérature (Beck et Guttman,[3] Reid,[13] Enterline et Schoenberg[7]).

L'évolution cylindromateuse peut toutefois se rencontrer dans plusieurs tumeurs épithéliales, mais Masson,[10] dans son dernier volume sur les tumeurs humaines, signale que le nom de cylindrome doit être réservé aux tumeurs qui présentent cet état dans toute leur étendue.

Des cinq cas rapportés ici, deux étaient localisés à la glande sous-maxillaire (Fig. 1), et un à la glande parotide; les deux

TABLEAU II.

Cas	*Traitement*	*Evolution*
J.U.T.	Excision de la tumeur. Evidement cervical. 5 excisions de récidives. Radiothérapie.	5 récidives. Métastases pulmonaires. Mort 8½ ans après le début du traitement.
C.H.D.	Excision de la glande. Radiothérapie. Excision de 2 ganglions. Radiothérapie.	Mort (métastase cérébrale) 5 ans après le début du traitement.
A.B.	Excision de la tumeur. Radiothérapie. Excision des récidives.	5 récidives. Mort 2½ ans après le début du traitement.
F.B.	Excision locale.	Survie de 3 ans. Absence de récidive.
L.D.	Excision locale.	Cas récent.

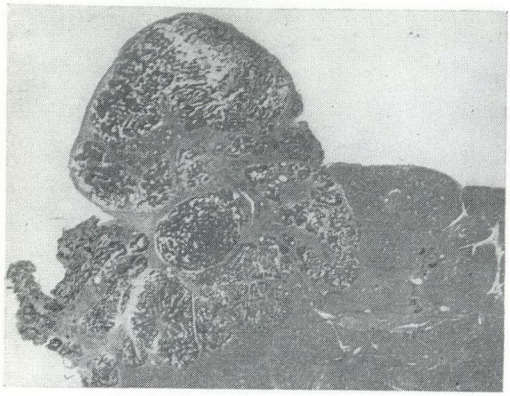


Fig. 1.

autres s'étaient développés dans le voisinage de glandes salivaires mineures labiales. Dans une série plus importante Quattlebaum rapporte des localisations à la parotide et à la sous-maxillaire dans respectivement 15 et 18% des cas.

Symptomatologie

L'apparition d'une tuméfaction, qui devient souvent douloureuse par la suite, est parfois l'indice d'une tumeur salivaire maligne plutôt que d'une tumeur mixte. D'après l'expérience des spécialistes de la Clinique Mayo, cette douleur, qu'elle soit vive ou lancinante, est plutôt propre au cylindrome qu'à la tumeur mixte.

Pathologie

Macroscopiquement, quatre des cas de cylindromes rapportés ici étaient des tumeurs bien encapsulées, nodulaires, fermes et blanc grisâtre à la coupe, tandis que le cinquième était mal délimité avec des prolongements dans les tissus voisins. L'aspect macroscopique du cylindrome n'a rien de bien caractéristique et est assez semblable à celui de la tumeur mixte ou de l'adénocarcinome (Fig. 1).

L'aspect histologique du cylindrome est caractérisé par des cordons de petites cellules rondes à noyau petit et ovoïde, entouré d'un protoplasme légèrement acidophile. Ces cordons orientés en tous sens s'anastomosent entre eux et délimitent des espaces ronds, acellulaires, de dimensions variables, tantôt vides, tantôt remplis d'une substance, hyaline ou mucoïde (Fig. 2). On rencontre des formes à stroma purement hyalin ou purement mucoïde et d'autres qui présentent les deux types de stroma. Le néoplasme infiltre souvent la capsule et les tissus du voisinage et on retrouve fréquemment des boyaux néoplasiques dans les lymphatiques périneuraux (Fig. 3). Les cellules en mitose sont très rares.

Traitement et Evolution

Dans nos cas rapportés, les tumeurs primitives ont été traitées par la chirurgie et les récidives par la chirurgie suivie de radiothérapie. Les survies de 2½, 3, 5 et 8½ ans sont rapportées. Dancot[5] signale des intervalles de 10 à 15 ans entre les les récidives.

Commentaires

Les cylindromes des glandes salivaires sont des tumeurs vraiment malignes, différentes des tumeurs mixtes et des adénocarcinomes vrais. Leur croissance est plu-

Fig. 2.

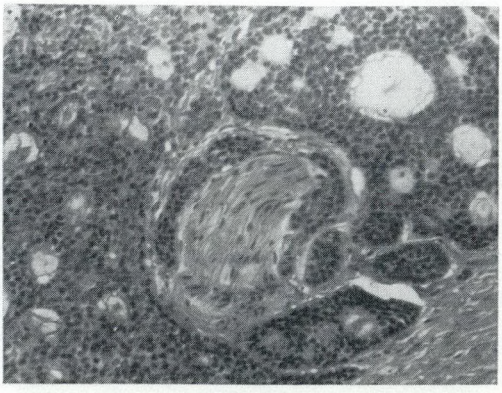


Fig. 3.

tôt lente, mais souvent douloureuse. Excisées localement, ces tumeurs se révèlent au microscope plus infiltrantes qu'on ne le croyait macroscopiquement et récidivent fréquemment. C'est pourquoi, nous croyons que le traitement du cylindrome demeure une chirurgie extensive comprenant la dissection radicale des chaînes ganglionnaires correspondantes. A la chirurgie devrait être associée la rœntgenthérapie. La question de l'irradiation est encore discutée; en effet, depuis plusieurs années on reconnaît que les cylindromes sont des tumeurs radio-résistantes. Deux de nos cas, dont les récidives ont été traitées par la chirurgie et la radiologie, ont peu ou pas répondu aux traitements par l'irradiation, D'après Rawson,[12] les récidives après radiothérapie sont de règle, bien que la réponse initiale soit favorable. Cependant, Lemaître et Baclesse[1] ont noté la radiosensibilité des cylindromes dans quelques cas. L'irradiation soit par les rayons X, soit par l'implantation d'aiguilles de radium, soit par l'or radioactif, pourrait être un traitement logique d'essai lorsque la tumeur est difficilement accessible à la chirurgie, ou encore un traitement complétant l'exérèse chirurgicale.

Résumé

Nous avons présenté cinq cas de cylindromes salivaires en tentant de démontrer leur malignité caractérisée par de fréquentes récidives et par leurs métastases à distance. Une chirurgie extensive est considérée comme le traitement idéal de ces tumeurs.

Bibliographie

1. Baclesse, F.: Radiosensibilité et métastases observées au cours des cylindromes et des tumeurs mixtes des glandes salivaires, *Bull. Assoc. franç. étude cancer.*, **29:** 260, 1940.
2. Bauer, W. H. and Fox, R. A.: Adenomyoepithelioma (cylindroma) of palatal mucous glands, *Arch. Path.*, **39:** 96, 1945.
3. Beck, J. C. and Guttman, M. R.: Basaloma or so-called cylindroma of the air passage, *Ann. Otol. Rhin. & Laryng.* **45:** 618, 1936.
4. Billroth, T.: *Virchows Arch. f. path. Anat.*, **17:** 357, 1859.
5. Dancot, H.: Les cylindromes des glandes salivaires, *Bull. Assoc. franç. étude cancer*, **40:** 383, 1953.
6. Dockerty, M. B. and Mayo, C. W.: Cylindroma (adenocarcinoma, cylindroma type), *Surg. Gynec. & Obst.*, **74:** 1033, 1942.
7. Enterline, H. T. and Schoenberg, H. W.: Carcinoma (cylindromatous type) of trachea and bronchi and bronchial adenoma, *Cancer*, **7:** 663, 1954.
8. Kirklin, J. W.: Parotid tumors, *Surg. Gynec. & Obst.*, **92:** 721, 1951.
9. Kramer, R. and Som, M. L.: Cylindroma of upper air passage, *Arch. Otolaryng.*, **29:** 356, 1939.
10. Masson, P.: Tumeurs humaines, Libraire Maloine, Paris, 2nd ed., 1956.
11. Quattlebaum, F. W., Dockerty, M. B. and Mayo, C.W.: Adenocarcinoma, cylindroma type, of the parotid gland, clinical pathologic study of 21 cases, *Surg. Gynec. & Obst.*, **82:** 342, 1946.
12. Rawson, A. J. *et al.*: Tumors of the salivary glands, clinicopathologic study of 160 cases, *Cancer*, **3:** 445, 1950.
13. Reid, J. D.: Adenoid cystic carcinoma (cylindroma) of the bronchial tree, *Cancer*, **5:** 685, 1952.
14. Wawro, N. W. and McAdams, G.: Cylindromata of major and minor salivary gland origin, *A.M.A. Arch. Surg.*, **68:** 252, 1954.

Summary

We have presented five cases of cylindromas of mixed salivary glands and have tried to demonstrate their malignant nature as characterized by their frequent recurrence and metastasis to distant viscera. Finally extensive surgical excision is considered to be the ideal treatment of cylindromas.

Cylindromas of salivary glands are true malignant tumours, differing from mixed salivary tumours and (true) adenocarcinomas. Their growth is rather slow but frequently painful. When excised locally these tumours are seen, on microscopic examination, to infiltrate much more widely than would be suspected from macroscopic examination, and they frequently recur. For this reason, it is desirable to perform extensive surgical excision together with radical dissection of the corresponding lymph nodes. The surgical treatment should be followed by radiotherapy.

The question of irradiation is a debatable one. For many years the general belief has been that cylindromas are radio-resistant tumours. Of the five cases described here, two recurrent tumours were treated by surgery and radiotherapy, but there was little response to the irradiation. According to Rawson, recurrence after radiotherapy is usual even though the initial result is favourable. However, Lemaître and Baclesse have noted radiosensitivity in some cases of cylindroma.

CARCINOMA OF THE BREAST: A REASSESSMENT*

ROBERT M. JANES, M.D., F.R.C.S., *Toronto*

FOR MORE THAN fifty years the Halsted radical mastectomy or some modification of it received acceptance as the best and the most rational treatment for cancer of the breast. It has been interesting to re-read what Halsted actually did say in his original report in January 1894 concerning 50 cases. None were followed up for more than three and a half years and many for only a few months. Despite the fact that carcinomatous granulations were excised from the wound four weeks after the original operation, Case 18 was reported as having no local or regional metastases. Case 9 had skin nodules over the right breast and right axilla, but since she had had a left radical mastectomy she was listed as having had no local recurrence. Many of the patients lived only a matter of months. Two cases were not traced and it is evident that some of the reports were by mail. Under these circumstances too much stress would not be placed today upon the figure of 6% for local recurrence. It did seem remarkable, in that the local recurrence rate reported from the various German clinics up to that time had varied from 50% to 80%. The writings of McKinnon[1-4] and of Park and Lees[5] cast doubt upon the claims for cure of the disease and must be given due credit for the revaluation of the problem that has been evident in recent years. Whether or not treatment is actually influencing the final mortality from cancer of the breast is extremely difficult to prove. Harrison *et al.*[6] have discussed the problem recently and their findings are illustrated in Fig. 1. That treatment is of value in its contribution to the mental and physical welfare of the patient has, I think, not been questioned. In this connection it is interesting to recall that Halsted said 60 years ago:[7] "The efficiency of an operation is measured truer in terms of local recurrence than of ultimate cure. In some lives are rescued only by repeated operations for local recurrence and others free from local recurrences are lost from internal metastases."

Recent demonstrations of the frequency with which cancer cells can be found in the blood are of great interest. Tables I and II are from a publication by Moore, Sandberg and Schubarg.[8] Note the frequency with which cancer cells were discovered in blood from a regional vein and from a peripheral vein in patients with breast cancer. It is true that these were advanced cases but they were not terminal. Fisher and Turnbull[9] have shown that cancer cells can be demonstrated in the inferior mesenteric vein in approximately one-third of cancers of the descending colon and rectum. Jaimet and Amy[20, 21] have demonstrated cancer cells in the bone marrow smears of a great many patients suffering from different kinds of cancer. Jaimet has stated recently,[22] "I can assure

TABLE I.—PERIPHERAL BLOOD*

	Total	*Positive*	*Atypical*
Gastrointestinal malignancies	57	25	1
Breast	59	36	5
Lung	13	6	3
Sarcoma	15	7	1
Ear, nose and throat	9	2	–
Others†	26	17	1
	179	93	11
Benign lesions	9	–	–

*Moore, Sandberg and Schubarg.[8]
†Kidney, thyroid, pancreas, malignant melanoma, testes etc.

TABLE II.—REGIONAL VEIN

	Total	*Positive*	*Atypical*
Gastrointestinal malignancies	59	31	7
Breast	17	12	1
Lung	9	7	–
Sarcoma	2	2	–
Ear, nose and throat	7	1	–
Others†	15	7	5
	109	60	13
Benign lesions	7	–	–

*Moore, Sandberg and Schubarg.[8]
†Kidney, thyroid, pancreas, malignant melanoma, testes, etc.

---

*From the Department of Surgery, University of Toronto. Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, held in Vancouver, B.C., January 1959.

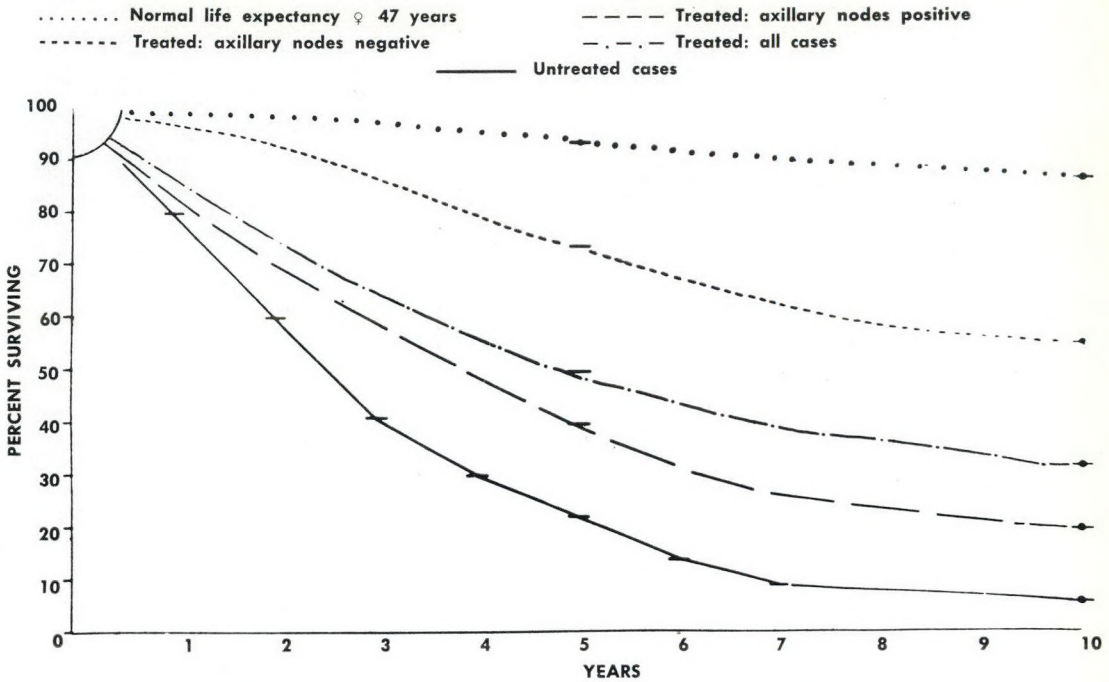


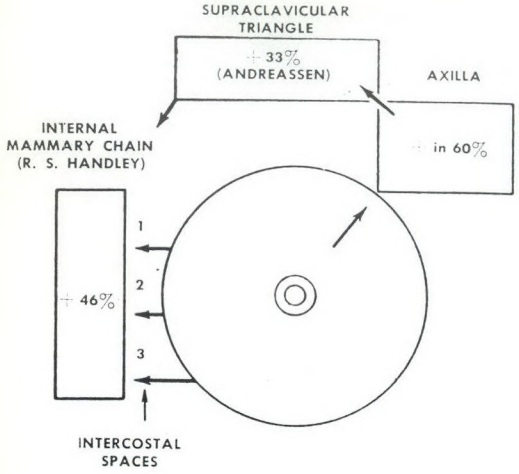
Fig. 1—Survival in cancer of the breast. (Harrison, R. C. *et al.*[6])

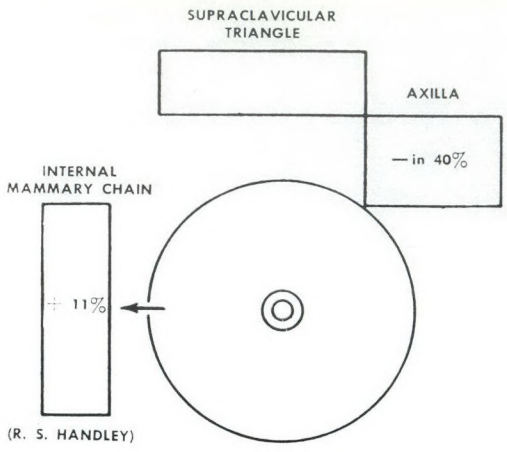
you without a doubt that we now have many hundreds of cases diagnosed as carcinoma by the bone marrow where there was either no other evidence of its presence or where it had been previously unsuspected." So far it has not been possible to relate prognosis to the finding of cells in the blood or to the number of cells found. Surely it does seem probable, however, that cells may break away frequently from most malignant tumours and that whether or not they find suitable niduses and set up metastases is dependent upon the resistance of the host. If this be true, the tendency to regard the treatment of cancer as an emergency lest cells should break away before the growth is removed is not well founded. It has been customary to say that whether or not a particular patient will be cured depends upon the kind of cancer he has; may it not be that what we really mean is the degree of immunity possessed by the patient.

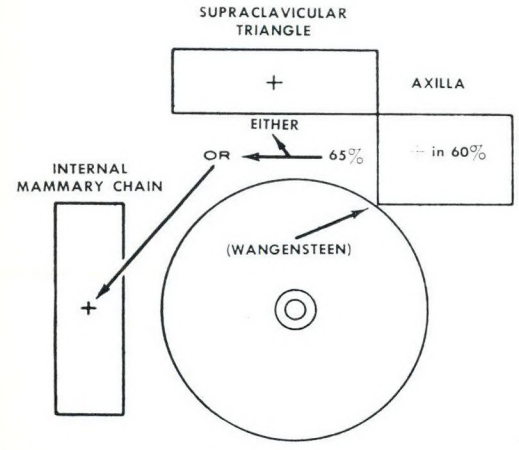
Figs. 2 and 3 are taken from an article by Ash, Peters and Delarue.[10] They illustrate well the frequency with which the various groups of lymph nodes are invaded. It must be admitted that these findings do not support the argument for radical mastectomy as the sole method of treatment for cancer of the breast.

A Toronto Survey

Some of the findings from a recent and unpublished survey of all patients with cancer of the breast admitted to the public wards of the Toronto General Hospital between 1937 and 1949 inclusive seem worth recording. Private patients and those who had received treatment whether by operation, radiotherapy or hormones before admission were excluded from the analysis. Every other patient was included no matter how advanced the disease. There were 830 cases in the study, of which only 12 (or 1.5%) were lost to follow-up. Slides were available for a review of the pathology of 91% of cases. These were assessed as to accuracy of diagnosis and graded by the late Prof. W. L. Robinson and Prof. Wm. Anderson of the department of pathology without knowledge of what had happened to the patient. The absolute five year survival rate computed on 742 cases seen between 1937 and 1947 inclusive was 32.5%. It is appreciated that this hospital receives rather more than its share of cases







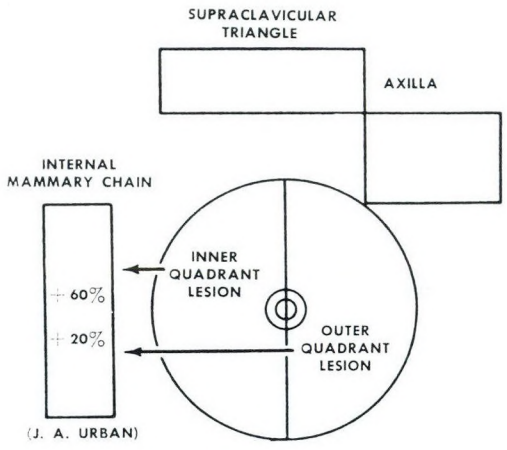


Fig. 2.—Lymph node involvement in mammary carcinoma. Cases thought suitable for radical mastectomy.

Fig. 3.—Mammary carcinoma. Further statistics regarding the frequency of internal mammary node involvement.

of advanced cancer of all types. Fig. 4 illustrates the relative proportion of stage I and stages I and II cases in the province of Saskatchewan and the Toronto General Hospital series.

Because of our interest in radiotherapy, a more detailed staging of breast cancer than that commonly employed has been used and it is necessary to present this in order that the charts may be understood (Table III). The reason for this classification will be discussed in more detail in dealing with radiation. It has come to be known as the Richards classification, and was described originally by him in the Skinner Lecture, May 1947.[11] Its relation to the Steinthal and Portmann classification is shown in Fig. 5. The Steinthal stage II was subdivided because the behaviour, so far as local recurrences were concerned, was quite different, and Steinthal stage III (Portmann stage IV) because in the presence of remote metastases it was obvious that only limited palliation could be accomplished.

Fig. 6 shows the survivals according to stage, Fig. 7 the five year survival according to duration of symptoms, and Fig. 8 the proportion of patients with positive axillary nodes by duration of symptoms. The prognosis was definitely better within the first three months and after two years. The incidence of involvement of lymph nodes was surprisingly similar throughout the first two years. It follows that there must have been almost as many

TABLE III.—Classification of Stage of Disease

Stage	*Skin*	*Tumour*	*Metastases*
I	Uninvolved	Localized, less than 3 cm.	None
II	Uninvolved	Localized, less than 6 cm.	Movable axillary nodes only.
III (Signs indicate host reaction)	Retraction phenomena only	Attachment to fascia, greater than 6 cm.	With or without axillary nodes (if present may show evidence of retraction phenomena).
IV (Signs indicate spread of cancer)	Evidences of neoplastic invasion	Fixation to chest wall	With or without axillary nodes (if present may show evidence of local neoplastic invasion).
V (Distant metastases)	As in any other stage	As in any other stage	Distant lymphatic metastases; supraclavicular nodes, contralateral axillary nodes, inguinal nodes, satellitic skin nodules beyond limits of breast. Hæmatogenous metastases.

stage I cases among those with a history of two years' duration as among those of five months or less, the only group showing a significantly greater incidence of invasion being those in the seven to nine month period. These findings would seem to support the contention that the stage of the disease is related to the degree of advancement and does not bear any direct relation to the duration of symptoms.[12] Fig. 9 shows the five year survival by grade of tumour and Figs. 10 and 11 the effect of positive or negative nodes. Kraus[13] has suggested that those patients who present themselves with a short history actually have a more actively growing lesion, which for that reason has become apparent in a shorter time. Fig. 12 from the article by Ash, Peters and Delarue illustrates this concept in a graphic fashion. Unfortunately the present survey has not provided an answer to that contention. Fig. 13 illustrates the relation between age and survival and Fig. 14 the relation between grade of tumour and age. It is apparent that on the basis of histology there is no evidence that cancer is more malignant in the younger age group and that while the 40-49 year decade shows a higher survival rate, there is little difference between the other decades. Many other interesting findings emerge from this survey but none of them is new or much different from published reports of similar analyses of cases.

The History of Treatment

That there has been great progress in the treatment of breast cancer becomes apparent from a reading of the older literature. One hundred years ago, wide excision of the tumour and partial or complete removal of the breast was practised. Volkmann[14] added removal of the contents of the axilla to removal of the breast in the 1870's. Billroth[15] in 1876 reviewed 176 of

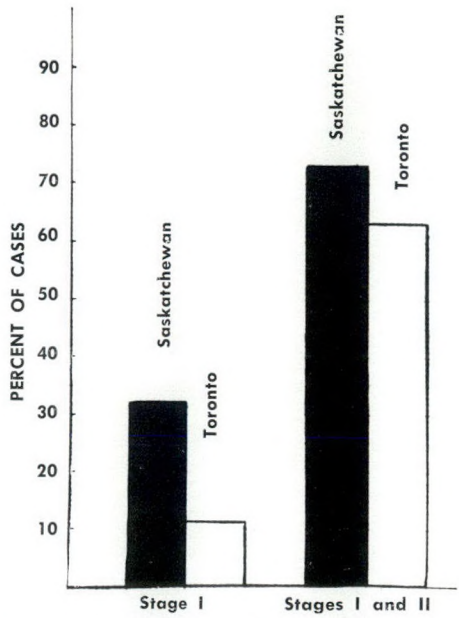


Fig. 4.—Proportion of breast cancer cases by stage in Saskatchewan and Toronto.

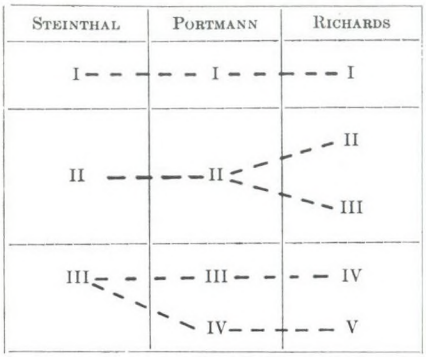


Fig. 5.—Relation of methods of staging breast cancer.

his own cases and reported an operative mortality of 20%, erysipelas being the greatest killer. Three years after treatment only 4.7% of his patients were clinically free of the disease and 82% had recurrence of the disease locally. By means of the radical mastectomy which he described in 1894, Halsted was able to reduce local recurrence to 6%. In 1927, Greenough[16] and his colleagues reported their experience in the treatment of 466 cases of breast cancer admitted to the Massachusetts General Hospital between 1894 and 1904. Their operative mortality was 36%, and only 14% of the total group were alive and well three years after operation. The reason is not easy to assess, but by 1923 an absolute five year survival rate of about 25% and by 1930 of around 35% was being reported. Today the average figures seem to have reached 40% or better. It would appear from recent reports that the results of treatment by radical mastectomy are continuing to improve. Every purely surgically treated group is of necessity a selected group, and it is probably true that there is a greater tendency to reject advanced cases than formerly. Since it is doubtful whether the operation is being done better than formerly in good clinics, it follows that the improved results must represent the influence of greater selection or earlier diagnosis. It is well to remember that this change has occured. To strive for treatment of each case at as early a period as possible seems reasonable and there is at least some evidence of its effectiveness.

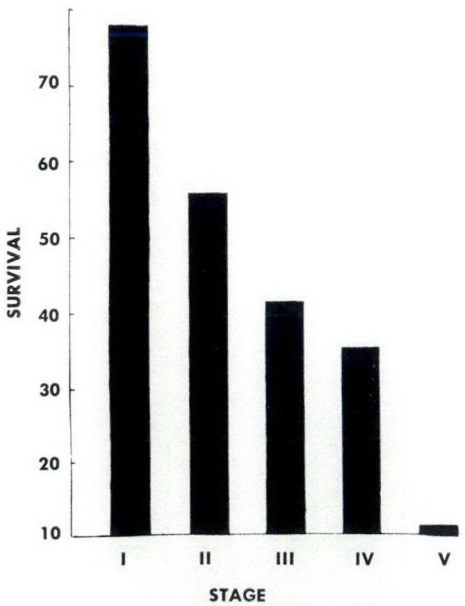


Fig. 6.—Five year survival by stage.

Reassessment of the Problem

In the light of newer knowledge our approach to the problem needs reassessment. There are several possible therapeutic approaches:

1. Continue to treat all cases in which the disease is apparently not too far advanced by the standard radical mastectomy. It is difficult to see how much better results can be expected from this approach, since the outcome in those cases in which the mediastinal and supraclavicular nodes are invaded is inevitable. The only hope lies in earlier treatment and it has been shown that nodal invasion does not vary greatly with duration of symptoms.

2. Extend the radical operation to include the internal mammary chain of nodes, as is being done by Urban in New York, Margotinni and Bercalesse of the Cancer Institute of Rome and Milan, Dahl-Iversen of Copenhagen and others.[17] It would appear that this can be done without much addition to the mortality or morbidity. Whether or not the increase in the percentage of survivors will justify this more major procedure remains to be seen.

3. Extend the radical operation to a super-radical one as advocated by Wangensteen, removing the internal mammary

chain and the supraclavicular nodes.[17] It would seem very doubtful whether this heroic approach will be justified. The long term results will have to be very good indeed before it will be widely adopted.

4. Leave the pectoralis major muscle. Richard Handley and David Patey[18] of Middlesex Hospital, London, believe that removal of the pectoralis major muscle, unless it be invaded, is not important. They have contracted the operation by dissecting the axilla without removal of the pectorals and extended it by the removal of the internal mammary nodes. There would seem to be a good deal of logic in this approach and one awaits with interest publication of the results achieved.

5. Treat by simple mastectomy followed by radiotherapy, as McWhirter[17] of Edinburgh has done since 1941. He has reported an absolute five year survival rate of 42% and an absolute 10 year survival rate of 25%. His figures are as good as those of the best surgical clinics. The intensive radiation necessary carries its own danger and complications, but such excellent results speak well for the method and throw doubt upon the value of radical mastectomy as far as prolongation of life is concerned. It must be remembered, however, that Halsted's early argument for radical mastectomy was its effect upon local recurrence and axillary involvement.

6. Continue the widely adopted routine of radical mastectomy for early cases ("early" as regards local advancement of the disease), radical mastectomy and post-operative irradiation for those with axillary involvement or more extensive local disease, and radiotherapy only for those with inoperable cancer.

There is some evidence that postoperative radiotherapy improves the survival rate

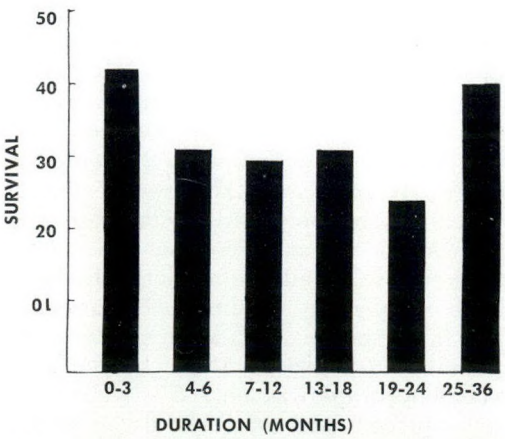


Fig. 7.—Five year survival by duration of symptoms.

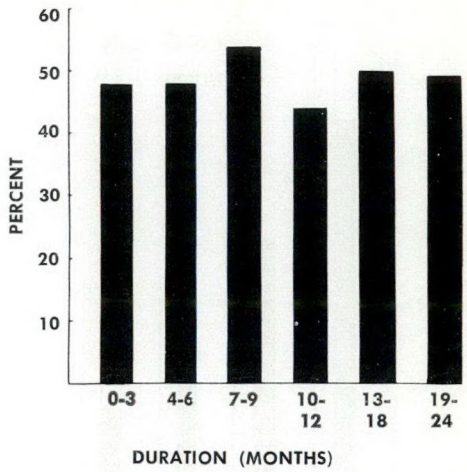


Fig. 8.—Proportion of patients with positive nodes by duration of symptoms.

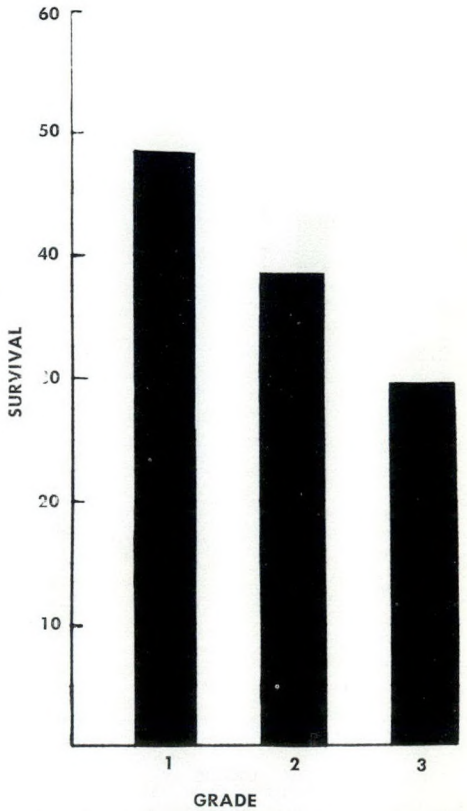


Fig. 9.—Five year survival by grade of tumour.

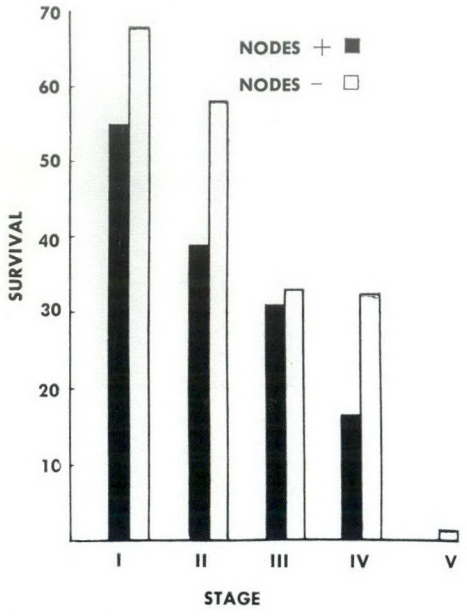


Fig. 10.—Five year survival by stage according to involvement of nodes.

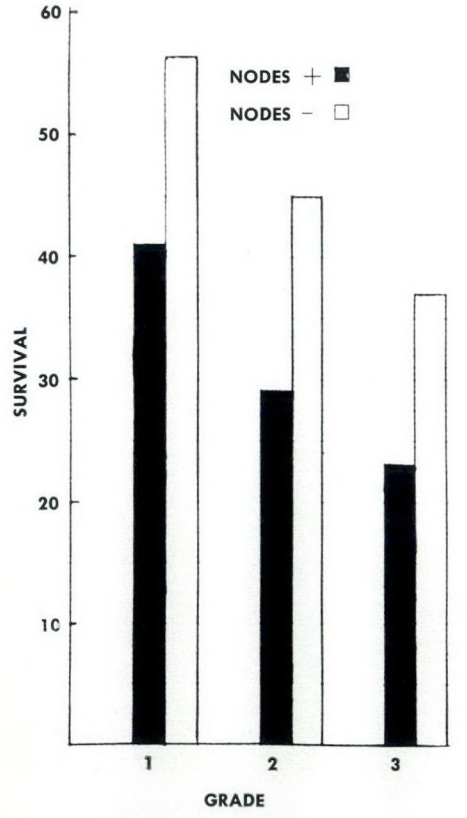


Fig. 11.—Five year survival by grade of tumour according to involvement of nodes.

in the middle group of cases but its main effect is upon local recurrences and the supraclavicular nodes. As will be shown later, it seems less effective in the control of chest wall recurrence than preoperative therapy.

7. Employ preoperative irradiation. In his Skinner lecture before the Faculty of Radiologists in May 1947, Dr. Gordon Richards[11] spoke of the place of preoperative radiotherapy in the treatment of breast cancer, and of the experience of the Toronto clinic with the method up to that time. In spite of the fact that the method has not gained general acceptance, we have continued it and the tendency is to widen rather than limit its use. Preoperative irradiation was used first in advanced cases with large primary growths or with extensive involvement of the skin and in the presence of involvement of the axillary nodes to such a degree that it seemed doubtful whether they could be removed surgically. The radiation was followed by radical mastectomy. From earlier explorations of the method it became apparent that the dissection of the axilla was not rendered unduly difficult and that, providing a sufficient period of time was allowed between the completion of radiation therapy and operation for the skin to recover, wound healing did not present a serious problem. The minimum interval required is six weeks but it may be much longer; even three to four months does not seem to alter the prognosis. It is better to wait too long than not long enough. At the beginning of the experiment the Portmann method of staging was used and stages III and IV were considered suitable for this type of therapy. An early survey showed, however, that the local recurrence rate was only 5% in these advanced cases that had received preoperative therapy as against 10% in stage II cases treated by radical mastectomy and postoperative irradiation. It was apparent that some of the stage II cases should be receiving the benefit of preoperative treatment. It was found that in the cases which were subsequently included in Richards' stage II, the local recurrence rate was only 2% whereas in those placed later in Richards' stage III, the local recurrence rate was 12.7%. For

that reason, stage II of Portmann's classification was divided into stage III (see Table III and Fig. 5), and stage III cases of this new classification were given preoperative therapy. The theoretical advantages of preoperative radiation therapy are:

1. The ability of ionizing radiation to destroy actively growing cancer cells without destroying the normal cells in the irradiated field.

2. The indirect control of tumour growth by the subsequent post-irradiation fibrosis which creates a fibrous tissue barrier through which the disease is slow to extend, chiefly because of the diminished blood supply. The perilymphatic fibrosis also retards the extension of the disease by the direct lymphatic route.

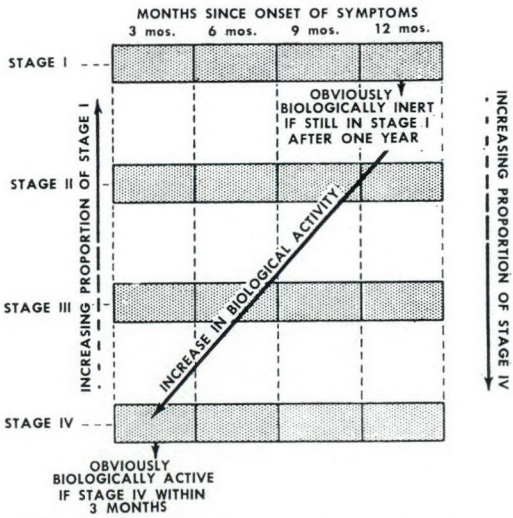


Fig. 12.—Relation of stage of disease and delay in initiating treatment.

3. The possibility of delivering a higher tumour dose to the affected area preoperatively with less risk of permanent irradiation damage to normal tissue, for the following reasons:

(a) The blood supply is intact.

(b) The presence of the breast and underlying musculature reduces the amount of radiation absorbed in the rib cage.

(c) The most heavily irradiated area is subsequently removed by the surgical procedure.

4. The possibility, although this has been questioned, that normal tissue previously irradiated is less receptive to tumour cell implantation.

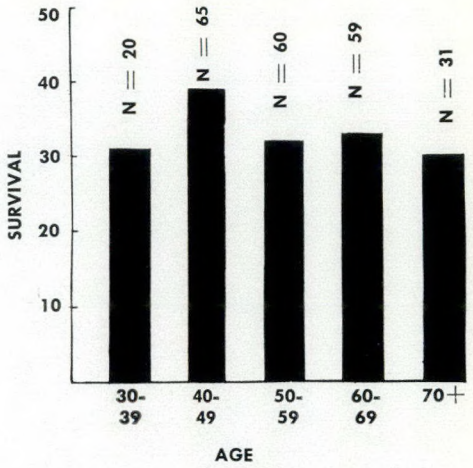


Fig. 13.—Five year survival by age.

5. The greater likelihood that spread of devitalized cells by operative trauma will be innocuous.

I am indebted to Drs. C. L. Ash and Vera Peters for the following outline of the technique which they consider essential if satisfactory results are to be obtained from preoperative therapy.

"For the past 20 years the apparatus employed for this procedure has been a 400 kV Picker unit, with a H.V.L. of 3.0 mm. Cu., 5 mA, 0.6 mm. Sn filter, F.S.D. 100 cm.

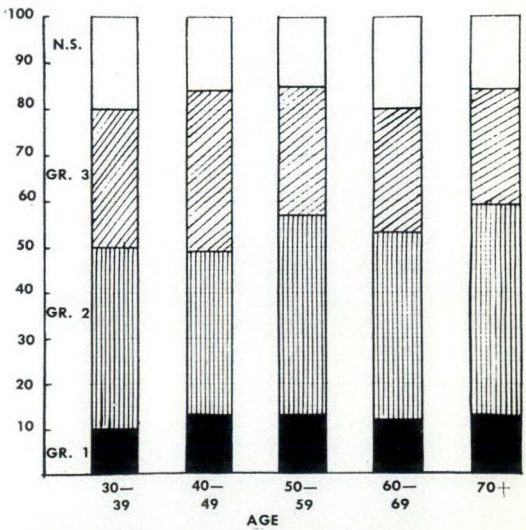


Fig. 14.—Grade of tumour by age.

Fig. 15.—Position of the arm to simplify exposure of the axilla.

The block of tissue comprising the supraclavicular and axillary regions is irradiated employing opposing 10 x 20 portals, delivering 3000 r to the entire area in two weeks. The chest wall is irradiated by opposing 10 x 20 portals, covering an area from the posterior axillary line to 2 cm. beyond the midline of sternum, in order to include the internal mammary chain of the affected side. In some cases a direct parasternal field is used. A tumour dose of 3000 r in 12 treatment days is delivered without undue reaction, a dry desquamation only being produced. When indicated, further local irradiation can be directed to the residual tumour mass of the breast giving a supplementary tumour dose of 1500 to 2000 r over a period of one week."

The obvious question that arises is—why not abandon radical mastectomy in favour of simple mastectomy as practised by McWhirter? The answer is that our results have been so relatively good in stage III and stage IV cases with radical mastectomy. The local recurrence rate is small and axillary recurrences are so rare as to constitute no problem, whereas in a limited experience with local mastectomy this did not seem to be the case. The mortality of radical mastectomy is negligible even in patients of advanced age and the disability is not as a rule great. The single troublesome problem is swelling of the arm, and this is not obviated by leaving involved nodes in the axilla or by substituting more intensive irradiation for surgery. For a good many years I have practised dissection of the axilla without removal of the pectoral muscles in frail patients of advanced years, and following the suggestion of Richard Handley I have been inclined recently to extend the use of this modified type of radical mastectomy. Only time will tell whether this more limited operation is justifiable, and it should therefore be employed with caution. If this technique is to be adopted the positioning of the arm as shown in Fig. 15 simplifies exposure of the axilla.

Handley has said: "I think, in fact, that in breast carcinoma the internal mammary chain is the principal highway of death."[17] It would seem probable that failure to control this extension of the disease accounts in part for the rather disappointing results achieved by postoperative radiotherapy. In the past the mediastinum has not received heavier radiation because of the effect upon the lung. With today's more extensive sources of radiation heavier dosage can be applied to the internal mammary chain, which has a constant anatomical location and is sufficiently near the surface to be affected by such radiation without damage to other structures. It is possible that all cases should receive radiation. Thus far it has been restricted to advanced cases and those in whom the tumour lies in the medial half of the breast. Whether surgical removal of the internal mammary chain in selected cases will give better results remains to be demonstrated. In our experience, radiation has controlled the extension to the supraclavicular nodes unless massive recurrence was present when the patient first presented for treatment, or unless a supraclavicular biopsy had been performed.

Management of Recurrences

Surgery plays a small part in dealing with local recurrences. A single recurrence on the chest wall, particularly if the patient

TABLE IV.—Response to Hormone Therapy: Distribution by Age Group Incidence

Age group	Satisfactory response	Poor response
	Œstrogen: 83 cases	
Premenopausal	0	1
Concurrent with or within 5 years after menopause	2	4
Postmenopausal (5 years after menopause and older)	48	28
Totals	50	33
	Androgen: 282 cases	
Premenopausal	38	52
Concurrent with or within 5 years after menopause	34	51
Postmenopausal (5 years after menopause and older)	61	46
Totals	133	149

has already received preoperative or postoperative radiation therapy and especially if the lesion is adherent to the chest wall or stuck firmly to a rib, is often best treated by removal, when necessary with the underlying rib. Nodules of a doubtful nature which appear upon the chest wall should always be excised for biopsy since reaction about a ligature or a nerve end or fat necrosis may readily be mistaken for tumour. Multiple reappearances in patients who have not had the benefit of radiation should receive x-ray therapy. Multiple soft tissue metastases of a widespread character may respond well to hormone therapy.

Bony metastases of a local character should be treated first by radiation. Widespread bony metastases should be treated with hormones but even here local areas which may result in disintegration of the bone, particularly if in the lumbar spine, pelvis or femora where collapse or fracture would preclude ambulation, should be irradiated while hormone therapy is being given. If symptoms suggest metastases in the spine, radiation should be advised without waiting for radiological proof. Extensive bony deposits may be present for many months before x-ray changes occur. Radiation probably prevents spinal cord damage.

Dr. Vera Peters reviewed and published in 1956[19] the experience of the department of radiotherapy of the Toronto General Hospital with hormone therapy. I shall quote extensively from her article. The findings are mainly but not entirely in agreement with those of other and similar surveys. The study included 330 patients who had received an adequate trial of either androgen or œstrogen or both, since 1940: 282 were treated with androgen, 83 with œstrogen and 35 received a trial of both hormones. The criteria for initiating hormone therapy were (1) clinical evidence of generalized metastases, (2) clinical evidence of metastases involving a single site not amenable to radiotherapy, i.e., lung or liver, (3) progression of metastases amenable to radiotherapy after the tolerance to radiotherapy had been exhausted, (4) advanced primary disease not previously treated by any recognized method, with multiple areas of involvement beyond the primary site. Tables IV and V are taken from Dr. Peters' article. It is observed that by combining the effects achieved by both hormones a worth while response was obtained in 50% of patients. Space does not permit of a more detailed consideration of this problem, but the following quotation from Dr. Peters is a fair summation. "When dealing with large numbers of patients one observes the occasional dramatic response to hormone therapy, but one's enthusiasm is dampened by a succession of poor responses. However, it is a satisfaction to the physician and a comfort to the patient, when more time tested

TABLE V.—Response to Hormone Therapy Versus Age Group with Relation to Menopause

Age group	Satisfactory palliation		Poor response		Total
	No.	Per cent	No.	Per cent	
Premenopausal	38	42	53	58	91
Concurrent with or within 5 years after menopause	36	40	55	60	91
Postmenopausal (5 years after menopause or older)	109	59	74	41	183
Totals	183	50	182	50	365

methods have failed, to be able to suggest hormone control. Ignoring completely the statistical results, the gratification from observing the comparatively few excellent results makes the treatment worth while for all who are eligible."

Prophylactic castration has not been considered justifiable in our clinic for many years. The irradiation of metastases in the bony pelvis of younger persons usually results in cessation of menstruation. As an elective procedure in itself, œstrogen deprivation by either irradiation or surgical castration is usually reserved for those patients in the premenopausal group who are showing signs of exacerbation of their disease after favourable remission with androgens. Ovariectomy may be used also in the premenopausal group as the initial method of altering hormone environment when widespread active disease becomes apparent.

When adrenalectomy was suggested as a form of therapy it was considered wise to ask one member of the surgical staff to assess the problem. Dr. Norman Delarue has performed 75 adrenalectomies. Dramatic improvement has been obtained in up to 40% of cases and incomplete objective improvement in another 20%. In 40% of cases the treatment was ineffective or the patient succumbed to the disease within one month. The best results were obtained in those with a long history of control of metastatic disease by other methods, in other words, by those who had already demonstrated a good tumour-host relationship. In the group showing dramatic improvement the period of relief averaged 12-15 months and the period of survival 18-20 months. In the presence of liver or pulmonary metastases the results were unimpressive. When resistance did break down again, the patients were definitely worse off than had the adrenals been intact.

The place of hypophysectomy in the management of disseminated breast cancer is not yet clear. At least two of the seven cases so treated in our hospital have had satisfactory remissions. It is our hope that cortisone derivatives may give results comparable to adrenalectomy and hypophysectomy, thus sparing these unfortunate patients a major surgical procedure.

Summary

Changing concepts as expressed in many recent articles upon cancer have made it necessary for clinicians to review their records with an open mind in an attempt to determine what is really worth while. It is unfortunate that the efforts of some to demonstrate that treatment is not influencing the ultimate mortality have been interpreted sometimes as meaning that no treatment is worth while. This is almost certainly not their purpose, and indeed if it were it would be necessary to contend that, since in most instances the writers are not clinicians, they are not competent to form such a judgment. It is apparent that some of our ideas must be changed. On the other hand, no one who has been permitted to watch over the past thirty-five years the changes in the course of breast cancer can have any doubt that much has been accomplished. While admitting the difficulties of the cellular pathologist and as a result accepting the possibility that a few of the lesions called cancer are really benign, the clinician is unable to accept a suggestion that because a growth remains localized for a long time it is incapable of metastasizing, since he has seen many instances in which such an apparently innocent growth has suddenly and for no apparent reason become active and been widely disseminated. He has also seen many patients in whom local growth was controlled by treatment and who remained well for years before metastases, which must of course have been present all the time, suddenly became active. This is the unknown factor of resistance of the host or immunity which clinicians have recognized for many years as a thing of great importance. It must not be forgotten that as a result of treatment vast numbers of such patients have remained free of clinical evidence of disease and well and happy even though they died finally of cancer. There is, therefore, room for discussion and for difference of opinion regarding the best form of treatment and need for a continued search for something better, but there should be no doubt that present methods are worth while or that the

individual patient should receive treatment as early as is reasonably possible.

Something needs to be said, also, regarding the value of palliation in cancer. There is a tendency for certain doctors, who of course communicate their philosophy to the laity, to assume that because a cancer cannot be cured no treatment is worth while. Nothing could be further from the truth since the lives of many, one might almost say the majority, can be prolonged for periods of months or years. The shortest period that makes treatment worth while is something that only the individual can decide. The question of how comfortable he will be and how useful to himself and others should be considered in every instance. The psychological value of treatment is another subject, but one which should not be neglected.

Conclusion

1. Changing concepts expressed in the literature have made it necessary for the clinician to review his thinking.

2. There is room for difference of opinion regarding the best form of treatment, but for none as to whether treatment is worth while.

3. Some of the findings from a recent survey at the Toronto General Hospital included in the article show the effect of age, stage, duration of the lesion and involvement of axillary nodes upon survival, and also the effect of duration of symptoms upon stage and survival.

4. While staking seems to be of prognostic value in a large series of cases, it is not considered very useful in the individual case.

5. Preoperative radiation therapy is preferable to postoperative radiation therapy for advanced cases. In the selection of cases the Richards classification is used.

6. Super-radical operations will probably not be justified by the results.

7. Hormonal control of the disease is worth while. It is not yet proved that the surgical procedures employed for altering hormone environment offer enough to justify surgical rather than medical therapy.

8. The value of palliation should not be underestimated.

Acknowledgments

The survey of cases at the Toronto General Hospital was supported by a grant from the Ontario Cancer Treatment and Research Foundation. Most of the work was done by Dr. Harold Warwick, Chief Physician to the Princess Margaret Hospital, with the able assistance of Mrs. Shirley Gordon, Chief Medical Record Librarian, Princess Margaret Hospital. Dr. A. J. Philips, statistician, the National Cancer Institute of Canada, kindly prepared the various tables from the factual material presented to him and is responsible for their accuracy. Dr. Vera Peters has given me the details regarding radiotherapy. I am indebted to Dr. Clifford Ash and Dr. Norman Delarue for advice and criticism. Finally, I desire to express my thanks to all my colleagues whose patients were included in the survey.

References

1. McKinnon, N. E.: Breast cancer mortality, Ontario 1907-1947, *Canad. J. Pub. Health*, **40:** 257, 1949.
2. McKinnon, N. E.: Cancer mortality, Ontario, 1921-1947, *Canad. J. Pub. Health*, **40:** 341, 1949.
3. McKinnon, N. E.: Cancer mortality trends under different control programs, *Canad. J. Pub. Health*, **41:** 7, 1950.
4. McKinnon, N. E.: Cancer mortality trends in different countries, *Canad. J. Pub. Health*, **41:** 230, 1950.
5. Park, W. W. and Lees, J. C.: The absolute curability of cancer of the breast, *Surg. Gynec. & Obst.*, **93:** 129, 1951.
6. Harrison, R. C. *et al.*: Breast carcinoma: some controversial aspects, *Canad. M. A. J.*, **77:** 610, 1957.
7. Halsted, W. S.: The results of operation for the cure of cancer of the breast performed at the Johns Hopkins Hospital from June 1889 to January 1894, *Johns Hopkins Hosp. Rep.*, **4:** 297, 1894; *Ann. Surg.*, **20:** 497, 1894.
8. Moore, G. E., Sandberg, A. and Schubarg, J. R.: Clinical and experimental observations of the occurrence and fate of cancer cells in the blood stream, *Ann. Surg.*, **146:** 580, 1957.
9. Fisher, E. R. and Turnbull, R. B., Jr.: The cytological demonstration and significance of tumor cells in the mesenteric venous blood in patients with colorectal cancer, *Surg. Gynec. & Obst.*, **100:** 102, 1955.
10. Ash, C. L.. Peters, M. V. and Delarue, N. C.: The argument for preoperative radiation in the treatment of breast cancer, *Surg. Gynec. & Obst.*, **96:** 509, 1955.
11. Richards, G. E.: Mammary cancer; the place of surgery and of radiotherapy in its management, *Brit. J. Radiol.*, **21:** 109, 1948.
12. Paterson, R.: Why do cancer patients delay? *Canad. M. A. J.*, **75:** 931, 1955.
13. Kraus, A. S.: A review of the effectiveness of early treatment of breast cancer, *Surg. Gynec. & Obst.*, **96:** 545, 1953.
14. Volkmann, R. von: Beiträge zur Chirurgie, anschliessend an einen Bericht über die Thätigkeit der chirurgischen Universitäts-Klinik zu Halle im Jahre 1873, Breitkopf u. Härtel, Leipzig, 1875 (referred to by Halsted).

15. Billroth, C. A. T. and Winiwarter, A. von: Beiträge zur Statistik der Carcinoma, Stuttgart, 1876.
16. Greenough, R. B., Simmons, G. E. and Barney, J. D.: The results of operation for cancer of breast at Massachusetts General Hospital from 1894 to 1904, *Surg. Gynec. & Obst.*, **39**: 1907.
17. Lewison, E. F.: Breast cancer and its diagnosis and treatment, Williams & Wilkins Company, Baltimore, Md., 1955.
18. Handley, R.: Personal communication, 1957.
19. Peters, M. V.: The influence of hormone therapy on metastatic mammary cancer, *Surg. Gynec. & Obst.*, **102**: 545, 1956.
20. Jaimet, C. H. and Amy, H. E.: Cancer diagnosis by bone marrow smears, *Ann. Int. Med.*, **44**: 617, 1956.
21. Thode, H. G., Jaimet, C. H. and Amy, H. E.: Ontario Cancer Treatment and Research Foundation Annual Report, 1957-58, p. 74.
22. Jaimet, C. H.: Personal communication, Dec. 18, 1958.

Résumé

Dans la littérature actuelle de nombreuses conceptions nouvelles ont fait leur apparition à propos du cancer du sein; il devient nécessaire de passer tout cela en revue avec impartialité et de tâcher d'en sortir ce qui est valable.

Il est malheureux de constater que les conclusions de certains, selon lesquelles le traitement n'influerait en rien sur la mortalité globale, aient pu être interprétées comme un aveu d'impuissance de la thérapeutique. Ceci n'est certainement pas dans l'esprit de ces auteurs. Pour qui a suivi l'évolution de ce problème durant ces 35 dernières années, il est évident que des progrès considérables ont été accomplis.

Il faut reconnaître les difficultés du cytopathologiste et admettre la possibilité que des lésions bénignes soient parfois étiquettées cancer. Cliniquement il faut repousser le concept de la tumeur incapable de donner des métastases parce que sa croissance s'est faite pendant longtemps localement; et l'on voit fréquemment de telles tumeurs d'apparence innocente devenir, sans raison connue, subitement actives et largement extensives. On voit également de nombreux cas chez qui une croissance localisée est contrôlée par le traitement pendant des années, et où des métastases, sans doute latentes, se réveillent et deviennent brusquement actives. Nous rencontrons là cette notion d'un facteur de résistance individuelle du sujet, ou peut-être d'immunité, qui a été reconnue depuis longtemps déjà comme très importante par les cliniciens.

Il faut se souvenir qu'un grand nombre de ces malades restent indemnes de toute manifestation pathologique pendant longtemps sous l'influence du traitement, mais qu'ils meurent cependant de cancer. C'est la raison pour laquelle la discussion doit continuer et les travaux de recherche se poursuivre; nos méthodes actuelles de traitement ne sont pas sans valeur et les malades doivent en bénéficier aussi précocement que possible.

Certains médecins ont malheureusement tendance à répandre dans le public l'idée qu'aucun traitement n'a de valeur pour le cancer. Rien n'est plus éloigné de la vérité. La valeur psychologique de la thérapeutique est enfin un facteur qu'il ne faut pas négliger.

HISTOCHEMISTRY OF CARCINOMA OF THE BREAST

The distribution of phosphatases in the breast in normal and pathological states is discussed by Fanger and Barker (*A.M.A. Arch. Path.*, 67: 293, 1959), who comment on the striking contrast between the constant presence of acid phosphatase and infrequent presence of alkaline phosphatase in carcinoma of the breast. They say:

". . . However, the occurrence of acid phosphatase and the relative rarity of alkaline phosphatase in breast cancer are not unique to this neoplasm. Acid phosphatase occurs in carcinoma of the prostate, breast, kidney, ovary, stomach and colon. The largest amount of the enzyme occurs in prostatic tumor, both benign and malignant. We have demonstrated the presence of acid phosphatase and absence of alkaline phosphatase in adenocarcinoma of the stomach, pseudomucinous cystadeno-carcinoma of the ovary, metastatic undifferentiated adenocarcinoma of the lung, fibrosarcoma of the breast, and leiomyosarcoma of the uterus. Epithelial neoplasms are noteworthy because of the absence of alkaline phosphatase in them.

"Although a number of epithelial neoplasms have a pattern of acid- and alkaline-phosphatase activity similar to breast cancer, it may have a special significance in this sex-linked organ. In the normal breast, alkaline-phosphatase activity is under the influence of estrogen, whereas acid phosphatase is not. Thus, acid phosphatase activity in the breast does not vary with age. If alkaline phosphatase in breast cancer were dependent on ovarian estrogen production, the tumors showing this activity should be in young women. Eighty per cent of our carcinoma cases showing this enzyme were in women over 50 years old. This observation tends to militate against a relationship between alkaline-phosphatase activity and estrogen in breast cancer. Specimens came from equal groups of pre- and post-menopausal patients. The concurrent study of vaginal cytology and alkaline-phosphatase activity in cases of breast cancer in post-menopausal women might furnish valuable information concerning a relationship between this enzyme and estrogen.

"Drennan was of the opinion that breast cancers which contained alkaline-phosphatase activity might respond favourably to sex-hormone therapy. We are following our cases of breast cancer in order to determine the validity of this impression."

CRUSH INJURIES TO THE CHEST

W. R. GHENT, M.D., C.M., F.R.C.S.[C],* *Kingston, Ont.*

THE PURPOSE of this paper is to discuss the problems encountered in the management of the acutely crushed chest.

In 1956, on Canadian highways, 73,594 people were injured. In the same 12-month period 3183 people were killed. Twenty-five per cent of this mortality rate was due to crushing injuries of the chest.[1] Any body system that accounts for this percentage of fatalities deserves special attention.

Crushing injuries to the chest are sustained in a variety of ways. The most frequent etiological agent in civilian practice is the "unguided missiles" which populate our highways. In contradistinction to other bodily injuries, crush injuries to the chest are usually sustained by the driver. The terrific deacceleration forces of high speed crashes are abruptly applied to the anterior or antero-lateral chest wall by the steering post. These forces can be better distributed if seat belts are worn, as has been proven conclusively by the Cornell Medical School Research Team.

The term "crush injury to the chest" has been selected as best portraying the pathological anatomy. Various other terms have been suggested. These include "flail chest", "steering-post chest", and "swinging chest". The common denominator in all of these injuries is major or minor instability of the chest wall.

A classification of these injuries into types is artificial and perhaps hazardous. As a basis for discussion I would like to present the following classification.

CLASSIFICATION

A. *Minor*
   1. Fracture of one to three ribs.
   2. Fracture of the sternum without displacement and without rib fractures.

B. *Major*
   1. Unilateral fracture of three to nine ribs.
   2. Fracture of the sternum with accompanying rib fractures.
   3. Bilateral multiple rib fractures.

This classification is concerned with the bony chest cage. To this, I would like to append the following as intrathoracic complications.

Complications

1. Pneumothorax with or without tension.
2. Hæmothorax due to
   (a) Rupture of the intercostal vessels
   (b) Laceration of lung tissue
   (c) Rupture of internal mammary vessels
   (d) Rupture of great vessels
3. Contusion of the lung with parenchymal hæmatoma and/or lung abscess formation.
4. Rupture of intercostal musculature with lung herniation
5. Cardiac contusion producing:
   (a) Myocardial infarction
   (b) Epicarditis and pericarditis
   (c) Rupture with tamponade.
6. Rupture of the diaphragm
7. Rupture of bronchus
8. Rupture of œsophagus.

In addition to complicating intrathoracic lesions, associated body injuries are common.

Associated Injuries

1. Ruptured spleen
2. Ruptured liver
3. Ruptured kidney
4. Maxillo-facial injuries
5. Head injuries.

MORBID PHYSIOLOGY

The cause of death in crush injuries to the chest is anoxia, and the mechanism producing death may be termed a lethal chain. The initial link in this chain is forged at the accident when greater or lesser portions of the chest wall are rendered unstable. The remaining links are quickly joined and the chain becomes a vicious circle, increasing in severity with each cycle until death supervenes.

During active inspiration the diaphragm descends, the chest cage enlarges and the

*From the Department of Surgery, Hotel Dieu Hospital and Queen's University, Kingston, Ont.

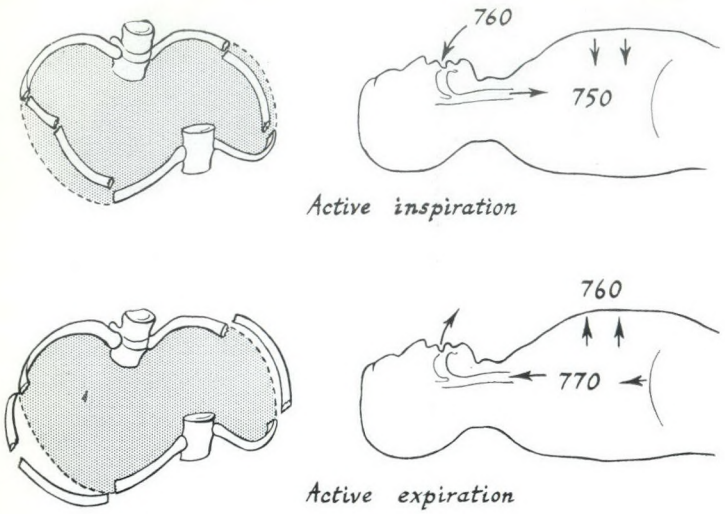


Fig. 1.—Diagram depicting the displacement of the unstable chest wall during active respiration.

intrathoracic pressure becomes sub-atmospheric. Normally this pressure gradient is resisted by the semi-rigid chest wall. However, when a portion of the chest wall is unstable, the extrathoracic or atmospheric pressure forces this area inwards as the remainder of the chest expands (Fig 1). In expiration, the reverse pressure gradients are in effect and the unstable portion is forced outwards. These are paradoxical movements. If the injury is unilateral, paradoxical respiration takes place (Fig. 2). In this situation, CO_2-laden air is forced back and forth across the carina with the addition of only minimal amounts of fresh air. The concentration of alveolar O_2 falls and of alveolar CO_2 rises. The CO_2 tension in the blood increases, and this in turn stimulates the respiratory centre. The respiratory centre directs the muscles of respiration to greater and greater excursions in a vain attempt to rid the body of CO_2. This greater effort is reflected in a greater difference between intrathoracic and extrathoracic pressures. Thus the paradoxical movements increase in magnitude, and with this increase the mediastinum may develop a pendulum movement (Fig. 3). This mediastinal flutter further embarrasses the cardio-respiratory system by causing a decrease in cardiac filling, a fall in cardiac output and consequently a fall in arterial pressure and a rise in venous pressure. Bilateral instability of the chest produces hypercarbia not by paradoxical respiration but simply by inadequate exchange.

These patients are also unable to cough because of the pain of fractured ribs and the weak expulsive efforts of the unstable chest wall. This complication increases in severity in direct proportion to the magnitude of the chest injury. Mucus and blood accumulate in the tracheo-bronchial tree and cause atelectasis and pneumonia which embarrasses respiration still further.

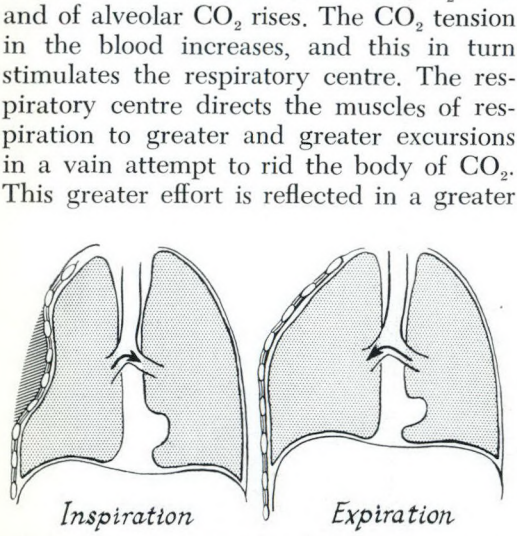


Fig. 2.—Diagram representing the mechanism of paradoxical respiration.

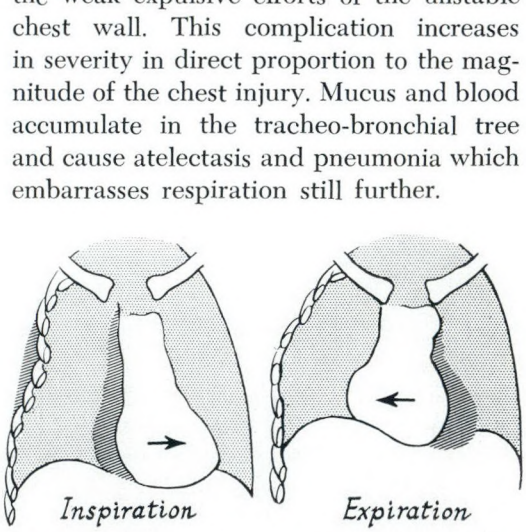


Fig. 3.—Diagram showing the development of mediastinal pendulum or flutter.

Diagnosis

The diagnosis of the major crush injuries is quite apparent. The paradoxical movements of the chest wall are marked by the time of arrival at hospital. To confirm the extent of the injuries to the chest, both bony and intrapleural, a film of the chest with the patient upright or semi-upright should be taken as soon as possible.

In the more minor forms of crushed chest, the extent of the injury may often be overlooked for some time. The nebulous term "a few fractured ribs" covers a multitude of sins of omission. In a few hours the true magnitude of the chest condition becomes apparent as the respirations rise, the pulse rate rises and the blood pressure falls. The blood pressure may show an initial rise due to the stimulation of excess CO_2. This rise is transient. Cyanosis, of course, indicates a severe degree of hypercarbia.

Treatment

The aim of treatment is the restoration of the normal physiology of respiration. To accomplish this, the following points must be realized.

Aims of Treatment

1. The correction of anoxia
2. The correction of hypercarbia
3. The re-adjustment of altered intrapleural mechanics
4. The prevention of paradoxical respiration
5. The prevention of mediastinal pendulum
6. The expulsion of retained secretions in the tracheo-bronchial tree
7. The relief of blood loss
8. The assessment and treatment of associated injuries.

The treatment of chest injuries must be individual. The steps in treatment that I will suggest are based on the previous classification, with a word of caution that if in the minor group a patient's condition deteriorates immediate re-evaluation is necessary and perhaps re-assignment to the major group.

The form of treatment to be discussed deals primarily with the bony chest cage. The complicating intrathoracic injuries are dealt with as they arise on the basis of well established methods.

A. *Minor*

1. *Fracture of one to three ribs.*

This type of injury is frequent and as a rule does not present any difficulty in treatment. The chest and ribs are x-rayed in all cases to assess the state of the intrathoracic structures. An electrocardiogram is taken if the contusive force has been applied to the cardiac region. This is repeated in five to seven days. If the chest wall is stable, these injuries are treated by intercostal blockade with 1% procaine. This will control pain, but it may be necessary to repeat this procedure in 12 to 24 hours. After this the patients are comfortable and are able to cough effectively. This is of the utmost importance in the older age group. If the chest wall in the fracture area is unstable with evidence of paradoxical movements, the intercostal blockade is augmented by applying a pressure pad over the area, held in place with adhesive tape. This will control the minor instabilities. This group of patients is always admitted to hospital and observed on a 30 to 60 minute basis for the development of signs of the "lethal chain".

2. *Fracture of the sternum without displacement and without rib fractures.*

This is a "steering post chest" and always produces a degree of instability. If the ribs and costal cartilages have not been fractured the instability is usually minor. These patients are always treated in hospital. Adhesive strapping is applied over a pressure pad on the sternum. Local 1% procaine at the fracture site may control pain but usually analgesics such as meperidine (Demerol) are indicated. In this respect, morphine is not used in treating chest injuries because of its severe respiratory and cough depressant effect. Electrocardiograms are taken at 48 hour intervals because this type of injury is frequently followed by signs of cardiac damage. The usual chest injury routine of frequent blood pressure, pulse rate and respiratory rate readings is followed. If these indicate hypercarbia, or if the sternum begins to

"swing", the injury must immediately be reclassified as major and treatment adjusted accordingly.

B. *Major*

In the past when instability of the chest wall existed, treatment was directed towards mechanical stabilization and thus indirectly to improvement of ventilation. Many ingenious methods have been employed to apply traction to the chest wall.[1] These include subpectoral traction, towel clip traction, and plaster jackets incorporating wires threaded around the ribs. The mortality rate remained depressingly high. In bilaterally crushed chests it reached 100%.

Carter and Giuseffi[2] in 1951 were among the first to suggest the advantage of tracheotomy in these injuries. This benefits the patient in four ways:

1. Dead space air is reduced from 150 c.c. to 25 or 50 c.c. This reduction allows the patient's weakened respiratory efforts to ventilate the lungs to better advantage.

2. Reduction of the paradoxical movements of the chest wall. This is accomplished by by-passing the larynx. It is at this level that the greatest amount of frictional resistance is met as the inspired and expired air passes through the slit-like aperture formed by the vocal cords. The removal of this resistance reduces the respiratory effort necessary for effective ventilation, but, of greater importance, it reduces the pressure differential between the atmospheric and intrathoracic air. This reduced pressure gradient immediately decreases the paradoxical movements of the chest wall.

3. The tracheo-bronchial tree can be suctioned at will preventing the development of atelectasis and/or pneumonia.

4. Pain is greatly decreased as soon as the tracheotomy functions. This is due to the quieter respirations and the stabilized chest wall.

Thus tracheotomy has become the basic step in the treatment of the major crushing injuries to the chest. If instability of the chest wall exists on admission to hospital the tracheotomy is performed as an emergency procedure. The patient's chest is assessed radiologically and then tracheotomy is carried out under general anæsthesia with an endotracheal tube in place. In this regard, marked improvement in the paradoxical movements of the chest wall and in the patient's general condition is noted as soon as the endotracheal tube is inserted. If this procedure is combined with assisted respiration in the emergency room, the lives of many patients with bilaterally crushed chest can be saved. In this degree of injury, by the time the patient reaches the hospital he is usually moribund. The utmost dispatch by the initial medical personnel in inserting an endotracheal tube and starting artificial respiration is necessary. After this resuscitation, the tracheotomy can be performed at a more leisurely pace and the remainder of the treatment instituted.

Fig. 4.—Diagram illustrating the application of bullet forceps to the sternum.

To return to our classification of major crush injuries:

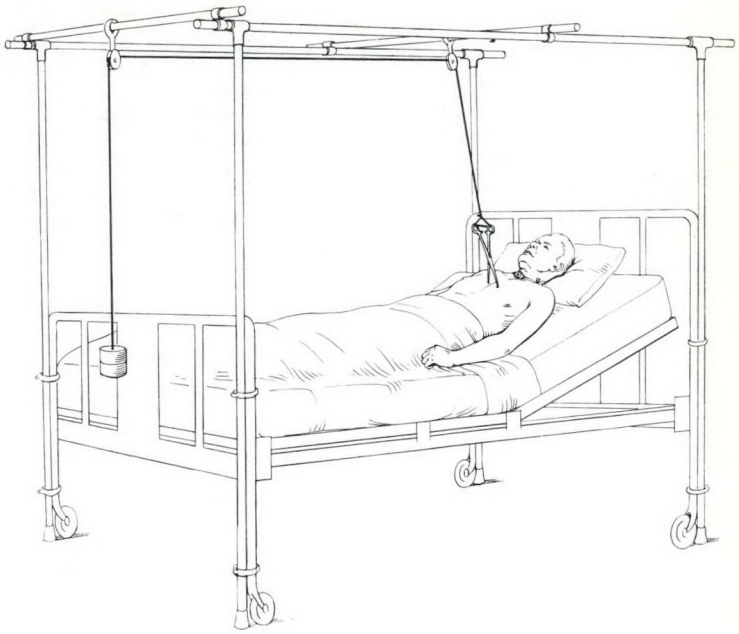


Fig. 5.—Diagram showing a patient with sternal traction applied following tracheotomy.

1. *Fracture of three to nine ribs unilaterally.*

These injuries are usually associated with an unstable chest wall. In the past, we treated this type of chest injury with intercostal blockade and pressure strapping. Tracheotomy was reserved until we found that the patient's condition was deteriorating. The immediate comfort and safety that tracheotomy provides has influenced us to carry out the operation earlier on these patients, as a semi-emergency after chest radiography and the general assessment have been completed. The care of the patient is greatly simplified by this regimen. The tracheotomy tube is removed about 14 days following injury.

2. *Fracture of the sternum with accompanying rib fractures.*

This injury is more severe and is accompanied by a high percentage of cardiac injuries. In this condition, the sternum is fractured at the angle of Louis, and the ribs on each side, unable to bend with the stress of impact, fracture between their anterior angles and the sternum. The sternum may be depressed by the force of the impact. Thus a relatively large area of the chest wall is rendered unstable, and paradoxical movements and respiratory distress are marked within a short time of the accident.

The treatment of these injuries is divided into two phases. The initial phase is carried out in the emergency room and is of the utmost importance in saving the patient's life. Under local anæsthesia, bullet forceps are applied to the sternum or towel clips to the costal cartilages (Fig. 4). Hand traction is applied to stabilize the sternum while resuscitation progresses. Chest radiographs are taken and the patient is prepared for tracheotomy. At operation the sternum is elevated if depressed and the fracture line stabilized with wire sutures. The tracheotomy is carried out and at this point one of two forms of treatment must be decided upon. The patient may be treated with sternal traction using bullet forceps and weights up to 10 lb. (Fig. 5). This form of treatment has been very successful in our hands. If the patient's condition is more precarious, an alternative method of treatment is available. The patient is joined to a fluctuating positive phase respirator, using tracheotomy. As a rule this suffices to control the sternal swing but may be combined with sternal traction. Efficiency of this respirator treatment will be detailed later.

Traction, tracheotomy and/or respirator treatment is continued until the chest wall

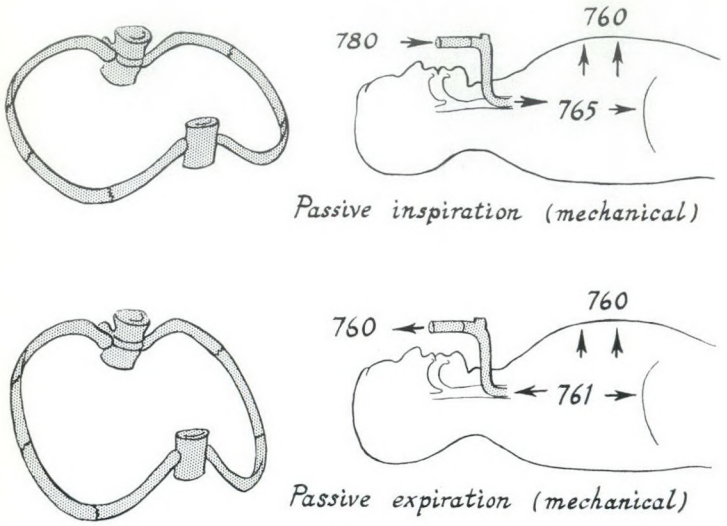


Fig. 6. — Diagrammatic representation of the pressure gradients during respirator treatment.

has become stabilized. This varies from 14 to 60 days. Serial electrocardiograms are essential during the first 14 days to detect and assess cardiac damage.

3. *Multiple bilateral rib fractures*

This is truly a "flail chest" and the integrity of the chest cage is entirely disrupted. The mortality rate, until recently, approached 100%. The first method of treatment that substantially lowered this mortality rate was suggested by Jensen in 1952.[3] The basis of his treatment was positive pressure oxygen therapy. This altered the pressure gradients and substantially reduced the paradoxical movements of the chest wall. Avery, Mörch and Benson[4] in a very astute and illuminating article described the final treatment to be applied to these injuries. They advocated the use of a fluctuating positive phase respirator designed by Mörch.

The basis of this type of treatment is a fixed volume respirator, used in conjunction with an uncuffed tracheotomy tube. The rationale of this treatment is to defunction the patient's respiratory centre by creating a respiratory alkalosis. Hyperventilation is the method used to maintain the blood pH between 7.45 and 7.5 and the rate and volume of the respirator are adjusted so that the blood pH is maintained at the desired level. This is checked twice daily during treatment.

Once the respiratory muscles have been relaxed the positive-negative pressure gradients of normal respiration are replaced by fluctuating positive pressure (Fig. 6). The fractured ribs ride passively on the pneumatic cushion of the lungs. The fractures heal and the chest cavity retains its normal volume.

The patients with severely crushed chests who reach emergency wards alive must receive immediate and vigorous therapy. The initial step, as outlined previously, is intubation and assisted respiration using an anæsthetic machine. This improves the patient's condition markedly and radiographs of the chest can be obtained. The complicating intrathoracic conditions are assessed and treatment is instituted.

A tracheotomy is then carried out, and a silver tracheotomy tube with a side arm for suction is inserted. The respirator is connected to the tracheotomy tube and the patient sent to the recovery ward. The patient is kept in this ward for some days until his condition is stabilized. The rate and depth of the respirator are adjusted until the respiratory efforts of the patient cease. The degree of alkalosis is checked twice daily by determining the blood pH.

This treatment is continued for 30 to 60 days until the chest wall has stabilized and the fractures have healed. Antibiotics are exhibited as dictated by the respiratory flora.

A word of caution must be interjected at this point. The patients who reach the

emergency room with a blood pressure at shock levels should not be transfused until it has been determined that the fall in blood pressure is due primarily to blood loss and is not in fact a manifestation of a "swinging chest wall".

SUMMARY

A review of the current treatment for acutely crushed chest is presented.

The striking decrease in the mortality rate when tracheotomy and hyperventilation therapy are used justifies their universal acceptance.

ACKNOWLEDGMENTS

I wish to thank Miss Janet Seaton, formerly Medical Artist at Queen's University, for preparing the excellent drawings for this paper.

REFERENCES

1. HUDSON, T. R., MCELVENNY, R. T. AND HEAD, J. R.: Chest wall stabilization by soft tissue traction, *J. A. M. A.*, **156**: 768, 1954.
2. CARTER, B. N. AND GIUSEFFI, J.: Tracheotomy; a useful procedure in thoracic surgery with particular reference to its employment in crushing injuries of the thorax, *J. Thoracic Surg.*, **21**: 495, 1951.
3. JENSEN, H. K.: Recovery of pulmonary function after crushing injuries of the chest, *J. Dis. Chest*, **22**: 319, 1952.
4. AVERY, E. E., MÖRCH, E. T. AND BENSON, D. W.: Critically crushed chests, *J. Thoracic Surg.*, **32**: 291, 1956.

RÉSUMÉ

Le nombre toujours croissant des accidents d'auto rend le problème des blessures par écrasement de la poitrine toujours plus important. Le conducteur de la voiture se trouve être le plus fréquemment la victime: les forces de décélération considérables qui naissent lors d'une collision, sont appliquées avec intensité sur la paroi thoracique antérieure par le volant. Dans tous ces cas, le facteur le plus grave est la non-rigidité de la paroi thoracique. De nombreuses classifications ont été proposées; l'auteur en apporte une qui catalogue ces lésions en bénignes et graves, selon le degré d'extension des fractures costales et sternales. Les complications possibles sont, par ordre de gravité: le pneumothorax, l'hémothorax, la contusion pulmonaire, la rupture de muscles intercostaux entraînant la hernie du poumon, la contusion cardiaque, la rupture du diaphragme, la déchirure d'une bronche et enfin la déchirure de l'œsophage. Bien entendu d'autres blessures associées peuvent se rencontrer.

La cause de la mort dans ces lésions par écrasement est l'anoxie. Le manque de rigidité de la paroi thoracique réduit le vide pleural et entraîne une ventilation pulmonaire insuffisante: le CO^2 s'accumule dans les bronches, puis dans le sang. Si les blessures sont unilatérales, la respiration devient paradoxale; si des mucosités ou du sang encombrent l'arbre trachéo-bronchique, la situation empire encore, puisque ces malades sont incapables de tousser.

Dans les cas graves, le diagnostic est évident. Il est plus délicat dans les formes bénignes: il se fera surtout à l'aide de la radiographie.

Le traitement consistera à rétablir une respiration physiologiquement normale. Il faudra donc lutter contre l'anoxie; corriger l'hypertension de CO^2; rétablir les mécanismes intrapleuraux; prévenir l'installation de la respiration paradoxale et le balancement du médiastin; nettoyer l'arbre bronchique des sécrétions qui l'obstruent; remplacer le sang perdu, et traiter les blessures concomitantes.

Dans les fractures d'une à trois côtes, le traitement ne présente guère de difficulté: on calmera la douleur par des infiltrations locales de novocaïne et l'on appliquera de la compression. On se comportera de façon analogue dans les fractures simples du sternum sans déplacement. Il faut arriver à faire tousser le patient; on se méfiera de la morphine à laquelle on préferera le démérol. Dans les cas graves, l'expérience montre que la trachéotomie peut être des plus utiles: elle réduit l'espace mort de 125 à 50 c.c. et permet une aspiration efficace de l'arbre bronchique; les mouvements paradoxaux disparaissent rapidement et la douleur diminue.

LA TRACHEOTOMIE DANS LES TRAUMATISMES THORACIQUES*

"La *trachéotomie*, qui a fait depuis peu son apparition dans le traitement des traumatismes graves du thorax, échappe aux inconvénients de ces deux méthodes [aspiration transnasale et bronchoscopie]. L'ouverture de la trachée améliore la respiration, car elle supprime l'obstacle à la circulation des gaz que sont le larynx souvent spasmé, le pharynx, les fosses nasales; elle diminue donc les résistances, et diminue d'une manière considérable l'*'espace mort'* cause de mauvaise oxygénation et de rétention du gaz carbonique. La canule trachéale permet une aspiration fréquente et parfaitement bien supportée par le malade, de tout le contenu trachéal; la mécanique de la respiration est à ce point améliorée par la trachéotomie que les conséquences fâcheuses du volet mobile s'atténuent. Elle ne présente aucun inconvénient pour le blessé, elle permet l'oxygénothérapie par la voie la plus directe qui soit; la pénicilline que l'on introduit par son orifice supprime la bronchopneumonie qui la faisait redouter autrefois, et stérilise un appareil broncho-pulmonaire qui n'a que trop tendance à s'infecter. Elle se présente comme un geste très efficace dans le traitement des gros traumatismes du thorax, dont elle a complètement modifié le pronostic dans ces dernières années; elle ne saurait être surestimée."

*SOURNIA, J.-C.: Les traumatismes du thorax, G. Doin et Cie, Paris, 1958, p. 42.

THE CROSS FINGER PEDICLE FLAP IN THE REPAIR OF FINGER TIP INJURIES

D. C. ROBERTSON, M.D.,* *Toronto*

TRAUMATIC loss of part of the finger tip is a common accident. A frequent cause is the industrial accident in which the finger tip is pinched off between two gears, or between a belt and a pulley. There are many non-industrial accidents also, in which part of the finger tip is lost as the result of a slip of a knife, or while trying to clear the blade of a power lawn mower, or in an accident with a car door.

Surgeons who deal with this type of injury are frequently asked to see bad results of treatment. The shrivelled finger tip, scarred and atrophied, tender and sensitive to cold, may be the result of such an injury, at first considered minor and treated by dressings only, until it has healed with a tender scar adherent to bone. Perhaps treatment has been with a split thickness skin graft applied directly to exposed bone. Often the bone has not been exposed, but a considerable portion of nail and nail bed has been lost, and healing with the resulting contracture of scar has drawn a painful and deformed nail over the tip of the finger.

The disability is very great when the result of treatment is poor. The finger tip is a sense organ as well as a working surface, and any interference with these functions reduces the ability of the unfortunate victim to earn a living. The repair of the injury must come reasonably close to the normal condition in size and shape of the finger tip, in the absence of fixed scar, and in mobility of soft tissues.

The kind of injury under discussion is the finger tip injury where part of the skin and subcutaneous tissue has been lost. Such an injury is the guillotine amputation of the tip, exposing or transecting bone, or the loss of the flexor surface pad of the terminal segment of the finger, or a dorsal defect in which much of the distal part of the nail and nail bed is gone. A defect of this kind is a problem which is all too often solved by further shortening of the terminal segment of the finger so that primary closure may be achieved. This is obviously a poor solution. The palm to finger flap has been widely used, but this requires acute flexion of the injured finger for about two and a half weeks. In the adult this may lead to permanent stiffness of the interphalangeal joints. In addition to this drawback, there may be a tender scar on the palm of the hand which is an important disability.

Fig. 1.—Cross-finger pedicle flap for loss of tip of ring finger at eight days.

The cross finger pedicle flap is an ideal solution to the problem. Further shortening is avoided. There is no stiffness of the finger which is the result of treatment. There is no scar on the flexor surface of the palm. The defect is filled with subcutaneous tissue and covered with a type of skin natural in the finger. The result is supple and mobile tissue in the defect with no tenderness or painful adherent scar.

Two operations are required to transfer this flap, and both may be done under local anæsthesia on out-patients. Most patients are back at work in four weeks, which may be less time than for a simple amputation, and is much less time than is necessary in a repair by free skin graft.

At the Toronto General Hospital we have carried out this kind of repair for these injuries for some years, and have developed

*From the Toronto General Hospital and Department of Surgery, University of Toronto.

Fig. 2.—Cross-finger pedicle flap for loss of flexor surface of terminal segment of index finger.

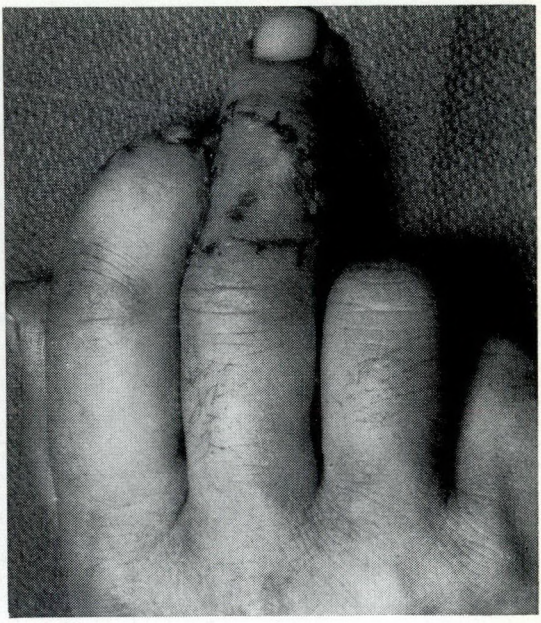


Fig. 3.—Same case as in Fig. 2 at one week showing donor site of pedicle on dorsum of middle finger repaired by split skin graft.

a fairly constant technique. The first stage of the repair is done as an emergency. The operation is begun with a thorough mechanical cleansing of the hand. We prefer 2% lidocaine (Xylocaine) without adrenaline, and inject it into the wound through a No. 27 hypodermic needle to anæsthetize the whole area. A digital nerve block is unnecessary. The required flap is then outlined in ink on the dorsum of the adjacent donor finger, care being taken to provide a wide base. It is wise also to arrange the

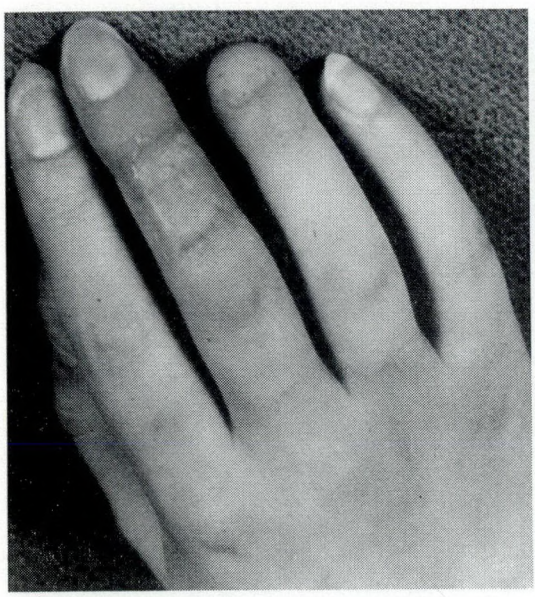


Fig. 4.—Completed cross-finger pedicle flap three weeks after completion of second stage. This defect involved the tip and dorsum of the ring finger.

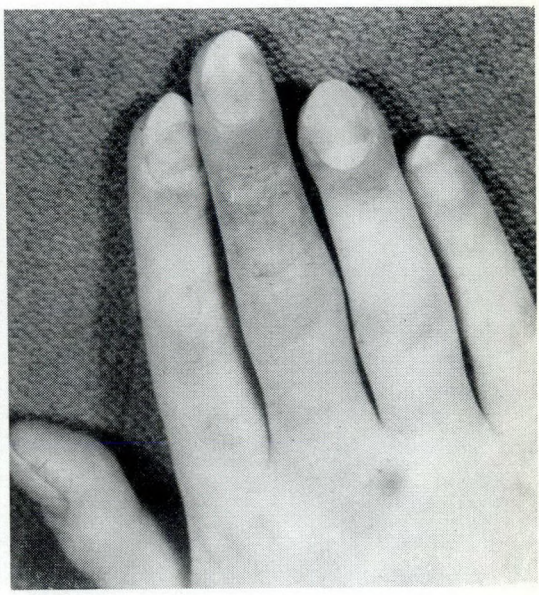


Fig. 5.—Same case as in Fig. 4, showing appearance when a false nail is used. It is fixed in place with dermatome glue.

flap so that it is possible to lengthen it by extending the incision if it should become necessary later on.

This area on the donor finger is then anæsthetized with lidocaine and the flap is raised. Care must be taken to dissect it through the proper plane. A layer of areolar tissue must remain over the extensor tendon, as bare tendon is a poor bed for the split graft which must be used to cover it. All bleeding from the donor site of the flap is controlled, and the flap is applied to the defect in the injured finger to be sure that it fits. It is at this point that the flap may be found to be too short and may require to be lengthened.

When a good fit is assured, attention is turned to covering the donor site of the flap. It has been very satisfactory in our experience to obtain the split thickness skin graft from the upper arm on the injured side. This site has been found to be most comfortable when dressed. The graft can be obtained with a Padget dermatome blade. For those who do not wish to cut the skin graft free hand, the Silver* dermatome is ideal. This little instrument provides a guarded razor blade for cutting, and the thickness of the graft can be adjusted. The donor site of the split graft is dressed with 1% scarlet red ointment and the dressing is glued to the skin with Mastisol or dermatome glue. This prevents the slipping of the dressing which can be so uncomfortable on donor areas.

The split graft is sutured carefully to the edges of the area from which the flap was raised. A small pledget of absorbent cotton is pressed down over the graft and held in place with a few turns of one inch gauze bandage. This bandage must not restrict the circulation either of the pedicle flap or the finger as a whole. It remains now to suture the flap to the defect in the injured finger.

The two fingers are dressed together. Absorbent cotton is placed between them to prevent maceration of the skin and then both fingers are enclosed in Tubegauze.

At the end of eight days the dressing is taken off completely and the sutures are removed from the edge of the split graft only. The dressing is then reapplied in the same way. Thereafter the dressings are changed every few days as may be required until about the 18th day, when the second stage operation is performed.

There are many who feel that the second stage can be performed as soon as the 10th day. However, it has been our impression that the flap is much healthier and the final healing is more rapidly achieved when the interval is more than two weeks. By this time the attachment of the pedicle is firm and œdema in the pedicle has subsided.

The second stage operation is a simple one. Under local anæsthesia, infiltrated directly into the base of the pedicle so as to anæsthetize both the dorsum of the donor finger and the flap, the pedicle is divided. It is always wise, before the actual division of the pedicle, to make certain that the incision is to be in the right place. The unwitting division of the pedicle leaving too small a graft is a blunder which will spoil the result.

The recipient finger is now infiltrated through the wound at the cut edge of the flap. This wound is trimmed so that closure by primary suture can be done. The wound on the dorsum of the donor finger is dealt with in the same way.

In about 48 hours the dressings are removed and all sutures are taken out. After this, only a simple dressing is required for another week.

As time passes the split graft on the dorsum of the donor finger becomes more and more normal in appearance, and in a few months it is very hard to see anything unusual at all. The flap itself also improves, and may become quite invisible in those cases in which the injury did not produce a deformity such as shortening which would draw attention to the finger.

Summary

Traumatic loss of soft tissue of finger tips, provided that such an injury is not too extensive, is very well repaired by means of a cross finger pedicle flap. This two stage operation has been described. It can be done as an out-patient procedure. It does not require complete immobilization of the fingers and so does not result in

*J. F. Hartz Co., Limited, Toronto.

stiffness. It will produce a good result and does not incapacitate the patient for a long period of time.

References

1. Gurdin, M. and Pangman, W. J.: Repair of surface defects of fingers by trans-digital flaps, *Plast. & Reconstruct. Surg.*, **5:** 368, 1950.
2. Horn, J. S.: The use of full thickness hand skin flaps in the reconstruction of injured fingers, *Plast. & Reconstruct. Surg.*, **7:** 463, 1951.
3. Curtis, R. M.: Cross-finger pedicle flap in surgery, *Ann. Surg.*, **145:** 650, 1957.

Résumé

La perte du bout du doigt est un accident fréquent, survenant par exemple à l'usine, lorsqu'un doigt est pincé dans un engrenage. Les résultats que l'on observe après ce genre de blessures sont souvent mauvais: le bout du doigt est atrophié, recouvert d'une cicatrice adhérente à l'os et sensible au froid. Ceci vient lorsque ces lésions, considérées comme peu graves, ont été simplement traitées par un pansement.

En réalité, le traitement de choix est la greffe pédiculée croisée interdigitale. On évite ainsi le raccourcissement du doigt, son atrophie et son ankylose. La plupart des patients reprennent leur travail au bout de quatre semaines.

Deux opérations sont nécessaires: dans un premier temps, après avoir soigneusement nettoyé la main et anesthésié localement les doigts intéréssés, on lève un lambeau de taille convenable: cette dissection doit être faite avec soin, car il faut laisser du tissu conjonctif au-dessus de la gaine du tendon extenseur du doigt donneur. L'hémostase doit être précise. Le lambeau est suturé en place et le lieu de prélèvement est recouvert par un greffon de peau libre prise au niveau du bras. Les deux doigts sont pansés ensemble.

Après deux semaines le second temps est effectué, qui consiste à séparer la base du lambeau et à faire les sutures nécessaires.

Les résultats sont excellents, au point que toute trace d'opération disparaît.

LES AMPUTATIONS DES DOIGTS*

"Les lésions de la main dans la chirurgie du travail tiennent une place importante, puisque les statistiques que P. R. Bize vient de publier montrent que, sur 250 accidents dans les usines de métallurgie, il y a 73,2% d'accidents portant sur les mains et les doigts. M. Iselin a déjà longuement insisté sur la complexité de cette question, mais il nous a semblé utile de préciser les indications thérapeutiques. Ces traumatismes se terminent fréquemment par des amputations ou des désarticulations de un ou de plusieurs doigts. Or, il est étonnant de noter combien sont différentes les règles de conduite, suivant les auteurs, en ce qui concerne les niveaux d'amputation. De nombreux facteurs sont à considérer chez ces blessés et forment un tout où la technique de l'intervention, le choix du niveau d'amputation et les soins postopératoires sont indissociables.

"Le niveau des amputations et désarticulations de doigts dépend de facteurs anatomiques, physiologiques et opératoires (valeur des parties molles, taille des lambeaux). . . .

"Dans l'étude de la physiologie de la main du travailleur, Ch. Rémy a, dès 1903, classé les différents modes de préhension de la main armée d'un outil. La préhension à pleine main est réalisée par les doigts s'enroulant sur l'outil, créant un fourreau fermé par l'anneau pouce-index, mais la longueur suffisante de ce fourreau est assurée par le cinquième doigt: c'est souligner ainsi la valeur du doigt auriculaire; la disparition d'un des doigts intermédiaires dans cette préhension n'a qu'une faible importance. Le deuxième mode de préhension est la pince digitale pour les outils légers et fins: l'instrument est tenu entre le pouce et l'index. Cette fonction repose sur une longueur suffisante du pouce et l'existence d'une commissure large, libre et profonde entre le pouce et les autres doigts. Il existe une pince multiple entre le pouce et plusieurs doigts réunis. La troisième fonction est celle du crochet: ce qui importe pour elle, c'est la longueur des quatres derniers doigts; le crochet disparaît avec la perte des deux dernières phalanges.

"Ainsi, un certain antagonisme se manifeste dans les indications opératoires, selon qu'on envisage de faire passer la conservation de telle ou telle fonction avant les autres. Lorsqu'on compare les facteurs qui commandent la conservation de ces fonctions, on voit qu'il faut garder un aussi bon anneau pollici-digital que possible avec enroulement du 5e doigt sur l'éminence hypothénar, une mobilité et une longueur aussi grande que possible du pouce avec commissure libre entre le pouce et le doigt opposé."

*Desfosses, L.-N. et Brodowsky, R.: Amputations et désarticulations des doigts chez les accidentés du travail. Notes de techniques chirurgicales de la Presse Médicale, recueillies par Lucien Leger. Masson et Cie, Paris, 1958.

SCALENE NODE BIOPSY – A POST-MORTEM STUDY

ALLAN R. DOWNS, M.D.* and LAWRENCE S. McMORRIS, M.D.,† *Winnipeg, Man.*

SINCE DANIELS'S[7] report in 1949, scalene node biopsy has been used as an aid in the diagnosis of intrathoracic disease and as one of the methods of assessing operability in cancer of the lung.[5, 8, 10]

There is considerable variation in the incidence of reported positive node biopsies. Schwippert[14] reports 8% and Connar[5] 31% positive biopsies in carcinoma of the lung. Scott[15] found 87% positive in sarcoidosis. However, in his series of 160 cases of all types of intrathoracic disease, the diagnosis was confirmed by scalene biopsy in only 20%. This discrepancy has been attributed to the nature of the disease and the adequacy of the biopsy.

In the literature reviewed,[1-18] no one, with the exception of Connar, defines an adequate biopsy. He stresses the importance of finding anthracotic pigment in the nodes. Baer[1] reports that 16% of his biopsies did not contain lymph nodes, but no other author comments on the presence or absence of lymphoid tissue in negative biopsies. Consequently, the present study was undertaken in an attempt to determine the average lymph node content of the scalene fat pad and to assess the significance of anthracotic pigment.

METHOD AND TECHNIQUE

Bilateral scalene node dissections were performed by one of us (A.R.D.) at 50 non-consecutive autopsies in which supraclavicular nodes were not palpable. These cases were otherwise unselected. Each of the 50 cases thus provided two specimens, yielding a total of 100 scalene node biopsies.

With the exception of the routine autopsy incision, the technique used was similar to that described by Daniels and given in more detail by Connar.[5] At a point 1 cm. above the clavicle, the lateral border of the sternomastoid muscle was sectioned transversely for a distance of 1.5 cm. and retracted medially. The omohyoid fascia was removed and the underlying fat pad dissected free. The dissection began at the lower border of the posterior belly of the omohyoid muscle and was carried laterally to the external jugular vein, medially to the internal jugular vein and inferiorly to the subclavian vein. The inferior and medial extension of the fat pad as depicted in Fig. 1 was also removed. In performing the dissection, the transverse cervical and supravascular veins were transected where they passed through the fat pad. The corresponding arteries were not transected, although this may be done surgically with impunity. The phrenic nerve was visualized deep to the prevertebral fascia lying on the scalenus anticus muscle as shown in Fig. 1. The thyro-cervical trunk was also demonstrated as shown. On the left side, the thoracic duct was demonstrated at the junction of the subclavian and internal jugular veins.

Lymph nodes measuring 0.2 cm. or more in diameter which were visible to the naked eye, were removed from the fat pad, fixed in formalin and embedded in a single paraffin block. All of the remaining fatty tissue was similarly treated. This resulted in two blocks from each scalene fat pad. Single microscopic sections of each block were stained with hæmatoxylin and eosin. The number of lymph nodes seen in each section was recorded. In those sections in which no anthracotic pigment was seen initially, multiple deeper sections were examined.

RESULTS

TABLE I.—MACROSCOPIC LYMPH NODES IN SCALENE FAT PADS

	Right	*Left*
Number of biopsies with no nodes (gross or microscopic)	1	2
Number with only 1 node over 0.2 cm.	3	4
Number with 2 - 6 nodes over 0.2 cm.	39	41
Number with more than 6 nodes over 0.2 cm.	7	3
Total	100	

*Fellow in the Department of Pathology, Winnipeg General Hospital, Graduate Student, Department of Surgery, University of Manitoba.

†Fellow in the Department of Pathology, Winnipeg General Hospital.

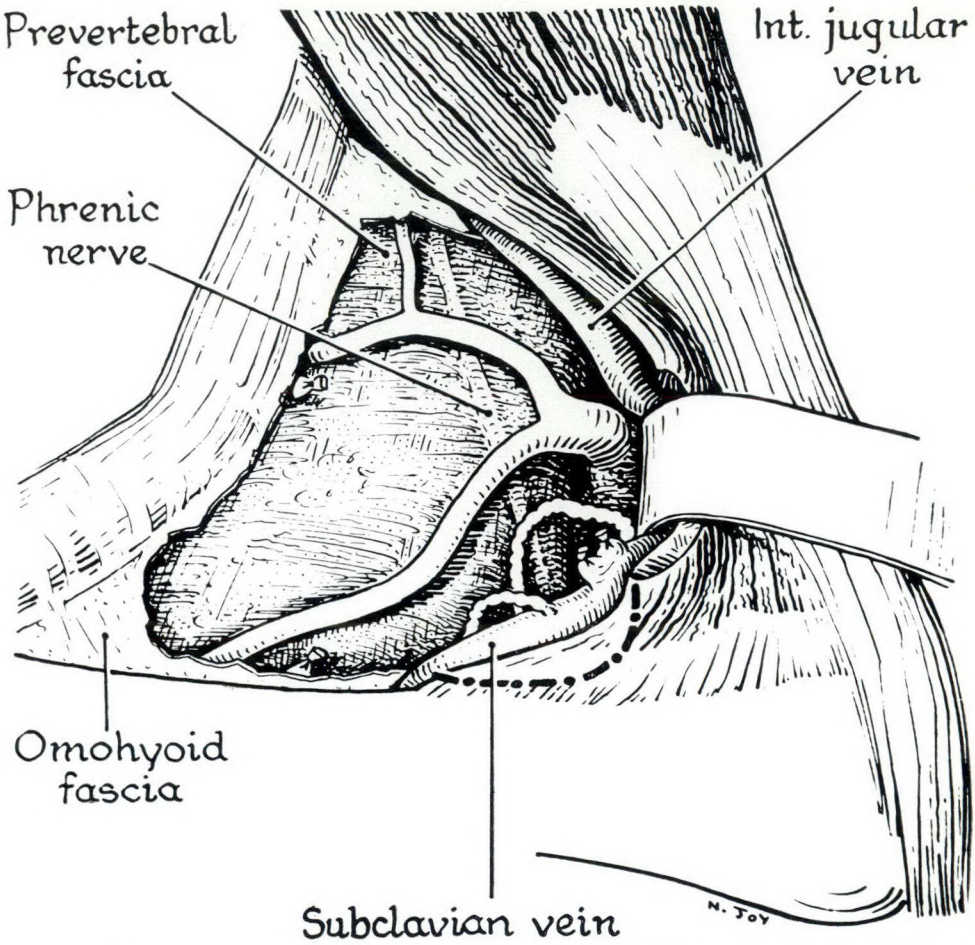


Fig. 1.—Anatomy of right scalene fat pad region. The inferior and medial boundaries of the fat pad are shown by the dotted line deep to the lateral and inferior portion of the right sternomastoid muscle.

The numbers of lymph nodes visible to the naked eye are recorded in Table I. Many specimens contained additional microscopic nodes. The total number of nodes (gross and microscopic) found per biopsy specimen varied from one to 23, with an average number of five nodes. The age of the patients in this series ranged from 22 to 92 years, the majority being over 40 years. The number of nodes found in those under 40 years old did not appear to differ from those in the age group over 40. Anthracotic pigment was found microscopically in 43 of the 100 biopsy specimens.

Discussion

It is seen from the table that only three out of 100 biopsies did not contain nodes either grossly or microscopically. This indicates that when a careful scalene fat pad dissection is performed, almost all specimens will contain lymph nodes. This finding is in disagreement with that reported by Bennett and Carr[3] in a similar post-mortem study, for they found 53 of 148 biopsy specimens without nodes.

Mention has been made by Connar[5] of the necessity of finding microscopic anthracotic pigment as an indication of the adequacy of scalene node biopsy. Our study does not bear this out, as slightly less than half of the scalene fat pads showed nodes with demonstrable pigment in the microscopic sections. Because of this, the presence of pigment would not appear to be a reasonable criterion for adequacy of scalene fat pad resection. On the other hand, if

anthracotic pigment is invariably present in nodes draining the mediastinum, its absence may indicate that the nodes do not drain this area. Thus, some scalene nodes may remain free of disease in cases with extensive mediastinal involvement in which spread to the scalene nodes would be expected to occur.

If it could be shown that scalene nodes do not invariably drain the mediastinum, this would strengthen the case for extending the biopsy to include paratracheal and upper mediastinal nodes as has been advocated by Harken.[10]

Summary

The technique of scalene biopsy is reviewed. Bilateral scalene node biopsy was performed at 50 autopsies. Macroscopic lymph nodes were present in 97% of the scalene fat pads. The significance of anthracotic pigment in scalene nodes is discussed.

Acknowledgments

We are indebted to Miss Nancy Joy of the Department of Surgery, University of Manitoba, for the diagram, and to the Staff of the Pathology Department of the Winnipeg General Hospital for technical assistance and helpful criticism.

References

1. Baer, L. S.: Diagnostic value of biopsy of nonpalpable retroclavicular paratracheal lymph nodes, *Stanford M. Bull.*, **10:** 104, 1952.
2. Beahrs, O. H. *et al.*: The value of biopsy of lower deep jugular (scalene) nodes as a diagnostic procedure, *Minnesota Med.*, **40:** 152, 1957.
3. Bennett, W. A. and Carr, D. T.: Scalene lymphadenopathy; a post-mortem study, *Am. Rev. Tuberc.*, **76:** 503, 1957.
4. Carstensen, B., Norviit, L. and Odelberg, A.: Experiences with retroclavian lymph node biopsy in diagnosis of certain intrathoracic diseases, *Dis. Chest*, **25:** 443, 1954.
5. Connar, R. G.: Prescalene and deep cervical lymph node biopsy, *Surg. Gynec. & Obst.*, **101:** 733, 1955.
6. Cuykendall, J. H.: Use of prescalene lymph node biopsy in absence of palpable supraclavicular nodes: report of 41 cases. *J. A. M. A.*, **155:** 741, 1954.
7. Daniels, A. C.: A method of biopsy useful in diagnosing certain intrathoracic diseases, *Dis. Chest*, **16:** 360, 1949.
8. Delarue, N. C.: An evaluation of scalene node biopsy, *Canad. J. Surg.*, **1:** 94, 1957.
9. England, N. J.: Scalene node biopsy in the diagnosis of intrathoracic disease, *Canad. M. A. J.*, **75:** 268, 1956.
10. Harken, D. E. *et al.*: Simple cervicomediastinal exploration for tissue diagnosis of intrathoracic disease with comments on recognition of inoperable carcinoma of lung, *New England J. Med.*, **251:** 1041, 1954.
11. Johnson, J. E., Jr. and MacCurdy, J. M.: Pulmonary histoplasmosis diagnosed by scalene node biopsy, *Am. Rev. Tuberc.*, **66:** 497, 1952.
12. Josephs, B. N. and Woods, F. M.: Prescalene lymph node biopsy, *A.M.A. Arch. Surg.*, **76:** 93, 1958.
13. Rouvière, H.: Anatomie des lymphatiques de l'homme, Masson et Cie, Paris, 3rd ed., 1932.
14. Schwippert, H. and Macmanus, J. E.: A histologic study of routine scalene node biopsies, *Surgery*, **42:** 533, 1957.
15. Scott, S. M.: A critical review of 160 consecutive scalene node biopsies, *Am. Rev. Tuberc.*, **76:** 1002, 1957.
16. Shefts, L. M., Terrill, A. A. and Swindell, H.: Scalene node biopsy, *Am. Rev. Tuberc.*, **68:** 505, 1953.
17. Storey, C. F. and Reynolds, B. M.: Biopsy techniques in diagnosis of intrathoracic lesions including lung biopsy, mediastinal biopsy and resection of deep cervical fat pad and its contained nodes; report of 12 illustrative cases, *Dis. Chest.*, **23:** 357, 1953.
18. Weiss, H. A. *et al.*: Sarcoidosis diagnosed by means of subclavian lymph node biopsy; report of case, *J. A. M. A.*, **146:** 1221, 1951.

Résumé

Depuis le rapport de Daniels en 1949, la biopsie des ganglions scaléniques a été utilisée comme un moyen de diagnostic dans les maladies intrathoraciques et le cancer du poumon. La littérature publiée à ce sujet comporte de nombreuses contradictions; cette étude a été entreprise dans le but de déterminer l'importance du contenu de la graisse scalénique en tissu lymphoïde.

Une technique de prélèvement est décrite avec précision. Les pièces obtenues sont traitées par les méthodes histologiques courantes et colorées à l'hématoxyline-éosine.

Seulement 3% des biopsies ne contenaient pas de ganglions, ce qui permet de conclure que, en pratique, on trouvera du tissu lymphoïde dans tous les cas. La signification de la présence ou de l'absence de pigment anthracosique dans ces ganglions est discutée.

SIMPLE ULCER OF THE COLON: REPORT OF FOUR CASES*

MORRIS H. W. FRIEDMAN, M.D., M.Sc. and
WALTER C. MacKENZIE, M.D., M.S.(Surg.), F.A.C.S., F.R.C.S.[C], *Edmonton, Alta.*

SIMPLE ULCER OF THE COLON is a non-specific ulceration of the large bowel. The purpose of this report is to review the subject briefly, and to present four cases of simple ulcer of the colon treated at the University of Alberta Hospital.

HISTORY

Simple ulcer of the colon was first described by Cruveilhier[1] in 1830. Morel and Scheyron[2] in 1895 confirmed the presence of a non-specific ulcer by histological study. In 1902, Quenu and Duval[3] were the first to review the subject. Many of the early case studies were incompletely documented and it is probable that some of them were not true examples of simple ulcer. In a comprehensive review of 53 cases in 1927, Barron[4] considered simple ulcer of the colon to be a distinct and definite pathological lesion. In 1937, Sir David Wilkie[5] described several complications of simple ulcer of the cæcum. Sporadic reports have appeared in recent years, emphasizing the importance of this lesion.

INCIDENCE

About one hundred cases of simple ulcer of the colon have been reported in the literature but it is probably more common than this figure would suggest. Barlow[6] states that in any large hospital, some three cases occur annually. Simple ulcer is twice as common in men as in women and the average age incidence is about 42 years.[4]

Similar ulceration may occur anywhere in the gastrointestinal tract, from the œsophagus to the rectum. About 50% of all simple ulcers of the large bowel have occurred in the cæcum[7] (Fig. 1). The sigmoid colon has been the next commonest site with an incidence of 16%. About 9% were found in the ascending colon, and about 5% developed in the hepatic flexure, transverse colon, splenic flexure and descending colon. Another 5% occurred in the rectum, where differentiation from a stercoral or stasis ulcer must depend on a clinical history, since the histological findings are similar.

ETIOLOGY

The etiology of simple ulcer is unknown. These ulcers are not caused by the action of any known specific organisms, as are the ulcers of tuberculosis, syphilis, dysentery, or typhoid fever. The disease process of ulcerative colitis does not cause simple ulcer.

Numerous theories have been proposed to explain the etiology of simple ulcer.[4] These include thrombosis in local arteries or veins, intestinal stasis and infection, bacterial toxins and trauma of a mechanical nature. No theory appears entirely satisfactory.

Some authors,[8, 9] have considered simple ulcer of the cæcum a separate entity. In the cæcum, the ulceration often occurs near the ileo-cæcal valve and, in attempting to explain this lesion, an interesting analogy has been drawn comparing cæcal ulcer with peptic ulcer of the duodenum. Cameron[10] emphasized that both ulcers occur distal to a sphincter, the blood supply is relatively deficient and there is histological similarity in the non-specific inflammatory reaction. This analogy is not complete because the cæcum is not bathed by acid peptic juice.

DIAGNOSIS

Simple ulcer of the colon has very rarely been diagnosed before operation or autopsy.[11] The clinical features are variable, depending on the site of the ulcer, acuteness or chronicity of the lesion, and on the presenting complication in the individual patient.

Pathological diagnosis depends upon the exclusion of known causes of ulcer of the bowel and on the gross and microscopic findings of a non-specific ulcer. Simple

---

*From the Department of Surgery, University of of Alberta and Surgical Service, University of Alberta Hospital. Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, January 23-24, 1959, in Vancouver, B.C.

Fig. 1.—Incidence of simple ulcer of the large bowel (modified from Barlow[7]).

ulcers are usually single, and vary from a few millimetres to several centimetres in diameter. They are often circular in shape but may be oval, linear or annular. The ulceration may be superficial in the mucosa or a funnelling perforation throughout all layers of the bowel wall. Histologically, there is an acute, subacute or chronic non-specific inflammatory reaction.

An acute simple ulcer may resolve and heal. The frequency of this occurrence is difficult to assess, as a healing ulcer may be readily overlooked at operation.

The *complications* of simple ulcer usually account for the presenting complaints (Fig. 2). Perforation, often in a cæcal ulcer, is the commonest and most dangerous complication. More than 50% of patients showed evidence of perforation when they were first seen.[6] An acute perforation resulting in generalized peritonitis has been

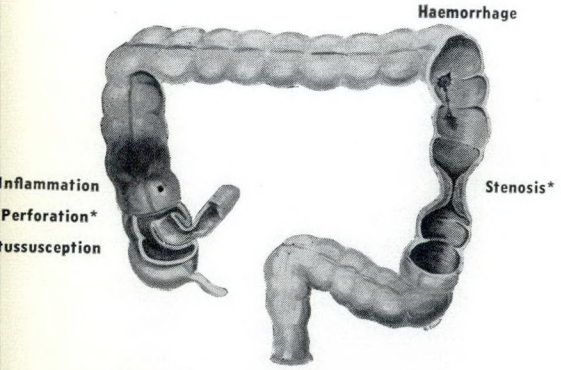


Fig. 2.—Complications—simple ulcer.

responsible for the high mortality rate in simple ulcer of the colon. These patients present as an acute abdominal emergency. Retroperitoneal perforation, with the formation of a retroperitoneal abscess, has also been reported.[5]

Stenosis is an important complication in the left colon. An extensive fibrosis accompanying a penetrating subacute or chronic ulcer may lead to stricture of the lumen, and result in bowel obstruction.

Diffuse inflammation of the adjacent bowel often accompanies an acute ulcer. This might mask the presence of the ulcer at operation, particularly in the cæcum, where a diagnosis of non-specific cæcitis can be made. An acute or chronic inflammatory mass may also be difficult to distinguish from carcinoma.

Massive hæmorrhage from a simple ulcer is unusual,[12] but occult bleeding is probably more common than has been realized. Intussusception associated with simple ulcer of the cæcum is a rare complication.[13]

The *differential diagnosis* is difficult, as the presenting features may mimic many intra-abdominal lesions (Fig. 3).

Acute appendicitis may be clinically indistinguishable from an acute simple ulcer of the cæcum in which perforation is imminent or has occurred. In about 70% of cases, a diagnosis of appendicitis was made.[6]

Diverticulitis of the cæcum is an uncommon lesion which may perforate and resemble a simple ulcer at the time of operation. Indeed, Fox is quoted[14] as believing that cæcal ulcers may be diverticula in which destructive inflammation has obliterated all traces of the wall, leaving only a residual ulcer.

Foreign body perforation or penetration of the bowel may be differentiated from simple ulcer by locating the foreign body at operation or by the histological presence of a foreign body reaction.

Carcinoma of the left colon may be diagnosed, clinically and radiologically, in a patient with a stenosing simple ulcer producing bowel obstruction. Differentiation grossly from a neoplasm may not be possible, even at operation.

Diverticulitis of the sigmoid colon may be suspected in a patient with a penetrat-

ing simple ulcer of the left colon, causing crampy abdominal pain.

Treatment

The treatment of simple ulcer of the colon has been varied. An acute simple ulcer which resolves and heals requires no therapy but is usually undiagnosed. A penetrating acute simple ulcer found at operation should be oversewn since perforation is a common and lethal complication.[15] There has been no reported survival without surgical treatment[10] and the operative mortality has in the past been as high as 40%.[9]

Simple drainage alone has always been fatal.[6] Oversewing of small perforations, or local excision, has been used with good results. Exteriorization procedures may be required for large perforations in poor risk patients, but there is a serious morbidity and further operation is required later.

With the advent of antibiotics, primary bowel resection has been the procedure of choice in recent reports.[16, 17] Where carcinoma could not be excluded at the time of operation in a simple ulcer of the cæcum associated with an inflammatory mass, right hemicolectomy has been successfully carried out on several occasions.[11, 14]

Case Reports

Case 1.—Miss L.W., a 49 year old matron, was admitted to the University Hospital on March 30, 1947, with a two-day history of crampy lower abdominal pain and diarrhœa. She had blood and mucus in her bowel movements. There was a history of "mucous colitis" about 20 years previously, which had subsided over a period of years. Since then she had been inclined to be constipated. Her appetite had been poor during the preceding two to three months and she had lost 15 lb. in this interval.

On examination, there was slight tenderness throughout the abdomen, particularly in the left lower quadrant. A mass was palpable in the region of the sigmoid colon. Temperature on admission was 99° F., hæmoglobin value was 13.2 g. %, and leukocyte count was 13,900. The clinical diagnosis was diverticulitis.

The patient's symptoms and abdominal tenderness subsided during the next few days. On April 2, a barium enema examination demonstrated a filling defect in the upper sigmoid colon, suggestive of malignancy (Fig. 4). On

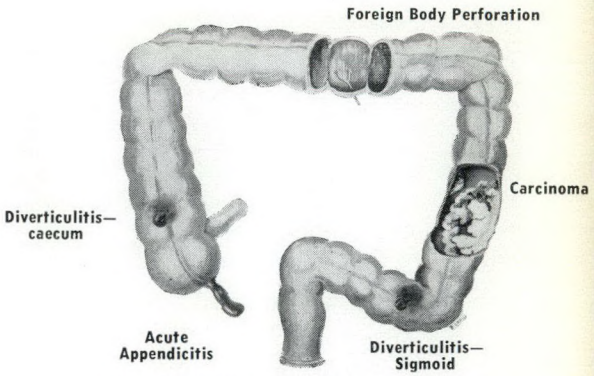


Fig. 3.—Differential diagnosis.

April 8, a repeat examination showed this defect to be persistent and irregular.

With a preoperative diagnosis of carcinoma of the colon, the bowel was prepared and the abdomen was explored by one of us (W.C.M.) on April 17. A firm indurated mass was found in the upper sigmoid colon, constricting the bowel lumen. This mass felt softer than a malignant tumour. However, it encircled the bowel and was of an obstructing nature, so a segmental resection of this portion of the colon was carried out with an end-to-end anastomosis restoring bowel continuity.

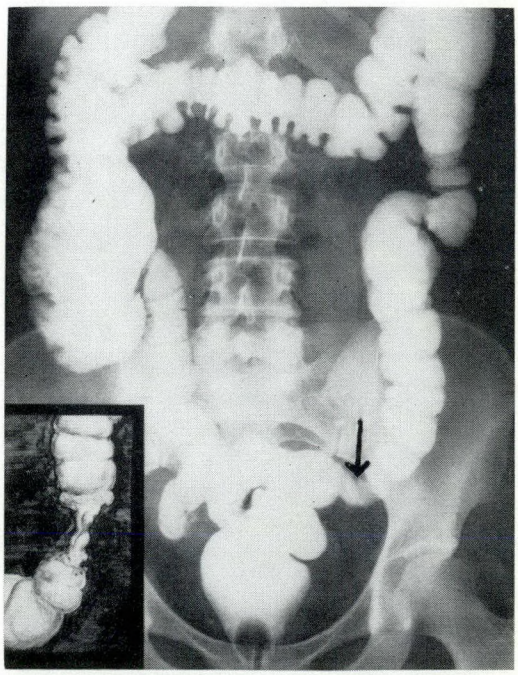


Fig. 4.—Case 1: The napkin-ring, constricting appearance of the lesion is portrayed in the corner insert. The large roentgenogram demonstrates a normal colon five years postoperatively; the arrow indicates the site of anastomosis.

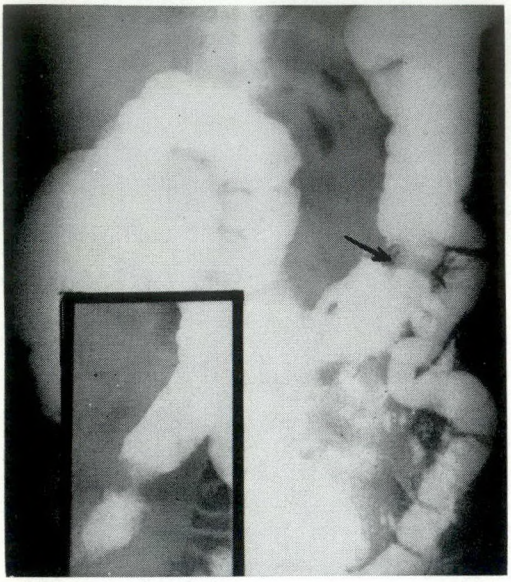


Fig. 5.—Case 2: The large roentgenogram and the spot film in the insert reveal a narrowed segment in the left transverse colon.

The gross specimen revealed a shallow ulcer in the bowel mucosa, about 3 cm. in diameter. Histological sections demonstrated a non-specific ulcer extending to the muscularis mucosa. The base was formed by granulation tissue and there was a moderate subacute inflammatory reaction of all layers of the adjacent bowel wall.

The patient's postoperative course was uneventful and she was discharged in satisfactory condition.

During a readmission in December 1952, for osteoarthritis of the spine, a follow-up barium enema examination showed no abnormality (Fig. 4). She has remained clinically well since then.

CASE 2.—Mrs. A.J., a 57 year old housewife, was admitted to the University Hospital on December 22, 1957, complaining of crampy upper abdominal and left upper quadrant pain of three days' duration. There was associated nausea, vomiting and diarrhœa. On March 1, 1957, a radical mastectomy had been carried out for scirrhous carcinoma of the left breast. Metastases had been present in the axillary nodes. Several months later, metastases appeared in the skin of the operative area but regressed after radiation and œstrogen therapy. Cortisone was not administered. She had no previous abdominal complaints.

On examination, there was marked tenderness and guarding in the epigastrium and left upper quadrant. An indefinite tender mass was palpable in the left upper quadrant. Temperature on admission was 99° F., Hb. value 12.1 g. %

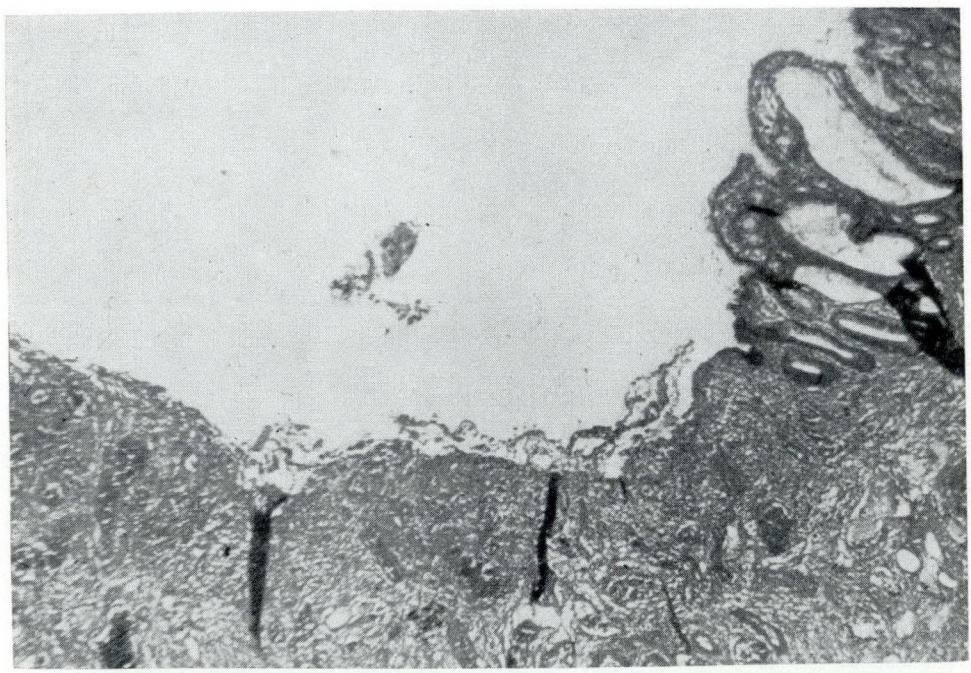


Fig. 6.—Case 2: Low-power photomicrograph (x 40) at edge of simple ulcer of the transverse colon (H & E stain).

and leukocyte count 12,150. Occult blood was present in her bowel movements.

Her abdominal symptoms and tenderness gradually subsided with supportive therapy. On January 3, 1958, a barium enema examination demonstrated a narrowing in the transverse colon near the splenic flexure. Multiple diverticula were present in the descending and sigmoid colon. The narrowed area in the transverse colon was persistent in a repeat examination on January 8, and appeared characteristic of a neoplasm (Fig. 5).

With a preoperative diagnosis of carcinoma of the colon, the bowel was prepared and the abdomen was explored by Dr. T. S. Wilson on January 14. A firm constricting mass was found in the left transverse colon, with omentum and a loop of small bowel adherent to the mass. A frozen section showed only inflammatory cells. The liver was normal. The colon was then mobilized and a segmental resection of the lesion was carried out with end-to-end anastomosis restoring bowel continuity. The specimen was opened in the operating theatre, revealing an annular, shallow mucosal ulcer about 1.5 cm. in diameter. On gross examination, the pathologist could not be certain whether this was a benign or malignant ulcer.

Histological sections revealed a benign, non-specific ulcer, penetrating to the sero-muscular layer. The base was formed by hyperæmic granulation tissue, infiltrated by large numbers of subacute inflammatory cells (Fig. 6).

The patient's postoperative course was uncomplicated and she was discharged feeling well.

CASE 3.—Mr. S.N., a 59 year old farmer, was admitted to the University Hospital on April 18, 1958, with a history of intermittent crampy pain in the left upper abdomen and diarrhœa of two months' duration. He had lost 30 lb. in weight during this period. There were no previous abdominal complaints.

Examination of the abdomen revealed slight tenderness to deep palpation in the left upper quadrant. Temperature on admission was 98° F., Hb. value 14.3 g. % and the leukocyte count 8600. A barium enema examination on April 19 demonstrated a persistent narrowing in the splenic flexure of the left colon (Fig. 7). There was moderate obstruction to the flow of barium at this site and the constricting lesion was suggestive of carcinoma.

With a preoperative diagnosis of carcinoma of the colon, the bowel was prepared and the abdomen was explored by one of us (W.C.M.) on April 28. A soft narrowed area was found in the splenic flexure which did not have the appearance or consistency of a malignant tumour.

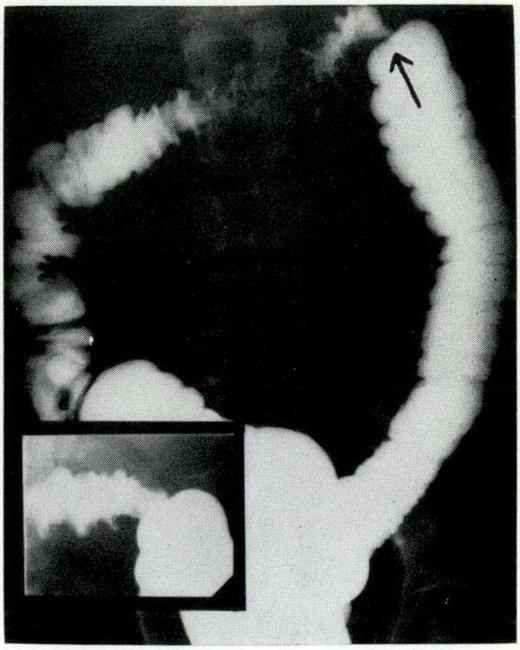


Fig. 7.—Case 3: The large roentgenogram and the spot film in the insert demonstrate incomplete obstruction at the splenic flexure.

A band of omentum had become adherent to the bowel in the region of the lesion, extending over the intestine anteriorly and causing constriction (Fig. 8). This band was freed and a soft palpable mass within the bowel wall was found, partially obstructing the lumen. A segmental resection of the splenic flexure was then performed and bowel continuity re-established with an end-to-end anastomosis.

The gross specimen demonstrated a shallow mucosal ulcer, about 1.5 cm. in width, encircling the bowel. The base of the ulcer was soft.

Histological sections demonstrated a chronic, non-specific ulcer. The ulcer floor was covered with a thin layer of necrotic debris, and inflammatory exudate. Beneath this was a layer of granulation tissue infiltrated by chronic inflammatory cells. Fibrosis extended throughout the colonic wall, and the serosal coat was thickened by fibrous tissue.

The patient's postoperative course was satisfactory and he was discharged well.

CASE 4.—Mr. A.M., a 74 year old insurance executive, was admitted to the University Hospital on September 26, 1958, with a five hour history of acute abdominal pain. The pain began suddenly in the peri-umbilical region, one-half hour after his evening meal, and was followed by vomiting. Severe pain rapidly became generalized

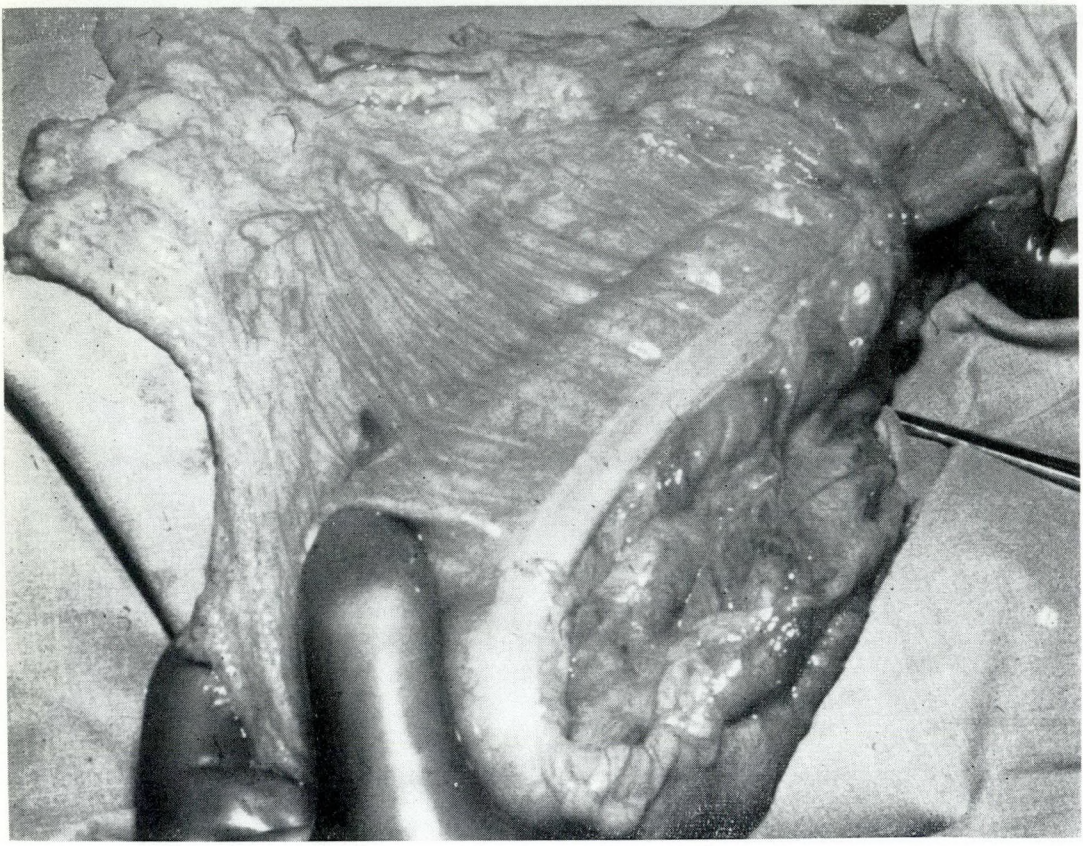


Fig. 8.—Case 3: Photograph at operation illustrates a band of omentum adherent to the constricted bowel at the splenic flexure.

throughout the abdomen with radiation to the right shoulder. He had no previous abdominal complaints other than occasional mild bloating and increasing constipation of 18 months' duration.

This patient was acutely ill with a rigid, tender abdomen. Temperature on admission was 97° F., heart rate was 130, Hb. value was 15.4 g. % and the leukocyte count was 6800. A flat film of the abdomen and a chest radiograph showed free air under the right diaphragm (Fig. 9). A preoperative diagnosis of perforated abdominal viscus was made, the most likely cause being considered a perforated peptic ulcer.

The abdomen was explored through a right paramedian incision by Dr. G. L. Willox two hours after admission to hospital. Half a litre of turbid fluid containing particles of bowel contents was found in the peritoneal cavity. There was no ulceration in the stomach or duodenum. On the medial aspect of the anterior wall of the cæcum, a perforation of the bowel was found. The adjacent cæcal wall was inflamed and œdematous. In addition, on the lateral aspect of the anterior wall of the cæcum, proximal to the perforation, there was a hard irregular mass, which felt like a carcinoma. Nodes in the adjacent mesentery appeared to be inflammatory. The liver was normal. Despite the early peritonitis, a definitive resection was considered the procedure of choice and a right hemicolectomy was performed with end-to-end anastomosis of ileum and transverse colon.

The gross specimen contained an ulcerating carcinoma about 2.5 cm. in diameter on the lateral cæcal wall, opposite the ileo-cæcal valve. The carcinoma was not producing obstruction. Several centimetres distal to the cancer, on the medial wall of the cæcum, was an acute perforated ulcer, about 0.5 cm. in diameter.

Histological sections of the tumour confirmed the presence of a Grade III adenocarcinoma of the cæcum. There were no metastases in the regional lymph nodes. Sections at the edge of the perforation showed an acute, non-specific inflammatory reaction in the bowel adjacent to the ulcer and no tumour in this area.

The patient's postoperative course was prolonged by a wound infection but was otherwise uneventful, and he was discharged feeling well.

Discussion

In the patient with a carcinoma of the cæcum and a perforation of the cæcum distal to the neoplasm (Case 4), it is probable that the acute ulcer was associated with the tumour but the relationship is not clear. Perforation with carcinoma of the colon is the presenting feature in about one out of twenty patients.[18] The perforation is at the site of the lesion in 80% of these patients.[19] Diastatic perforation, that is perforation in distended bowel (frequently the cæcum), which is proximal to an obstruction, occurs in the remaining 20%.

We have been unable to locate any description in the literature of a spontaneous perforation occurring distal to a malignant tumour of the colon and, therefore, have included the last case in this report as one of perforated simple ulcer distal to a carcinoma of the cæcum.

Perforation of the bowel in association with carcinoma is a complication of the utmost gravity, and primary resection has recently been advocated if the patient's general condition permits.[19]

The remaining three patients had a simple ulcer of the left colon. In each instance a preoperative diagnosis of carcinoma was made, based on clinical and radiological findings. In no patient could malignancy be definitely eliminated as a possibility at the time of surgery and a limited bowel resection was performed in each case with satisfactory results.

In the right colon, simple ulcer of the cæcum resembling appendicitis has received attention in the past. Undoubtedly, if the appearance of the appendix does not serve to explain the clinical findings in a patient operated upon for acute appendicitis, careful examination of the cæcum for a penetrating or perforating simple ulcer should be as mandatory as searching the terminal ileum for a Meckel's diverticulitis.

In the left colon, it appears worthy of emphasis that a penetrating simple ulcer may produce obstruction, simulating carcinoma clinically and radiologically. The differentiation cannot always be made with certainty, even at operation. Resection of the involved colon, whenever feasible, would seem to be the treatment of choice.

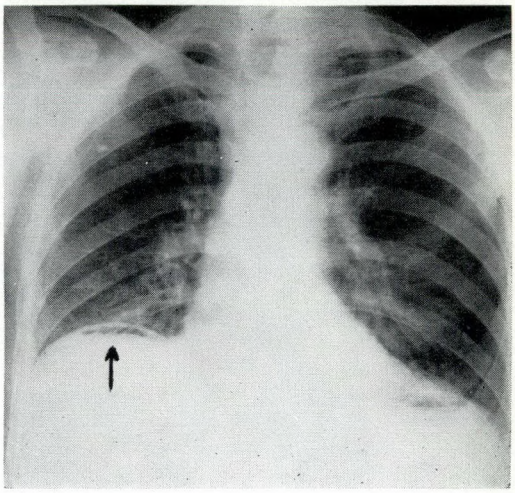


Fig. 9.—Case 4: Roentgenogram of the chest reveals free air beneath the right hemidiaphragm.

Summary

Simple ulcer of the colon is an uncommon lesion but one which surgeons must not overlook.

The lesion is probably more common than has been believed. The etiology is unknown. The diagnosis has very rarely been made preoperatively. Acute simple ulcer of the right colon resembles appendicitis; simple ulcer of the left colon may cause stenosis and mimic carcinoma.

Case studies of four patients have been presented: three patients with simple ulcer of the left colon and one patient with a perforated simple ulcer of the cæcum and a carcinoma of the cæcum.

Surgical therapy is required for the patient presenting with a complication of simple ulcer and resection of the lesion, whenever possible, is advocated.

References

1. Cruveilhier, J.: Anatomie pathologique: maladies des intestins, Baillière, Paris, 1830.
2. Morel and Scheyron: Ulcération du cæcum; pseudo-néoplasme inflammatoire; résection iléo-cæcale; guérison, *Soc. méd. Toulouse*, 1895, discussion by Quenu and Duval.[3]
3. Quenu E. and Duval, P.: L'ulcère simple du gros intestin, *Rev. de chir.*, **26:** 692, 1902.
4. Barron, M. E.: Simple, non-specific ulcer of the colon, *Arch Surg.*, **17:** 355, 1928.
5. Wilkie, Sir D.: Simple ulcer of the ascending colon and its complications, *Surgery*, **1:** 655, 1937.

6. Barlow, D.: "Simple" ulcers of the cæcum, colon, and rectum, *Brit. J. Surg.*, **28**: 575, 1941.
7. Barlow, D.: Simple ulcers of the cæcum, colon and rectum. *In*: Abdominal operations, R. Maingot, Appleton-Century-Crofts, Inc., New York, 3rd ed., 1955.
8. Rosser, G.: Simple penetrating ulcer of the cæcum, *Ann. Surg.*, **119**: 377, 1944.
9. Cromar, C. D. L.: Benign ulcer of the cæcum, *Am. J. Digest. Dis.*, **13**: 230, 1946.
10. Cameron, J. R.: Simple non-specific ulcer of the cæcum, *Brit. J. Surg.*, **26**: 526, 1939.
11. Eagleson, W. M.: Non-specific ulcer of the large bowel, *Canad. M. A. J.*, **67**: 653, 1952.
12. Bosien, W. R. and Crandell, W. B.: Hemorrhage from a benign ulcer of the sigmoid colon, *New England J. Med.*, **251**: 944, 1954.
13. Blum, L.: Nontumid ileocolic intussusception in an adult—report of a case with cecal ulcer, *S. Clin. North America*, **27**: 355, 1947.
14. Lipton, S. and Reisman, E. D.: Simple ulcer of the cecum, *Ann. Surg.*, **134**: 279, 1951.
15. Parker, R. A. and Serjeant, J. C. B.: Acute solitary ulcer and diverticulitis of the cæcum, *Brit. J. Surg.*, **45**: 19, 1957.
16. Watts, G. T.: Simple ulcers of the colon, *Postgrad. M. J.*, **31**: 414, 1955.
17. Durham, M. W. and Holm, J. C.: Simple perforated ulcer of the colon, *Surgery*, **34**: 750, 1953.
18. Witowski, L. J., Pontius, G. V. and Anderson, R. V.: Primary hemicolectomy for perforated lesions of the right colon, *A. M. A. Arch. Surg.*, **74**: 741, 1957.
19. Goligher, J. C. and Smiddy, F. G.: The treatment of acute obstruction or perforation with carcinoma of the colon and rectum, *Brit. J. Surg.*, **45**: 270, 1957.

Résumé

L'ulcération simple du côlon fut décrite pour la première fois en 1830 par Cruveilhier. On n'a recueilli qu'une centaine de cas de cette lésion dans la littérature médicale, bien que sa fréquence doive être plus élevée que ce chiffre ne le laisse croire. Les hommes en sont atteints deux fois plus souvent que les femmes. La moitié des cas intéressant le côlon se situe dans le cæcum. L'étiopathogénèse en est inconnue en dépit des multiples théories qui ont été avancées sur ce sujet.

On a rarement diagnostiqué l'ulcère simple du côlon autrement qu'à l'opération ou à l'autopsie. Les signes cliniques varient selon le site de la lésion, son acuité ou sa chronicité ainsi que ses complications. Au point de vue anatomopathologique elle se présente comme un ulcère isolé dont les dimensions peuvent varier de quelques millimètres à plusieurs centimètres, de forme ovale, linéaire ou annulaire. La proportion des ulcères qui guérissent spontanément est difficile à évaluer puisqu'ils laissent fort peu de traces visibles même à l'opération.

La perforation avec péritonite est sans doute la complication la plus fréquente (50%). La sténose cicatricielle vient en second lieu. L'inflammation des structures adjacentes peut causer une certaine confusion en simulant la présence d'une typhlite ou d'un carcinome. La grande hémorragie et l'invagination sont rarement impliquées. Le diagnostic différentiel comprend l'appendicite aiguë, la diverticulite du cæcum ou du sigmoïde, la perforation intestinale par corps étranger et le carcinome du côlon ascendant. Un ulcère pénétrant découvert à l'opération doit être enfoui afin d'éviter le risque d'une perforation. Le drainage seul a toujours été uniformément fatal. Une perforation de forte dimension en présence d'un mauvais état général du malade peut exiger un procédé d'extériorisation. Grâce aux antibiotiques la meilleure technique semble être la résection intestinale. Quatre cas sont présentés en guise d'illustration.

EMERGENCY SURGERY. Hamilton Bailey. International College of Surgeons. 1197 pp. Illust. 7th ed. John Wright and Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1958. $32.00.

The seventh edition of this well known book deserves high commendation. The entire field of emergency surgery is covered. The leading chapters deal with general considerations such as armamentarium, blood and fluid and electrolyte replacement, wounds, and infections. This is followed by several chapters on abdominal emergencies and genito-urinary emergencies, emergencies involving the thorax, the spine, head and neck, the extremities, ear, nose and throat, and tropical emergencies. There is a large appendix with addenda pertinent to several of the regions discussed. The subject matter is right up to date, and profusely illustrated with the quality that one has come to expect in Hamilton Bailey's texts.

There is little fault to find with this book. This reviewer finds little merit in the large number of illustrative case histories in the authors' (sic) own experiences, though this device will be approved by those who like this sort of emphasis in the presentation of facts. This is not a very serious shortcoming, because, all in all, this text is a handsome and useful production. It would be an especially appropriate gift for a young surgeon setting out to do general surgery (in the true sense of the term), but almost of comparable value to the experienced surgeon who requires a handy reference.

THE NORMAL MOVEMENT AT THE SUB-TALAR JOINT

MICHAEL C. HALL, M.R.C.S.,* *Toronto*

THE SUB-TALAR JOINT is frequently a site of deformity or injury and a full understanding of the normal movement is of advantage when considering the abnormal and in contemplating arthrodesis. This movement has been described in occasional articles in the anatomical literature, but it has not yet penetrated into many of the textbooks, and does not therefore appear to be general knowledge. Although some of these articles have been supported by good experimental work, in none of them is there a demonstration of the actual movement.

The work describing it has been performed by Manter,[1] Shephard[2] and Hicks.[3] They have all come to the conclusion, by different methods, that the sub-talar joint has a single axis around which the calcaneum rotates. This axis passes from the lower lateral posterior margin of the calcaneum, through the canalis tarsi, to the upper medial border of the neck of the talus.

Manter[1] made use of a fixed calcaneum and wires coming from the talus. During movement of the talus these wires described circles on glass plates above and below the foot. By connecting the centres of these circles the axis was calculated.

Shephard[2] arrived at the axis mathematically by connecting the centre of the circle, of which the articular area of the head of the talus formed an arc, with the centre of the circle of which the posterior talo-calcaneal joint formed an arc. The same axis was drawn. He compared movement at the sub-talar joint with the pronation and supination of the forearm.

Hicks,[3] after an extensive experiment using fresh frozen limbs, came to the same conclusion. He made use of a rod attached to one bone and malleable wires attached to the other. When he found by trial and error a position for the rod in which it did not deviate from the wires during movement, that position was taken to be the axis. This axis varies slightly from foot to foot, but is approximately at 46° to the horizontal and 16° to the sagittal axis of the foot. Manter came to the conclusion that there was a progression of the calcaneum along this axis comparable with a screw. Hicks[3] disagreed with this, and it

Fig. 1.

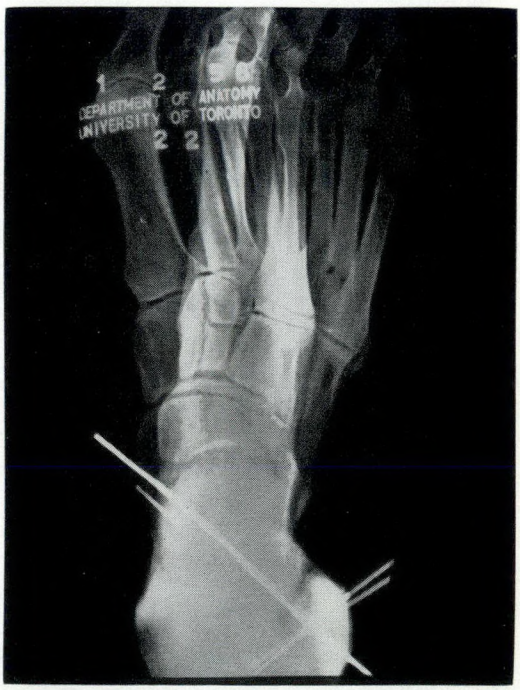


Fig. 2.

*Richardson Research Fellow, Department of Anatomy, University of Toronto.

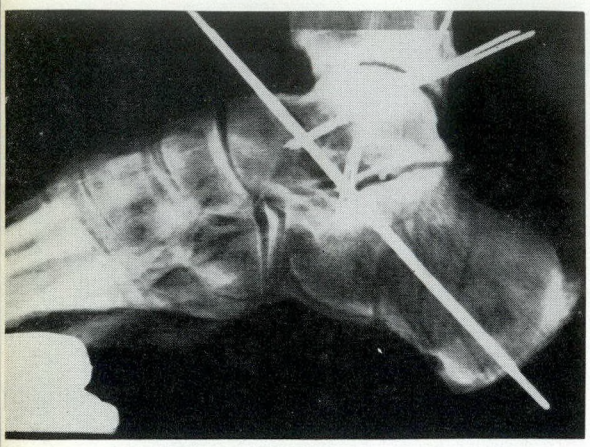


Fig. 3.

has not been shown in the findings to be described.

The descriptions in the standard texts, however, lack this simplicity. Gray[1] describes "a combination of movement and rotation" and refers to Philip Wiles. Wiles[5] described the interosseous ligament as the axis of rotation, but then broke down the movement into rotation around a vertical axis, tilting round a sagittal axis, and shift along both these axes. This would appear to be therefore multiaxial rather than a uniaxial movement. His diagram supporting his assertion is not from radiographs.

Grant[6] describes the talus sliding like a bolt on its long axis but in his first edition[7] he had compared the movement with that in the forearm. Frazer[8] also says that the talus slides on the calcaneum. Buchanan[9] describes a sliding movement. Morris[10] describes an axis similar to the one shown but emerging through the lateral side of the calcaneum. Cunningham[11] refers to Manter's work.

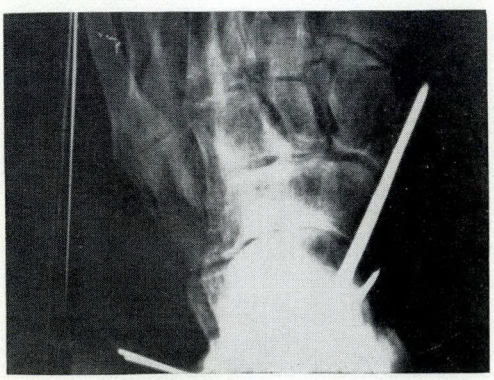


Fig. 4.

This general lack of clarity reaches its peak in Steindler's[12] application of Fick's[13] description—"a wobbly shift gliding about a series of instantaneous axes".

Methods and Materials

In this study cadaveric lower legs were used. Such tendons as were inhibiting movement, in fact the flexors, were divided. No ligaments were cut. The use of cadaveric material in this type of experiment is always open to criticism, but the character of a movement depends primarily on the shape of the articulation, which is not altered, and no conclusion has been drawn in regard to the range of movement.

The leg was fixed to a heavy wooden board by metal bands, thus allowing movement of the foot without movement of the leg, and permitting superimposition of the radiographs. Movement of the talus was eliminated by fixing it to the tibia with crossed Kirschner wires.

Results

With the talus in the neutral position radiographs were taken of the calcaneum in the everted and then the inverted position. If these are superimposed (Fig. 1) it will be seen that in the lateral view there is complete overlapping of the shadow of the postero-inferior calcaneal border, but that anteriorly the anterior border of the calcaneum in the inverted position is in advance of the everted, its shadow having lengthened by turning into the long axis of the foot. Since the superior border is closer to the axis of rotation, there is a lesser but quite definite alteration of position of it also. There is no shift in the postero-inferior border of the calcaneum, since this is the site of the axis of rotation and rotation alone is occurring. This is in contradistinction to the claims of Manter and Wiles.

In the antero-posterior projection the same is shown (Fig. 2). The anterior border of the calcaneum passes beneath the head of the talus in inversion. Although the calcaneum appears to be advancing in this position, it is known that this is not so from

the lateral projection which shows no shift in the posterior border. It is in fact due to the larger shadow cast by the bone when turned into the axis of the foot.

The contention that the axis of rotation is a single one, passing through the bones from the postero-inferior border of the calcaneum to the supero-medial border of the talus, is supported by the radiographs showing a heavy wire along this axis and the movements occurring around the wire in the same manner as those occurring without it (Figs. 3 and 4).

That the position of this axis is not dependent upon the position of the talus is shown when one radiograph, taken in plantar flexion and inversion, is superimposed on a second taken in dorsiflexion and eversion. The appearance is identical with that obtained when the talus was fixed (Fig. 5).

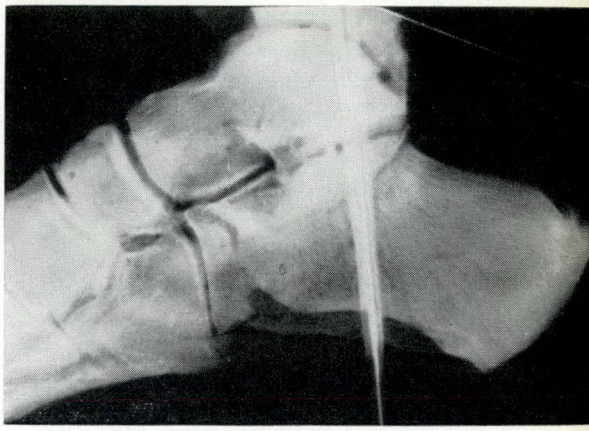


Fig. 5.

Interrelation of the Sub-talar and Transverse Tarsal Joints

These radiographs demonstrate that the movement of the sub-talar joint is around a single axis, and that there is no shift between the bones. The movement appears slight but it should be remembered that the foot is a long lever, and the excursion of the end of the lever is thereby greatly magnified. Since the tendons controlling this movement are inserted anterior to the transverse tarsal joint, when the calcaneum is at the limit of its rotation no further transverse tarsal joint movement is possible in the same direction, i.e., no forefoot abduction is possible in the fully everted foot or adduction in the fully inverted foot. Last[14] aptly describes active movement occurring at the sub-talar joint only when the transverse tarsal joint is first "wound up", and describes the uniaxial sub-talar movement. However, he says that relatively little movement is possible at the transverse tarsal joint.

Wiles[15] denied that the talo-navicular and calcaneo-cuboid joints formed together a functional unit and said that there was comparatively little movement in them when sub-talar movement was prevented. Inkster[16] wanted to discard the term "transverse tarsal joint" since he regarded all the tarsal bones except the talus as bound together as a comparatively rigid but elastic mass.

However, it can be shown that a significant rotation is possible at the transverse tarsal joint in a direction opposite to that at the sub-talar joint. This can readily be confirmed by maintaining the forefoot flat on the ground while lifting the outer or inner border of the calcaneum, or by holding the heel firmly in the hand and

Fig. 6.

Fig. 7.

rotating the forefoot with the other. It is demonstrated in the radiograph by blocking both ankle and sub-talar movement with Kirschner wires, and then rotating the forefoot. This rotation is frequently seen clinically in the child with the everted heel, and is of great importance when walking over rough ground and maintaining balance generally, particularly when standing on one foot. Jones[17] believes that the movement of this joint has a proprioceptive function in the maintenance of balance.

The radiographs show the lateral borders of the cuboids in the positions of forefoot abduction and adduction when the calcaneum is fixed in eversion (Fig. 6), and in inversion (Fig. 7), showing a significant excursion. Since active movement at the talo-calcaneal joint follows the "wound up" transverse tarsal joint, movement of the forefoot and hindfoot in opposite directions can only be passive and falls into Gray's[4] "primary accessory" group.

If the extent of this normal movement at the transverse tarsal joint is appreciated, it is easier to understand the range of inversion and eversion that may still be present in the adaptable foot of the young after a sub-talar arthrodesis.

Summary

The single axis of the sub-talar joint is described and the description is substantiated by radiographs demonstrating rotation of the calcaneum around this axis. Any shift at this joint is denied.

The rotation at the transverse tarsal joint opposite to that at the sub-talar joint is described, and demonstrated in radiographs. Its function in balance and posture is discussed.

Acknowledgments

I wish to express my gratitude to Professor J. W. A. Duckworth, to Professor F. P. Dewar, who suggested this work, and to Mr. C. Storton for photographic assistance.

References

1. Manter, J. T.: Movements of the sub-talar and transverse tarsal joints, *Anat. Rec.*, **80:** 397, 1941.
2. Shephard, E.: Tarsal movements, *J. Bone & Joint Surg.*, **33-B:** 258, 1951.
3. Hicks, J. H.: The mechanics of the foot; joints, *J. Anat.*, **87:** 345, 1953.
4. Johnston, T. B. and Whillis, J., Editors: Gray's Anatomy, Longmans, Green & Co., London, 30th ed., 1949.
5. Wiles, P.: Flat feet, *Lancet*, **2:** 1089, 1934.
6. Grant, J. C. B.: A method of anatomy; descriptive and deductive, Williams & Wilkins Co., Baltimore, 5th ed., 1952.
7. Grant, J. C. B.: A method of anatomy, William Wood & Co., Baltimore, 1st ed., 1937.
8. Frazer, J. E.: The anatomy of the human skeleton, J. & A. Churchill, Ltd., London, 4th ed., 1940.
9. Wood Jones, F., Editor: Buchanan's Manual of anatomy, Baillière, Tindall & Cox, London, 7th ed., 1946.
10. Schaeffer, J. P., Editor: Morris' Human anatomy, The Blakiston Company, Philadelphia, 10th ed., 1942.
11. Brash, J. C., Editor: Cunningham's Text book of anatomy, Oxford University Press, London, 9th ed., 1951.
12. Steindler, A.: Kinesiology of the human body under normal and pathological conditions, Charles C Thomas, Springfield, Ill., 1955.
13. Fick, I.: Handbuch d. Anatomie und Mechanik d. Gelenke, Fischer, Jena, 1911.
14. Last, R. J.: Anatomy: regional and applied, J. & A. Churchill, Ltd., London, 1954.
15. Wiles, P.: Essentials of orthopædics, J. & A. Churchill, Ltd., London, 2nd ed., 1955.
16. Inkster, R. G.: *J. Anat.*, **72:** 612, 1938.
17. Jones, R. L.: The human foot, *Am. J. Anat.*, **68:** 1, 1948.

Résumé

L'articulation sous-astragalienne est fréquemment le siège de traumatismes ou de difformités; une bonne compréhension de sa mécanique est de la plus haute importance dans la pathologie ou les interventions chirurgicales. Plusieurs auteurs (la littérature est ici passée en revue) se sont déjà occupés de cette question, sans toutefois étudier la mobilité réelle.

L'auteur du présent article a abordé ce problème expérimentalement; il utilisa des membres inférieurs de cadavres solidement fixés sur des supports par des bandes métalliques: les tendons furent sectionnés, mais tous les ligaments respectés. De cette façon, la jambe étant bien immobilisée, les mouvements du pied restaient tout à fait libres. L'astragale lui-même fut bloqué par des broches de Kirschner transfixiantes; des radiographies furent prises, notamment dans l'abduction et l'adduction.

On constate alors qu'il ne se fait aucun déplacement du bord postérieur et inférieur du calcaneum, mais une certaine rotation, dont l'axe part de ce même bord pour rejoindre le bord supérieur et interne de l'astragale. Les articulations transversales du tarse sont ensuite étudiées, en relation avec la sous-astragalienne. Là non plus, il n'existe aucun déplacement des os entre eux, mais un certain mouvement de rotation. Des considérations sur la fonction terminent cet article.

THE EFFECT OF TRIETHYLENE THIOPHOSPHORAMIDE (THIOTEPA) AND 17-ETHYL-19-NORTESTOSTERONE (NILEVAR) ON WOUND HEALING*

W. PISESKY, M.D., H. T. G. WILLIAMS, M.D., F.R.C.S.(Eng.), F.R.C.S.[C] and WALTER C. MacKENZIE, M.D., F.A.C.S., F.R.C.S.[C], *Edmonton, Alta.*

THE EXPERIMENTAL WORK of Warren Cole and others[1, 2] has demonstrated the value of chemotherapeutic agents in discouraging implantation of cancer cells. This has aroused a good deal of interest in the clinical application of these agents as adjuvants to the surgical management of neoplasms. One of the disadvantages of the chemotherapeutic drugs has been the interference with wound healing as demonstrated by an increase in the rate of evisceration following their use in abdominal surgery. It has long been known that the patient with cancer, when subjected to an operative procedure, runs an increased risk of wound dehiscence as a postoperative complication.[3] Therefore, the addition of these drugs with the possibility of further increasing this complication becomes a serious consideration.

Our experiment was designed, first of all, to test the clinical hypothesis that triethylene thiophosphoramide (Thiotepa, TSPA) does delay wound healing and, if so, to determine whether 17-ethyl-19-nortestosterone (Nilevar) would counteract the deleterious effect of the chemotherapeutic drug. Previous work in our laboratory has shown that this anabolic steroid will aid wound healing.[4]

MATERIALS AND METHODS

Young, healthy, male rats of the Wistar strain, weighing from 130 grams to 250 grams, were selected for this study. These rats were kept separately in individual cages and were fed with a standard diet of commercial food. A continuous supply of clean water was available. The rats were divided into three groups; the first was treated with Thiotepa, the second group was to act as a control having received no treatment, and the last group was treated with both Thiotepa and Nilevar.

The Thiotepa was prepared by dissolving the chemical in sterile water and passing the resultant solution through a Seitz filter. The drug was made up in a concentration of 4 mg./ml. and was used shortly after it was prepared. The dosage used was 1 mg. per rat given intraperitoneally at the time of operation. The Nilevar was mixed in the food and given in the amount of 5 mg. daily. Six doses preoperatively and 4 doses postoperatively were administered, so that each animal in group III received a total of 50 mg. of Nilevar. The oral route was chosen because it was shown by Kowalewski[5] that this route of administration was superior to the intramuscular route.

OPERATIVE PROCEDURE

Under ether anæsthesia, the abdominal wall of each rat was shaved and painted with 2.5% tincture of iodine. Under sterile conditions, a midline suprapubic incision, 2 cm. in length, was made and carried down through the linea alba into the peritoneal cavity. At this point, the Thiotepa was injected into the peritoneal cavity of the rats which were to receive this drug. The wound was then closed in two layers in a standard fashion, using two interrupted sutures of 000 chromic catgut on an atraumatic needle to close the peritoneum and fascia. The skin was approximated by means of two interrupted sutures of 0000 silk. Postoperatively, the wounds were inspected daily and a record kept of any infections. The infection rate was about 1% and these animals were excluded from the study. The skin sutures were removed on the seventh day postoperatively, or sooner if the animals were sacrificed earlier.

---

*Paper presented at the meeting of The Royal College of Physicians and Surgeons of Canada, January 1959, Vancouver, B.C.

From the McEachern Cancer Research Laboratory and Department of Surgery, University of Alberta, Edmonton, Alberta.

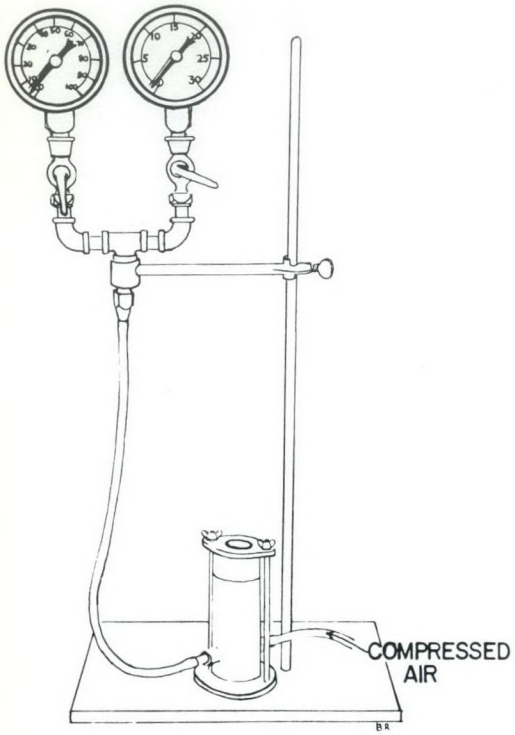


Fig. 1.—Device used for measuring bursting pressures of tissues. It is attached to a low pressure gauge and to a high pressure gauge by means of a set of valves.

The animals were killed on the 5th, 10th or 15th day postoperatively by an overdose of ether. The entire abdominal wall, comprising all the layers, was excised from each animal and placed in a device designed for measuring the tensile strength of tissues. The device is similar to the one described by Gouws and his colleagues.[4]

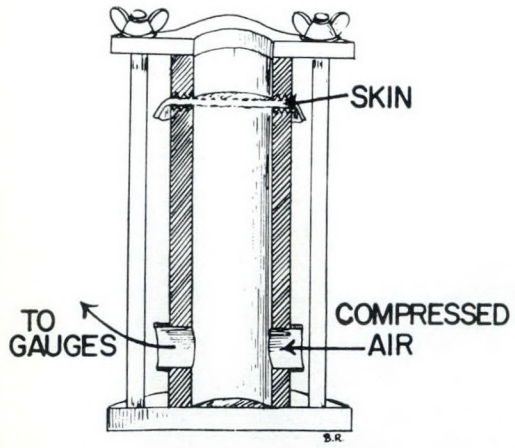


Fig. 2.—Cross section of the device used for measuring the bursting pressures of tissues. "Skin" refers to the entire abdominal wall of rats in this experiment.

Essentially, it consists of a hollow metal cylinder, closed at one end; over the open end the tissue to be tested is placed and held by a metal ring. The two parts come together making an airtight closure by virtue of the concentric grooves in the ring and in the top of the cylinder (Figs. 1 and 2). Entering the base of the cylinder are two openings, one of which leads to a source of compressed air; the other is connected to a low pressure gauge and to a high pressure gauge by means of a set of valves. These are self-registering gauges. In the line for the source of compressed air is a reducing valve which delivers the air at a constant rate of flow.

The surgical procedure and the technical measurement of the bursting pressures were carried out by the same operators at all times in order to try to eliminate the variations that may occur between operators.

With the tissue containing the wound clamped firmly into position over the open end of the cylinder, air under pressure was allowed to enter the cylinder until the wound disrupted. The pressure at which this occurred was automatically recorded on the gauge. The skin was then moved so that the intact linea alba of the abdominal wall was centred over the opening of the cylinder and the same procedure was carried out as for the wound. Care was taken at all times that all the layers of the abdominal wall were being treated. The tensile strength of the wound was then expressed as a percentage of normal, using the bursting pressure of the intact abdominal wall in each animal as 100%. Thereby, each animal acted as its own control. We feel that the method described is an adequate means for measuring the tensile strength of tissues.

Results

The results are summarized in Table I and depicted graphically in Fig. 3. From the table, it is apparent that the average bursting pressure of the Thiotepa treated wounds was 13.8 lb./sq. in., while the control wounds burst at 24.1 lb./sq. in., and

the wounds treated with Thiotepa and Nilevar burst at 30.7 lb./sq. in. five days after operation. There is a difference of 10.3 lb./sq. in. between the first two groups and a difference of 16.9 lb./sq. in. between the first and last groups. The differences are greater at the ten day period, but are about the same at the fifteen day period. When the tensile strength of the wounds is expressed as a percentage of normal, the difference between the various groups is slightly increased, but these remain approximately the same throughout the postoperative period.

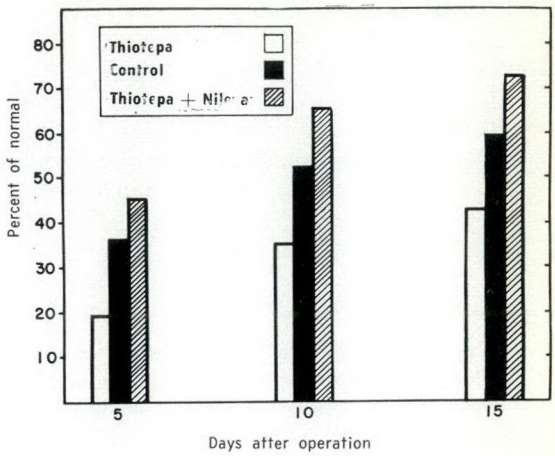


Fig. 3.—Tensile strength of experimental wounds expressed as a percentage of normal using the bursting pressure of the intact abdominal wall as 100%.

Discussion

Controversy exists in the current literature regarding the influence that cancerocidal chemotherapeutic drugs may have on wound healing. Hardy *et al.*[6] have shown that gastrotomy wounds in adult dogs were apparently unaffected by the administration of nitrogen mustard and Thiotepa under certain experimental conditions. Musselman and his co-workers attempted to demonstrate the influence of nitrogen mustard[7] and Thiotepa[8] on wound healing in rats. They found that nitrogen mustard retarded wound healing but Thiotepa had no demonstrable effect. Krementz and his associates,[9] in a study of this problem, were able to show a significant weakness in gastrotomy wound tensile strength in guinea pigs following the administration of triethylene melamine (TEM).

The active radicle of nitrogen mustard, Thiotepa and TEM is the ethylamine group. The action of these drugs is similar and consists of a non-specific cytotoxicity on the nuclei of rapidly dividing cells.[10] One would therefore expect these agents to have some effect on fibroblastic proliferation. It would appear from our results that Thiotepa retards wound healing under the experimental conditions described.

A new synthetic anabolic steroid has been used both experimentally and clinically in the hope of aiding wound healing. Nilevar has been shown by Drill and Saunders[11] to have remarkable protein-sparing powers in the postoperative period. In addition, Kowalewski and Gouws in our laboratory[12] have demonstrated that radiosulfur is selectively incorporated into heal-

TABLE I.—Effect of Thiotepa and Nilevar on Bursting Pressures of Abdominal Wounds

			Average bursting pressure (pounds/square inch)			*Percentage tensile strength of normal*	
Days postoperative	*No. of rats*	*Group No.*	*Wound*	*S.D.*	*Intact abdominal wall*	*Average*	*S.D.*
5	12	I	13.8	6.9	74.2	19.5	16.4
	12	II	24.1	10.9	69.3	36.3	14.1
	12	III	30.7	7.2	68.3	45.4	11.6
10	12	I	21.6	9.4	61.6	35.3	12.9
	14	II	39.3	8.8	74.4	52.1	13.1
	11	III	47.6	8.2	73.4	65.5	12.2
15	12	I	30.1	12.7	74.1	42.3	11.6
	15	II	43.1	6.2	72.7	59.3	16.5
	11	III	51.1	8.9	69.9	73.3	11.5

Group I.—Thiotepa treated. Group II.—Control. Group III.—Thiotepa-Nilevar treated.

ing tissues in animals. This process is enhanced by the administration of Nilevar when compared to control groups. One might expect this drug to offset the deleterious effect that might be produced by Thiotepa. The exact mechanism through which this may occur is not known, but it would appear to have occurred under the conditions of this experiment.

Summary

The effect of triethylene thiophosphoramide (Thiotepa) on wound healing was investigated by measuring the bursting pressures of experimental wounds. This drug was combined with 17-ethyl-19-nortestosterone (Nilevar) and their effect on wound healing was similarly studied.

It would appear that, under the experimental conditions described, Thiotepa retarded the wound healing process and that Nilevar would counteract this delay. This may have clinical application when Thiotepa is being used as an adjuvant in the surgical treatment of cancer.

References

1. McDonald, G. O., Cruz, E. P. and Cole, W. H.: The effect of cancer inhibitor drugs on the 'take' of Walker carcinoma 256 in rats, *S. Forum*, **7**: 486, 1956.
2. Cruz, E. P., McDonald, G. O. and Cole, W. H.: Prophylactic treatment of cancer, *Surgery*, **40**: 291, 1956.
3. Starr, A. and Vason, L. H.: Postoperative rupture of abdominal wounds, *J. A. M. A.*, **100**: 310, 1933.
4. Gouws, F., Silbermann, O. and MacKenzie, W. C.: The effect of 17-ethyl-19-nortestosterone (Nilevar) on healing of experimental wounds, *Canad. J. Surg.*, **1**: 362, 1958.
5. Kowalewski, K.: Comparison of the effects of cortisone and certain anabolic-androgenic steroids on the uptake of radiosulfur in a healing fractured bone, *Endocrinology*, **62**: 493, 1958.
6. Conn, J. H., Leb, S. M. and Hardy, J. D.: The effect of nitrogen mustard and Thiotepa on wound healing, *S. Forum*, **8**: 80, 1957.
7. Farhat, S. M. *et al.*: Effect of mechlorethamine hydrochloride (nitrogen mustard) on healing of abdominal wounds, *A. M. A. Arch. Surg.*, **76**: 749, 1958.
8. Musselman, M. M. *et al.*: The effect of intravenous nitrogen mustard and triethylene thiophosphoramide (Thiotepa) on healing of abdominal wounds, *A. M. A. Arch. Surg.*, to be published.
9. Krementz, E. T., Giddens, W. R. and Chapman, W. L.: Effect of triethylenemelamine on wound healing, *S. Forum*, **8**: 83, 1957.
10. Gilman, A. and Philips, F. S.: The biological actions and therapeutic applications of the ethylamines, *Science*, **103**: 409, 1946.
11. Drill, V. A. and Saunders, F. J.: Effects of Nilevar. *In*: Proceedings of a conference on the clinical use of anabolic agents, Searle Research Laboratories Publication, 1956, p. 4.
12. Kowalewski, K. and Gouws, F.: The effect of 17-ethyl-19-nortestosterone (Nilevar) on the uptake of radiosulfur in the fractured humerus in the rat, *Surg. Gynec. & Obst.*, **105**: 1, 1957.

Acknowledgments

This study was supported in part by Research Grant No. 208 from The National Cancer Institute of Canada. Nilevar was supplied by G. D. Searle & Co., Chicago, Ill.

Résumé

Expérimentalement il a été possible de démontrer la valeur des agents chimiothérapeutiques dans l'inhibition de la croissance cancéreuse. Ceci a soulevé un grand intérêt en clinique, mais le principal inconvénient de ces drogues est de gêner la cicatrisation des plaies opératoires; on a alors assisté à une augmentation du nombre des éventrations post-opératoires.

Les auteurs ont étudié chez l'animal les deux problèmes suivants: d'abord savoir si réellement le triéthylène-thiophosphoramide retarde la cicatrisation; puis déterminer si l'association de 17-éthyl-19-nortestosterone est capable de réduire ou même de supprimer cet inconvénient.

Des rats blancs mâles, gardés et nourris de façon identique, furent répartis en trois groupes; le premier reçut du triéthylène-thiophosphoramide, à dose de 1 mg. lors de l'intervention; le second constitua un lot de contrôle, sans aucun traitement; le troisième la même dose de triéthylène-thiophosphoramide que le premier et en plus du 17-éthyl-19-nortestosterone en dix doses de 5 mg. dans la nourriture. Puis chacun de ces groupes subit une laparotomie de 2 cm. de longueur, dans des conditions aseptiques. Les animaux furent sacrifiés au cinquième, au dixième et au quinzième jour post-opératoire. La paroi abdominale fut excisée en totalité; on mesura alors la résistance de la cicatrice à la déhiscence grâce à l'appareil imaginé par Gouws et ses collaborateurs. Les résultats sont fournis sous forme de tableau; la conclusion des auteurs est que le triéthylène-thiophosphoramide retarde réellement la cicatrisation et que le 17-éthyl-19-nortestosterone est bien capable de s'opposer à cette action.

THE PRESENT STATUS OF THE GASTRIC ANTRUM – A COLLECTIVE REVIEW*

R. C. HARRISON, M.D., M.S., F.A.C.S., F.R.C.S.[C], *Edmonton, Alta.*

THE MAJOR MECHANISMS controlling gastric secretion were established by the classical work of Pavlov[1] and his associates half a century ago. These have remained undisputed, and have been termed the cephalic, gastric and intestinal phases. Pavlov showed that gastric acid production was stimulated by these three mechanisms and set the stage for further clarification of their pathways and interrelationships.

Although we are primarily concerned in this review with the role of the antrum, we will later consider the interrelationship between the antral and other phases, hence the reference to the other mechanisms. Certain emotional states, the sight, smell or taste of food, and hypoglycæmia result in a stimulation of the parietal cells. This is the cephalic phase, which has its origin in the hypothalamus and anterior pituitary and is mediated by the vagi. The by-products of protein digestion in the intestine stimulate acid secretion, and this is referred to as the intestinal phase. The gastric phase has become known as the antral phase, as it is now recognized that the stimulation of acid secretion resulting from food in the stomach is primarily antral in origin. In 1906, Edkins[2] suggested that food in the region of the antrum stimulated the production of a substance (which he called gastrin) which in turn stimulated the production of gastric acid. His postulate was not accepted as fact for many years, and during this period many supporting[3-6] and refuting[7] experiments were reported. Among these were the experiments of Ivy, who initially thought that the gastrin mechanism did not exist[8] and later concluded that it did.[9] It is now firmly established that the antrum does produce a humoral substance which stimulates acid production by the parietal cells, but because the actual agent has never been isolated or identified it is more correctly referred to as a humoral agent rather than a true hormone.

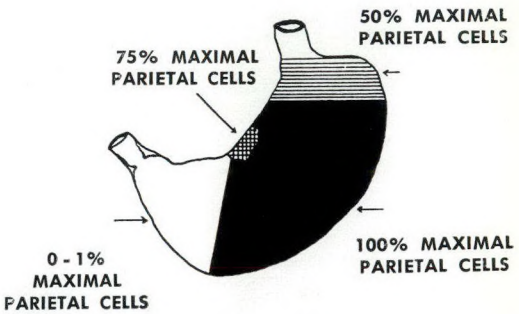


Fig. 1.—Distribution of parietal cells in stomach of human (after Berger, E. H., *Am. J. Anat.*, **54**: 87, 1934).

The antrum is that portion of the prepyloric area of the stomach whose proximal boundary is roughly identified by a line drawn from the incisura angularis to the greater curvature. It constitutes about 25% of the gastric mass, and whereas the remainder of the stomach secretes acid, pepsin and mucus, the antrum secretes an alkaline mucus. The general distribution of these areas is shown diagrammatically in Fig. 1.

It is apparent that the extent of distal gastrectomy, when expressed as a percentage, does not truly reflect the percentage of acid-secreting mucosa excised. Thus a 50% gastrectomy only removes one-third of the parietal cell mass, a 75% distal gastrectomy removes two-thirds, and a 65% gastrectomy only removes half of the acid-secreting mucosa. This is not universally appreciated.

Although the high ulcer recurrence rate after removal of only the antrum in the early days of gastric surgery tended to refute Edkins' theory, much supporting evidence for its importance came from the high incidence of ulcer after pyloric exclusion procedures, such as those described by Finsterer[10] and Devine.[11] Many surgeons[12, 13] reported healing of the recurrent ulceration after removal of the antrum, and not only

*From the Department of Surgery and the McEachern Laboratory, Faculty of Medicine, University of Alberta, Edmonton, Alberta.

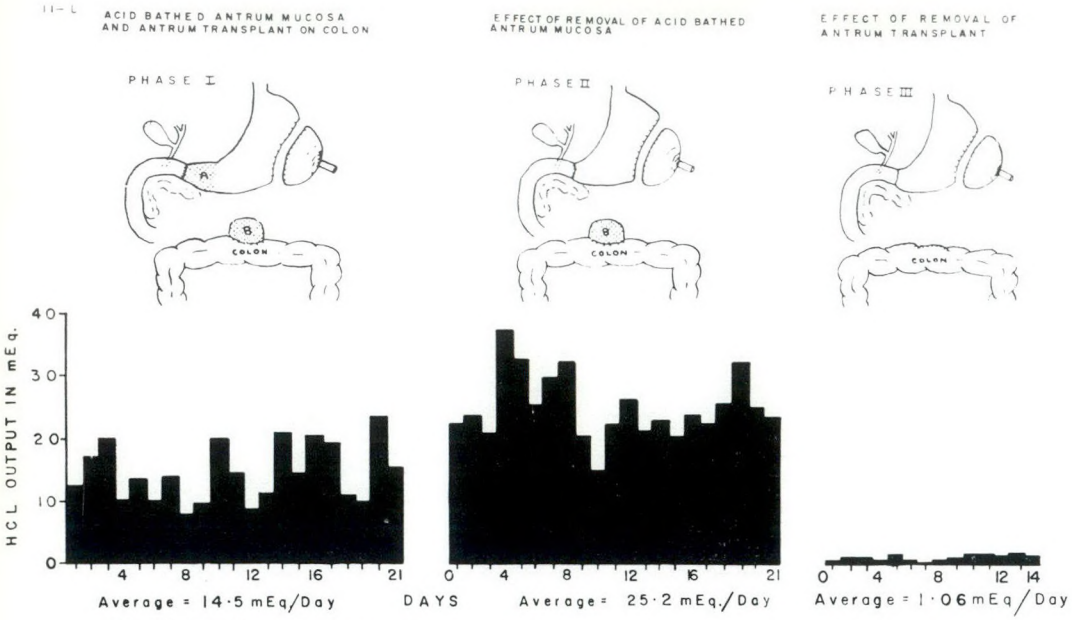


Fig. 2.—Evidence in favour of an antral inhibitor (from *Ann. Surg.*, **144:** 441, 1956).

lent clinical support to the gastrin theory but led investigators to consider the comparative influence of acid and alkali on the antral mechanism.

The antrum is stimulated to produce gastrin if food in a relatively alkaline medium is allowed to come in contact with the antral mucosa, or if the antrum is distended. Sokolov, working in Pavlov's laboratory, had shown many years previously that instillation of acid into the stomach tended to retard the further secretion of acid, and this had been confirmed by Wilhelmj.[14] This, coupled with the clinical evidence associated with pyloric exclusion, led to the investigation of the influence of pH changes on antral behaviour. Dragstedt[15] found that the usual stimulation of the antrum by food did not occur if the medium was acid. In logical sequence, it was debated whether or not this was due to a reduction in gastrin production, or due to the production of an actual inhibitor substance under these circumstances. In 1956, we demonstrated by means of a transplant of one-half of the antrum to the colon, that this mechanism of active inhibition probably did exist.[16]

Dragstedt[17] had previously demonstrated that, when the antrum was transplanted to the colon or duodenum, gastric pouch secretion was markedly stimulated. This appeared to be due to a stimulation of the gastrin mechanism, and this preparation was used by us to ensure that the remaining portion of the antrum would be in a highly acid environment. The effect on parietal cell secretion was measured by means of a gastric pouch, and it was found that when the gastric portion of the antrum was excised pouch secretion increased. This suggested the possibility of an active inhibitor substance (Fig. 2).

The history of gastric physiology, and particularly that with reference to the antrum, has been characterized by slow forward progress, in spite of the fact that every advance has been debated with conflicting results extending over several years before the evidence is finally accepted. We do not mean to suggest that this is also true with respect to the antral inhibitor, but to date there has developed a lively interest in whether or not an inhibitor does exist. As well as the experiment mentioned above, spontaneous secretion in gastric pouches has been found to be inhibited by acid irrigation of the antrum by both Uvnas[18] and Dragstedt.[17] Total gastric pouch secretion stimulated by histamine has increased in amount after removal of the antrum.[19] Woodward[20] has found that acid irrigation

of the antrum will inhibit histamine-stimulated pouch secretion, and has also confirmed the work of Margolus[21] by demonstrating inhibition of the intestinal phase. Jordan and Sand,[22] using double antral pouches, showed that acid irrigation is effective against stimulation provoked by alcohol, and Harkins[23] has shown that the innervated antrum transplanted to the colon does not stimulate acid production until it is denervated. It should be mentioned that, although both Dragstedt and Woodward in some of their experiments add to the evidence for an inhibitor, they do not believe that the thesis of an antral inhibitor has been proven. The negative evidence consists of experiments done by Dragstedt and Woodward[20] who did not confirm the findings of Harrison, Lakey and Hyde in similar experiments, and the failure to demonstrate inhibition of acid secretion when a double antral pouch is used.[24]

If gastric acid secretion is stimulated by any mechanism which is not antral in origin, its subsequent inhibition by acidification of the antrum suggests that an actual inhibitor substance is being produced in the antral area. The explanation for this reduction in secretion could hardly be a decrease in gastrin production, which is minimal or entirely lacking in such experiments. Thus, when Margolus[21] showed that acidification of the antrum inhibited secretion which was stimulated by the intestinal phase, it was important evidence. The suggestion that the secretion might have been secondary to mechanical stimulation by the cannula used in the antrum was discounted when removal of the cannula did not modify the results. Using insulin hypoglycæmia to stimulate acid secretion by the vagal route, Shimizu, Morrison and Harrison[25] found that acidification of the antrum also inhibited acid secretion of vagal origin. Dragstedt[26] did not demonstrate this to be so in earlier experiments, so confirmation of this experiment is needed. The findings of Margolus have been confirmed by Woodward, as previously noted. The fact that acidification of the antrum will reduce gastric acid production due to histamine stimulation certainly favours the concept of active inhibition by the antrum.

There is no question that the gastric mechanism is effective whether the antrum is vagally innervated or not, but there is some suggestion that the production of an inhibitor substance is affected by the presence or absence of intact vagal fibres to the antrum. In the experiments of Woodward, Margolus, and Shimizu, previously referred to, acid production was inhibited whether the antrum was innervated or denervated, but in Shimizu's experiment the inhibition was not as effective when the antrum lacked vagal innervation. Harkins[23] has experimental evidence to suggest that the innervated antrum inhibits acid production, whereas the denervated antrum has a stimulatory effect. Gouws and Harrison[27] repeated the experiment of Harrison, Lakey and Hyde using a denervated antrum, and did not find inhibition quite as effective. In repeating the experiment, Woodward also used a denervated antrum,[20] which might explain his negative findings. Dragstedt, however, left the antrum innervated and still failed to demonstrate evidence in favour of an active antral inhibitor.

Whether or not an actual inhibitor substance is produced by the antrum is of considerable practical, as well as theoretical, interest and whether or not this is modified by vagal innervation is also of importance. If there is such a mechanism, it would be more effective in terminating the secretion of the acid than a diminution in gastrin production alone, and moreover would also be more effective in controlling acid production stimulated by the other mechanisms. While it is by no means proven that an antral inhibitor does exist, there does not appear to be much doubt about the fact that the antrum does not stimulate acid production when its mucosa is bathed by acid. Under experimental conditions this is true if the pH is 5 or below, or if acid of the strength secreted by the parietal cells is used. At present, it is not known whether this acid-inhibiting mechanism is effective in the human, and how low the pH must fall before further acid production is inhibited. At present, also, procedures for the surgical treatment of duodenal ulcer can be divided into two large groups, depending on whether or not the antrum is

preserved. Those who resect the antrum argue that it is primarily a gastric acid stimulator, whereas those who preserve it point out that the antrum in an acid environment will not stimulate further acid production, that it secretes an alkaline mucus, that it might actually inhibit the over-production of acid, and with the pylorus is a very useful physiological mechanism for the control of gastric emptying.

It should be mentioned that the duodenum as well as the antrum undoubtedly exerts some control over acid secretion by parietal cells, and while this has not been as thoroughly explored as the antral mechanism, both stimulation and inhibition might occur. The most important contribution in this regard has been recently made by Dragstedt *et al.*,[28] who demonstrated that secretion not only stimulates the production of pancreatic secretion but also inhibits the parietal cell secretion of acid. It is quite possible, of course, that these inhibitory mechanisms which normally control the production of acid are no longer effective in patients with peptic ulcer, and this may be the explanation for their ulcer disease. There is no evidence that this thesis is correct, but it is worth consideration.

Possible interrelationships between the vagal, antral and intestinal phases are at present receiving a great deal of study. It is by no means clear how significant these interrelationships may be, or whether they exist as a physiological mechanism. It is important, however, to recognize that one phase may have a significant influence on remaining phases, and this might be modified by the presence or absence of vagal innervation.

After loss of one source of gastric stimulation, is there a tendency for the remaining mechanisms to become overactive and so restore acid secretion to the original level? There has been some suggestion that vagotomy in dogs reduces acid secretion only temporarily, but these experiments were done at a time when the vagotomy might have been incomplete.[29] Dragstedt has followed up dogs with vagally innervated pouches for a five year period after removal of the antrum, and there has not been any tendency for pouch secretion to increase with the passage of time. One interesting observation reported by both Dragstedt[30] and Harkins[31] has been that the acid output of a gastric pouch increased markedly after vagotomy of the main pouch. This has been termed "the paradoxical effect of vagotomy", and its mechanism appears to be due to the lowering of gastric acidity in the region of the antrum by vagotomy. Certainly this effect is secondary to hyperfunction of the antrum, for it does not occur if the antrum has been previously removed. Whether or not this antral hyperfunction can be eliminated by gastroenterostomy is still debatable.

There is no evidence that the release of gastrin potentiates secretion of vagal origin, but there is some evidence that vagal impulses can result in gastrin production by the antrum. Uvnas[18] and more recently Dragstedt[32] have demonstrated that vagal stimulation of the innervated antrum does result in gastrin production, probably secondary to antral peristalsis. No evidence of this interrelationship was found by Gouws,[27] and it is possible that the secretion which resulted was via the intestinal phase, rather than antral in origin.

It is well established that vagal innervation is not necessary for gastrin production under experimental conditions, but the latter might be greater under physiological conditions if the antrum was innervated.

The evidence that active inhibition of acid production by the antrum is more effective if the antrum is vagally innervated has been referred to previously. This question is by no means settled, but it has an important bearing on the theoretical differences between the Wangensteen I (segmental) and the Wangensteen II (tubular). Wangensteen first utilized the segmental type of resection to reduce the parietal cell mass, and later developed tubular resection, which accomplishes the same purpose without division of the vagi. Technically, segmental is a pie-shaped resection proximal to the antrum, which includes a short segment of the lesser and a long segment of the greater curvature. The vagus branches to the antrum and the remainder of the gastrointestinal tract are divided. In a

tubular type the lesser curve with the vagal branches is preserved. In practice he has found a higher ulcer recurrence rate following tubular, but this is technically a more difficult operation and the extent of resection might have been less in this series.

The source of gastrin and an inhibitor substance, if present, has not been determined, nor has the mechanism for its release been confidently defined. There is considerable evidence that these substances might arise deep to the mucous membrane, because the response to the stimulation of acid production by distension and by the presence of food in the antrum can be blocked by topical application either of cocaine or of atropine. The experiments of Uvnas suggest that these topical agents will also block secretion of vagal origin, but Dragstedt was unable to confirm this. Both Dragstedt[33] and Harkins[34, 35] have conducted experiments in which the superficial and deep intrinsic nerve plexuses of the antral wall have been interfered with, with results that are not yet conclusive. The results of their experiments can be interpreted in a number of ways, one of which is the suggestion that gastrin is produced in one portion of the antrum and the inhibitor substance in a second area. From a practical point of view, it is possible that we will be able to retain the antrum surgically and destroy its gastrin-producing potential without interfering with its ability to inhibit gastric acid production. The practical application of these possibilities is still very much in the future, and may not materialize.

This brief summary of antral physiology gives some indication of its present complexities and unanswered questions. As is so often the case the mechanism has been shown to be complex rather than simple. From the viewpoint of the surgeon operating on duodenal ulcer, the antrum might be worth retaining as a useful physiological mechanism for the control of acid secretion. It cannot be stated at this time, however, that the antrum should be retained, because it is not known whether these mechanisms exist in the human and at what pH they operate. The answers to these questions will undoubtedly be forthcoming within the next decade, and until then the surgical procedures for duodenal ulcer used at present should not be discontinued. On one point we can be quite dogmatic—a procedure by which the antrum is left in contact with alkaline secretion, thus markedly stimulating gastric and acid production, should never be used.

Summary

This paper is a summary of the present status of knowledge about the antrum; further consolidation of the material is not practicable.

References

1. Pavlov, I. P.: Lectures on the work of the digestive glands, translated by W. H. Thompson, Charles Griffin & Co., London, 1902.
2. Edkins, J. S.: The chemical mechanism of gastric secretion, *J. Physiol.*, **34:** 133, 1906.
3. Pavlov, I. P.: Lectures on the work of the digestive glands, translated by W. H. Thompson, Charles Griffin & Co., London, 2nd ed., 1910.
4. Keeton, R. W. and Koch, F. C.: The distribution of gastrin in the body, *Am. J. Physiol.*, **37:** 481, 1915.
5. Lim, R. K. S.: The question of a gastric hormone, *Quart. J. Exper. Physiol.*, **13:** 78, 1922.
6. Wilhelmj, C. M., O'Brien, F. T. and Hill, F. C.: The influence of the pylorus on the secretion of acid by the fundus, *Am. J. Physiol.*, **116:** 685, 1936.
7. Priestley, J. T. and Mann, F. C.: Gastric acidity with special reference to the pars pylorica, *Arch. Surg.*, **25:** 395, 1932.
8. Ivy, A. C. and Whitlow, J. E.: The gastrin theory put to physiological test, *Am. J. Physiol.*, **60:** 578, 1922.
9. Ivy, A. C. and Farrel, J. I.: The proof of a humoral mechanism, *Am. J. Physiol.*, **74:** 639, 1925.
10. Finsterer, H. and Cunha, F.: The surgical treatment of duodenal ulcer, *Surg. Gynec. & Obst.*, **52:** 1099, 1931.
11. Devine, H. B.: Basic principles and supreme difficulties in gastric surgery, *Surg. Gynec. & Obst.*, **40:** 1, 1925.
12. Ogilvie, W. H.: The approach to gastric surgery, *Lancet*, **2:** 295, 1938.
13. Graham, R. R.: Technical surgical procedures for gastric and duodenal ulcer, *Surg. Gynec. & Obst.*, **66:** 269, 1938.
14. Wilhelmj, C. M., O'Brien, F. T. and Hill, F. C.: The inhibitory influence of acidity of the gastric contents on the secretion of acid by the stomach, *Am. J. Physiol.*, **115:** 429, 1936.

15. Dragstedt, L. R. *et al.*: Interrelation between the cephalic and gastric phases of gastric secretion, *Am. J. Physiol.*, **171:** 7, 1952.

16. Harrison, R. C., Lakey, W. H. and Hyde, H. A.: The production of an acid inhibitor by the gastric antrum, *Ann. Surg.*, **144:** 441, 1956.

17. Dragstedt, L. R. *et al.*: Effect of transplantation of antrum of stomach on gastric secretion in experimental animals, *Am. J. Physiol.*, **165:** 386, 1951.

18. Uvnas, B. *et al.*: The influence of exclusion of the antrum-duodenum passage on the HCl secretion in Pavlov pouch dogs, *Gastroenterology*, **30:** 790, 1956.

19. Ragins, H. *et al.*: Studies on the physiology of the gastric antrum *A.M.A. Arch. Surg.*, **75:** 230, 1957.

20. Woodward, E. R. *et al.*: Does the gastric antrum elaborate on antisecretory hormone? *Am. J. Digest. Dis.*, **3:** 204, 1958.

21. Margolus, B. D. and Harrison, R. C.: Inhibition of the intestinal phase of gastric secretion by antral inhibitor substance, *Surg. Forum*, **7:** 360, 1957.

22. Jordan, P. H. and Sand, B. F.: A study of the gastric antrum as an inhibitor of gastric juice production, *Surgery*, **42:** 40, 1957

23. Jones, T. W. *et al.*: Heidenhain pouch secretory response to transplantation of the innervated and denervated antral pouch to the transverse colon, *Ann. Surg.*, **147:** 13, 1958.

24. Longhi, E. H. *et al.*: Question of an inhibitory hormone from the gastric antrum, *Am. J. Physiol.*, **191:** 64, 1957.

25. Shimizu, H. J., Morrison, R. T. and Harrison, R. C.: Inhibition of vagally stimulated gastric acid by the pyloric antrum, *Am. J. Physiol.*, **194:** 531, 1958.

26. Woodward, E. R. *et al.*: The physiology of the gastric antrum, *Gastroenterology*, **27:** 766, 1954.

27. Gouws, F. and Harrison, R. C.: The influence of the vagus nerve on antral function, *Canad. J. Surg.*, **1:** 337, 1958.

28. Greenlee, H. B. *et al.*: Inhibitory effect of pancreatic secretion on gastric secretion, *Am. J. Physiol.*, **190:** 398, 1957.

29. Vanzant, F. R.: Late effects of section of the vagus nerves on gastric acidity, *Am. J. Physiol.*, **99:** 375, 1931.

30. Evans, S. O. Jr. *et al.*: Stimulating effect of vagotomy on gastric secretion in Heidenhain pouch dogs, *Am. J. Physiol.*, **174:** 219, 1953.

31. Schmitz, E. J. *et al.*: The effect of vagotomy of the main stomach on Heidenhain pouch secretion, *Surg. Forum*, **2:** 17, 1952.

32. Oberhelman, H. A., Rigler, S. P. and Dragstedt, L. R.: Significance of innervation in the function of the gastric antrum, *Am. J. Physiol.*, **190:** 391, 1957.

33. Baugh, C. M. *et al.*: Studies on the site and mechanism of gastrin release, *A.M.A. Arch. Surg.*, **76:** 441, 1958.

34. Jones, T. W. *et al.*: The effect of antroneurolysis upon antral function of the stomach, *Surg. Gynec. & Obst.*, **105:** 687, 1957.

35. DeVito, R. V. *et al.*: Modification of gastrin mechanism by antroneurolysis, *Surg. Forum*, 1958, in press.

Résumé

L'auteur nous offre une synthèse des travaux les plus récents sur la physiologie de l'antre gastrique. Dans l'état présent de la question plusieurs problèmes demeurent encore insolubles, cependant il ressort de cette accumulation de données expérimentales et cliniques que la région antrale sécrète une humeur, la gastrine, qui provoque la sécrétion d'acide par les cellules pariétales. Le fait a été constaté et définitivement prouvé, mais puisque cette substance n'a jamais été isolée ou identifiée, on ne peut encore parler d'hormone mais seulement l' "agent humoral". L'antre comprend 25% de la masse gastrique et sécrète un mucus alcalin. Il est stimulé par sa distension ou par le contact avec sa muqueuse d'aliments relativement alcalins. L'instillation d'une substance acide dans l'estomac tend à retarder la sécrétion acide de l'estomac au lieu de la stimuler. Lorsque l'antre est greffé sur le côlon ou sur le duodénum on observe une forte stimulation de la sécrétion de la poche gastrique.

L'existence d'un inhibiteur provenant de l'antre semble probable sans toutefois avoir été démontrée. Cette substance dépendrait, pour sa sécrétion, de l'intégrité des fibres antrales du vague, mais la question n'a pas encore reçu de réponse définitive. Au point de vue chirurgical les opinions sont divisées entre ceux qui enlèvent l'antre en croyant qu'il est avant tout un mécanisme de stimulation de sucs gastriques acides, et ceux qui le laissent en place sous prétexte que dans un milieu acide il ne stimulera pas davantage la sécrétion d'acide, mais qu'au contraire il sécrète un mucus alcalin, peut freiner la surproduction de sucs acides et seconder le pylore de manière physiologique dans l'évacuation gastrique.

CASE REPORTS

CHONDROSARCOME DU BRAS GUERISON (?) APRES EVOLUTION DE 17 ANS

ARMAND GENEST, M.D., D.D.S.,* et ROGER GAREAU, M.D.,† *Montréal*

MME L.C., 64 ans, nous est référée pour une tumeur du bras droit, évoluant depuis 17 ans. La masse est énorme, plus grosse qu'une tête adulte (Fig. 1).

La patiente raconte l'histoire suivante: il y a 17 ans, lors d'un accouchement, elle reçut une injection près de l'épaule. Un petit nodule persista, qui grossit lentement au cours des années suivantes. Cinq ans plus tard, cette masse l'inquiétant, elle consulta un médecin qui la dirigea dans un hôpital de la métropole. Durant cette hospitalisation, des radiographies du bras (Fig. 2) et du thorax ainsi qu'une biopsie établirent un diagnostic certain de chondrosarcome. Avec insistance, la désarticulation du membre fut proposée à la patiente, alors âgée de 52 ans. On lui aurait même représenté qu'elle ne survivrait pas au-delà de six mois sans cette intervention. Ne pouvant parvenir à se résigner, la patiente refusa de se soumettre à la chirurgie et quitta l'hôpital.

C'est 12 ans plus tard que la patiente nous arrive. Deux faits saillants nous frappent: un très bon état général et une énorme masse au bras droit. La tumeur est toute latérale et semble pendre de l'épaule. Elle présente un pôle inférieur arrondi et ulcéré où trois hémorragies importantes sont survenues récemment. La masse est si encombrante que le membre est devenu impotent. Des cultures prélevées dans l'immense cratère nécrotique et nauséabond ont démontré la présence de trois organismes redoutables: un entérocoque, un *Pseudomonas æruginosa* et un *Klebsiella pneumoniae*. Quelques biopsies faites au même site ont finalement permis un histodiagnostic de tumeur maligne d'origine mésenchymateuse. De nombreux clichés radiographiques du bras, sous diverses incidences, ont révélé une néoformation arborescente, à point de départ huméral et de densité comparable à celle du cartilage, permettant d'affirmer qu'il s'agissait d'un chondrosarcome. Les films pulmonaire et médiastinal étaient normaux.

Pour les raisons énoncées plus loin, il nous semblait possible d'enlever cette tumeur en conservant le membre intact. Cependant il était convenable de chercher l'avis des orthopédistes plus versés dans ce chapitre de la pathologie. Leur opinion ne laissait aucune alternative et répétait celle énoncée ailleurs 12 ans auparavant. Devant ce problème il nous restait le seul espoir de présenter le cas à la Clinique des Tumeurs où la discussion fut ardue. D'un côté les orthopédistes dirigeaient les partisans de la désarticulation et de l'autre, nous présentions nos arguments en faveur de l'exérèse locale radicale avec préservation du membre et de ses fonctions. Notre jugement chirurgical reposait sur les motifs suivants: la durée de l'évolution en face d'un bon état général; l'absence de métastases cliniques et radiologiques, et la conservation intacte de la motricité et de la sensibilité jusqu'au bout des doigts. Il nous a paru indéniable qu'une tumeur aussi volumineuse, évoluant depuis tant d'années, ne pouvait pas être du type infiltrant; mais bien au contraire, nous pensions voir une tumeur qui avait grossi lentement en refoulant les structures environnantes. Par conséquent, cette néoplasie devait être encapsulée, ce qui expliquait que les muscles, les nerfs et les vaisseaux du membre étaient demeurés indemnes. Il nous semblait donc possible de la circonscrire chirurgicalement sans léser les structures vitales du membre.

L'exérèse radicale locale fut pratiquée en mars 1957. Nous nous proposions une hémi-section longitudinale de l'humérus, mais au premier trait d'ostéotome, la mince diaphyse cassa d'un travers à l'autre. L'humérus fut donc réséqué complètement sur une longueur de 8 cm., en continuité avec la masse tumorale. Il importe de noter que cette résection osseuse fut entière, y compris le périoste. D'ailleurs, aucun décolleur ne fut employé.

Confiant dans notre routine de préparation cutanée par une émulsion d'hexachlorophène en applications répétées (l'ulcère ayant été scellé au collodion la veille de l'opération), nous n'avons pas craint d'utiliser une plaque de vitallium pour conserver l'espace huméral, en attendant une greffe osseuse élective quelques mois plus tard. La plus longue plaque métallique à notre disposition mesurait 15 cm. Après enfoncement d'une extrémité de la prothèse dans la tête humérale, il fut possible de fixer l'extrémité distale par une seule vis. La résection radicale de cette masse nous avait laissé devant

*Chef, section de Chirurgie Plastique, Hôtel-Dieu de Montréal.
†Pathologiste, Hôtel-Dieu de Montréal.

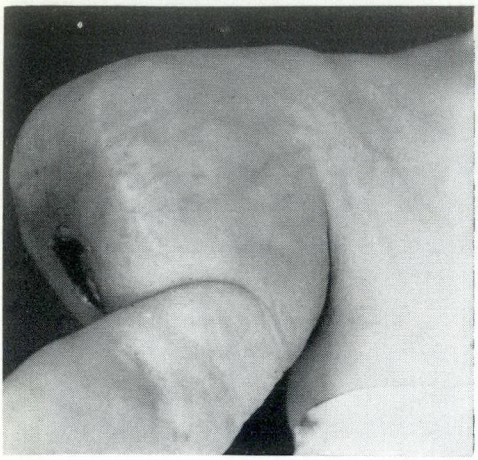


Fig. 1

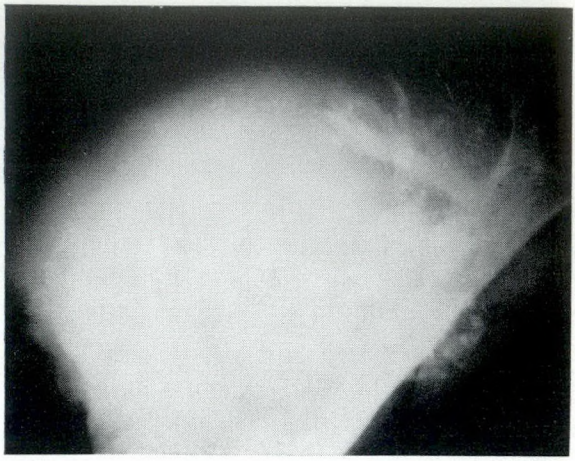


Fig. 2

un important déficit cutané. Ce fut l'occasion unique d'exonérer la chirurgie plastique d'accaparement ambitieux. Heureusement, notre métier de tailleur cutané réussit à perfection: guérison *per primam*. L'immobilisation plâtrée ne fut pas employée. La patiente, explicitement avisée de ne pas utiliser son bras, retourna chez-elle avec une écharpe de Maillard.

Six mois plus tard, pour des raisons incontrôlables, la greffe osseuse n'avait pas encore été pratiquée. La patiente fut convoquée pour examen et ce fut une surprise de constater une bonne fonction de son membre. Elle avoua avoir graduellement fait usage de son bras au point de s'en servir comme d'un bras normal. Des radiographies de contrôle montrèrent une évolution renversante des fragments de l'humérus. La régénération osseuse (Fig. 3) s'était faite le long de la plaque de vitallium à partir des deux fragments huméraux séparés par un espace de 8 cm. Le dernier contrôle radiologique du bras et des poumons a été pratiqué huit mois après l'intervention. Notre patiente se porte toujours aussi bien (Fig. 4) deux ans après le traitement chirurgical.

Anatomie pathologique

Le spécimen réséqué pèse 3850 g. A son pôle inférieur, on remarque une zone nécrotique de 14 cm. de diamètre en surface et de 4 cm.

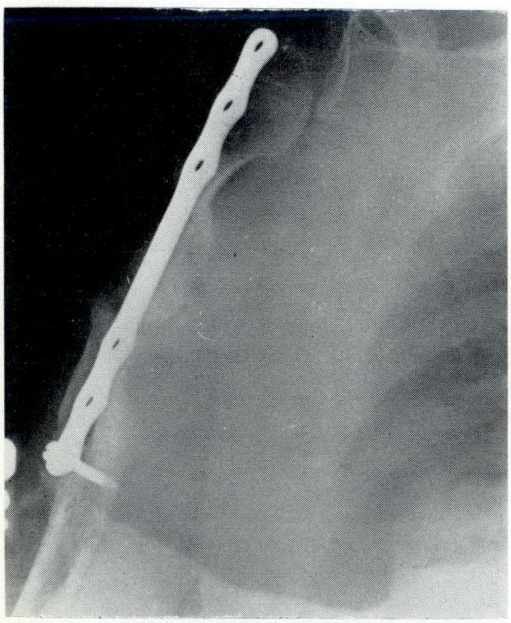


Fig. 3

Fig. 4

de profondeur. En avant, en arrière et du côté externe, la pièce est recouverte par la peau. Du côté interne, elle est limitée en partie par la ligne de résection chirurgicale et en partie par la peau. Une coupe longitudinale passant par la zone ulcérée montre la tumeur qui se compose de trois nodules superposés dont les deux supérieurs, bien délimités sur une certaine partie de leur circonférence, mesurent chacun 6 cm. de diamètre (Fig 5). Ils confluent par certains points et donnent naissance au troisième, plus large, mal délimité situé à la partie distale de la tumeur, en grande partie nécrotique et qui s'ulcère à la peau.

L'humérus mesure 8 cm. de longueur. La tumeur s'ossifie à son contact tout en ménageant entre les travées ossifiées des logettes remplies de substance colloïde. Ces logettes se retrouvent sur toute la surface de section de la tumeur. Elles sont habituellement creusées dans du tissu mou.

Au microscope, la tumeur se compose essentiellement de tissu cartilagineux. D'abord jeune et myxomateux, il comporte une substance fondamentale plus ou moins liquide muci-carminophile dans laquelle nagent des cellules mésenchymateuses étoilées. Par la suite, ces cellules s'arrondissent, s'entourent d'un espace clair, typique des chondrocytes, pendant que la substance fondamentale se transforme en chondrine colorable par le safran (Fig. 6). Le cartilage néoformé est découpé en îlots par des travées conjonctives vascularisées qui font office de périchondre, dont les fibroblastes atypiques se multiplient par mitose et donnent à certaines régions de la tumeur un aspect fibrosarcomateux. Aux points de tangence de la masse tumorale avec l'humérus, le périoste réagit, se ramifie et va ossifier le cartilage tumoral. A ce niveau, la moëlle humérale prend un aspect cicatriciel. Elle est découpée par des travées d'os spongieux reliées à l'os lamellaire de la corticale osseuse.

Considérée du point de vue histologique, cette tumeur remplit les critères de Lichtenstein[2] pour les chondrosarcomes et qui sont: (1) la présence de plusieurs cellules à gros noyaux; (2) la présence de cellules binucléées à gros noyaux; (3) la présence de cellules cartilagineuses géantes, mononucléées à gros noyaux multinucléées à noyaux chargés de chromatine grossière.

Un diagnostic de chondrosarcome a donc été porté sur cette tumeur.

Discussion Anatomo-pathologique

Il ressort de l'examen macroscopique et microscopique de cette tumeur qu'elle est de nature cartilagineuse. Elle possède des

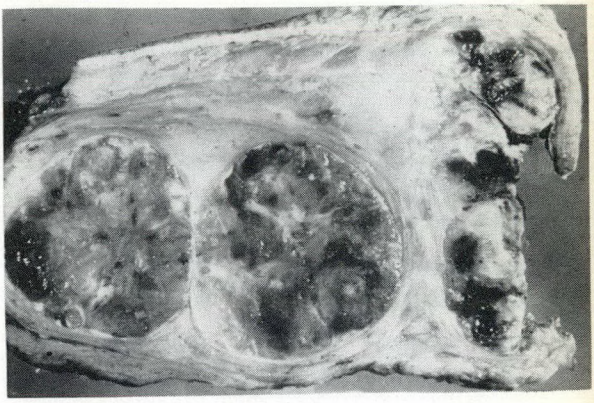


Fig. 5

critères de malignité—noyaux irréguliers, volumineux, cellules binucléées, zones de nécrose, etc,—qui la font classer parmi les chondrosarcomes. Les professeurs Pierre Masson[3] et Arthur Purdy Stout[5] à qui les coupes ont été montrées sont d'ailleurs d'accord sur ce point. Composée de tissu myxomateux qui se différencie graduellement en cartilage pour se calcifier ensuite et parfois s'ossifier, elle ressemble par certains points au mésenchymome malin. Il lui manque cependant une autre composante néoplasique maligne d'origine mésenchymateuse: léiomyosarcome, rhabdomyosarcome, réticulosarcome ou autre,[4] pour motiver le diagnostic de mésenchymome malin, car il y faut au moins deux tissus cancéreux d'origine mésenchymateuse, exception faite du tissu fibrosarcomateux.

Quant au point de départ de ce chondrosarcome, il est difficile à établir. L'humérus doit être éliminé comme lieu d'origine car si cela était, il serait considérablement dé-

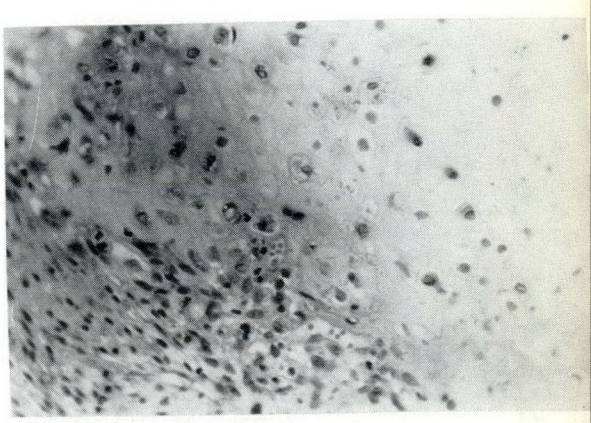


Fig. 6

formé et partiellement détruit. Les radiographies nous prouvent que le profil osseux est assez bien conservé en dépit de la réaction périostée.

Le chondrosarcome des tissus mous est très rare surtout au membre supérieur.[1] Il se développe généralement dans le muscle près d'une articulation qu'il peut envahir par la suite. Sa croissance est généralement rapide. Sauf exception, il n'envahit pas l'os. Il peut récidiver après exérèse chirurgicale mais n'a pas tendance à donner de métastases. C'est peut-être dans cette catégorie que se place cette tumeur mais il faut admettre que l'évolution ne semble pas favoriser cette hypothèse.

Le chondrosarcome dévelopé sur exostose ostéo-cartilagineuse est rare.[3] Il croît lentement et peut atteindre des dimensions énormes sans donner de métastases. Après exérèse chirurgicale, il peut récidiver localement mais n'a pas tendance à donner de métastases. De plus, la cancérisation du revêtement cartilagineux de l'extrémité distale des exostoses se fait à l'âge adulte parfois avancé. Cette hypothèse semble donc valable pour expliquer l'origine de ce chondrosarcome. Malheureusement, aucune radiographie humérale antérieure au développement de la tumeur ne vient confirmer la présence d'une exostose dans la région.

Résumé

Nous avons rapporté un cas de chondrosarcome huméral dont les débuts remontent à 17 ans. Après cinq années d'évolution, la patiente fut hospitalisée ailleurs et un diagnostic précis avait été posé. La patiente refusa alors de subir la désarticulation du membre et quitta l'hôpital. Après 12 ans, son état général est très bon mais la tumeur est devenue énorme. Elle est réséquée localement et radicalement y compris 8 cm. de l'humérus avec périoste. Une plaque de vitallium est utilisée dans un but temporaire pour conserver la longueur humérale. Une greffe osseuse était projetée mais ne fut pas pratiquée. La régénération humérale a été trouvée pour le moins suprenante, ayant réalisé la continuité osseuse tout le long de la plaque métallique. La fonction du bras est normale et l'état général aussi bon deux ans après l'intervention.

Bibliographie

1. Jaffe, H. L.: Tumors and tumorous conditions of the bones and joints, Lea & Febiger, Philadelphia, 1958.
2. Lichtenstein, L.: Bone tumors, C. V. Mosby Company, St. Louis, Mo., 1952.
3. Masson, P.: Communication personnelle.
4. Stout, A. P.: Tumors of the soft tissues. Atlas of Tumor Pathology, Sec. 2, Fasc. 5, Armed Forces Institute of Pathology, Washington, D.C., 1953.
5. Stout, A. P.: Communication personnelle.

Summary

We have reported a case of chondrosarcoma of the humerus. A 64 year old woman presented with a large tumour mass of the right shoulder. A small lump had first been noticed 17 years before. A diagnosis of chondrosarcoma was first established 12 years ago when the patient was hospitalized elsewhere, but she had refused treatment and left the hospital. In March 1957, she was referred to us with a huge tumour mass of the right shoulder. Her general condition was excellent.

The case was discussed before the Tumour Clinic of Hôtel-Dieu hospital. The orthopædists favoured disarticulation of the limb, while we proposed a local radical resection for the following reasons: the patient's general condition had been good while the tumour had been growing for 17 years; there were no demonstrable metastases; and the patient had normal function and sensory perception down to the fingertips.

The mass weighing nearly 10 lb. was resected along with overlying skin, deltoid muscle and 8 cm. of humerus, including periosteum. A vitallium bar was inserted as a space retainer, in the expectation that a bone graft would be necessary in a few months. Some six months later, as the patient had not reported back, she was reached for follow-up examination. It was astonishing to find that she was using her limb as normally as anybody. Radiographs showed complete regeneration of bone along the metallic plaque. Her general condition, two years postoperatively, is still as good as ever.

ABDOMINAL ACTINOMYCOSIS—REPORT OF AN UNUSUAL CASE AND REVIEW OF LITERATURE ON ANTIBIOTIC THERAPY*

G. R. DAVIES, M.D., and R. B. LYNN, F.R.C.S.,† *Saskatoon, Sask.*

ABDOMINAL ACTINOMYCOSIS usually occurs in relation to the ileocæcal region. The fungus is frequently an inhabitant of the mouth and fæces of healthy individuals, and an actinomycotic abscess develops in the peritoneal cavity only after some breach of continuity of the bowel wall permits egress of the organism. It is generally conceded that the most usual source is the acutely inflamed or perforated appendix. Armitage and Smith[1] found this to be so in only two of their nine patients; however, Putman and his colleagues[4] out of 122 case reports found the relationship to exist in 88. In 15 patients, abdominal actinomycosis complicated a variety of perforating lesions throughout the gastrointestinal tract, only one of which was the result of perforation by spicules of bone. This abscess involved the spleen and gastrosplenic ligament long after the bone had passed through the stomach wall. In our patient, a paracolic abscess developed in relation to the descending colon after perforation by a 1.5 cm. x 1 mm. spicule of long bone. All too frequently, at the time of diagnosis, the disease has advanced to the stage of multiloculated abscess with internal and external fistulæ. Our patient was fortunate in this regard but there were indications at operation that the transverse colon, at least, would soon have been drawn into the process.

The preoperative diagnosis is often that of malignancy; this occurred in eight patients in Putman's series, and on each occasion the correct diagnosis could only be obtained by biopsy. A malignant tumour was a distinct possibility in our patient preoperatively, and the gross appearance at operation was even more suggestive. The finding of a liver nodule bearing some resemblance to a metastasis inspired only pessimism until this nodule was excised and submitted to frozen section. It was found to be a hyperplastic liver nodule.

Sherry, Macdonald and Anderson[6] have recently reported two cases in which a solitary liver nodule obscured the true situation. Such a nodule, variously known as a solitary liver nodule, solitary hyperplastic nodule, focal cirrhosis, adenoma, benign hepatoma and hamartoma, is most commonly solitary, although rarely two or three may be found. They vary from a few millimetres to several centimetres in diameter. They do not possess a true capsule yet they are clearly demarcated from the surrounding liver tissue. Happily, they often occur on the free edge of the liver, a feature which facilitates total excision. These authors point out that, in a case of cancer, to mistake one of these nodules for a metastasis may discourage the surgeon from performing a wide bowel resection for cure. Removal is further justified on the grounds that such a nodule may be premalignant.

CASE REPORT

The patient, the 41 year old wife of a Saskatchewan farmer, complained of soreness in the left flank of several weeks' duration. The discomfort was aggravated by forward bending, straining, coughing, and prolonged or rough palpation. There was no radiation of the pain and it was impossible to elicit a history relating to food intake, bowel movement or urinary habits. Her menstrual history was entirely normal. She had lost 4 lb. in the week before admission, when anxiety over her condition had resulted in profound anorexia. Prior to this, however, she had not lost weight and her appetite had been normal. The remainder of the functional enquiry was non-contributory.

The patient was a short, slender woman, weighing only 84½ lb. Colour, nutrition and hydration were satisfactory. Blood pressure, pulse and temperature were normal. There was no regional lymphadenopathy. The head and neck were normal, as were the breasts, the heart and the lungs. There was a spherical mass in the left flank at the level of the umbilicus. It was about 6 cm. in diameter and seemed to be

*From the Department of Surgery, University Hospital, Saskatoon, Saskatchewan.

†Present address—Department of Surgery, Kingston General Hospital, Kingston, Ontario.

Fig. 1.—The pericolic mass has been bisected revealing the serosal surface of the resected segment of descending colon. The spicule of bone has been removed from the centrally placed abscess cavity.

slightly mobile over the posterior abdominal wall structures. It did not move with respiration. There were no other abnormalities in the abdomen, and pelvic and rectal examinations were negative.

The hæmoglobin value was 12.3 grams per 100 ml.; white blood cell count 10,000 with a normal differential; sedimentation rate 85 mm. in 1 hour. Urine, non-protein nitrogen and fasting blood sugar were normal. Chest radiograph, and kidney, ureter and bladder by intravenous pyelogram, were normal. The palpable mass could not be visualized in any of these soft tissue films. A barium enema showed spasm of the mid-descending colon in the region of the palpable mass. There was a very short segment here which did not fill well, but the mucosa appeared to be intact. The appearances were attributed to spasm or extraluminal pressure with displacement.

Laparotomy was performed through a left paramedian incision. A mass the size of a tangerine orange was found binding the descending colon to the muscles of the left flank. The greater omentum and the transverse colon were drawn to the area, although the transverse colon was not directly involved by the disease process. When these structures had been displaced, the mass was found to be kinking and distorting the descending colon. At this point the remainder of the abdomen was explored, whereupon a nodule about 2 cm. in diameter was found on the free edge of the liver near the midline. It was at first thought to be a metastasis, but certain features militated against this. It had a reddish hue and a rather multilobulated configuration. It was softer than a metastasis and was not umbilicated. Because of its size and its location on the free edge, it was excised *in toto*. Frozen sections showed it to be a benign liver nodule. A wedge was removed from the mass in the flank and this too was reported as benign. Consequently the mass and a segment of descending colon were resected. The specimen was examined by the pathologist and found to be a thick-walled abscess, intimately associated with the colon but without any apparent communication with its lumen (Fig. 1). Within the abscess cavity was a bony spicule measuring 1.5 cm. by 1 mm. (Fig. 2). Bowel continuity was re-established by end-to-end anastomosis and the abdomen was closed.

The patient's postoperative course was a difficult one. The usual postoperative ileus was slow in resolving, and when finally there was evidence of return of peristalsis she continued to have recurrent distension, nausea and vomiting. This was treated conservatively by naso-gastric suction and intravenous therapy. By the 15th postoperative day it was evident that she had a subacute bowel obstruction, which was not going to resolve with such conservative measures. She was therefore operated on for a second

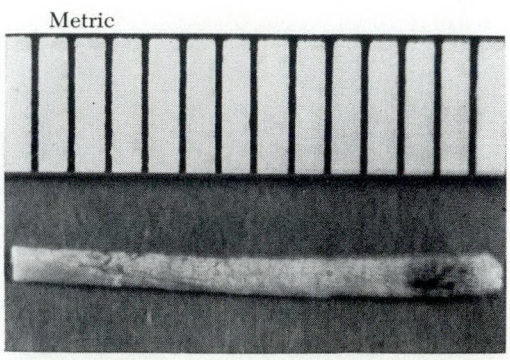


Fig. 2.—Bone found within abscess cavity.

time. At this exploration, surprisingly dense adhesions binding the small bowel to the incision and to the stab wound in the left flank were found, as well as many adhesions binding loops of small bowel together throughout almost its entire length. There was no evidence of an abscess or peritonitis. Recovery from the second operation was satisfactory.

The excised hepatic nodule was composed of proliferating hepatic tissue in which lobular architecture, although distorted, was fairly well preserved (Figs. 3a and b).

The pericolic mass consisted of a dense zone of subacute and chronic suppurative and granulomatous inflammation. Dense fibrous tissue involved the pericolic fat and surrounded zones of necrosis, inflammation and suppuration. There was a heavy infiltration with polymorphonuclears, lymphocytes, plasma cells and macrophages. Small numbers of multinucleated giant cells of the foreign body type were also present. In several of these masses of inflammation, colonies of fungus were seen which had the appearance of actinomycosis. Special stains (Glynn, periodic-acid-Schiff, and Grocott) confirmed this and demonstrated narrow branching filaments together with club-shaped processes (Fig. 4). Pus from the abscess cavity was examined with

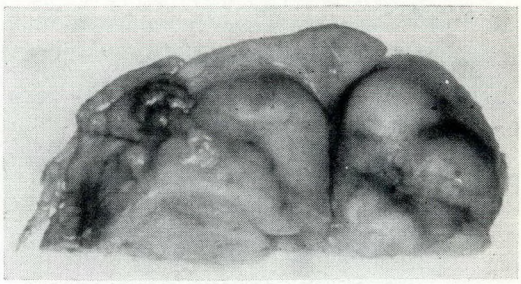


Fig. 3.—(a) Solitary liver nodule: serosal surface (x4).

Gram's stain. Gram-positive branching filaments were seen. Culture, however, failed to grow fungus after 30 days. It was assumed that this was an actinomycotic abscess due to perforation of the wall of the descending colon by the spicule of bone.

When this was discovered, the patient was given 5,000,000 units of crystalline penicillin daily. It was found most convenient to administer this in her intravenous infusion. A few days later, in order to afford her maximum protection, the dose was increased to 10,000,000 units per day. In this way she received 134.2 million units of penicillin over a period of 20

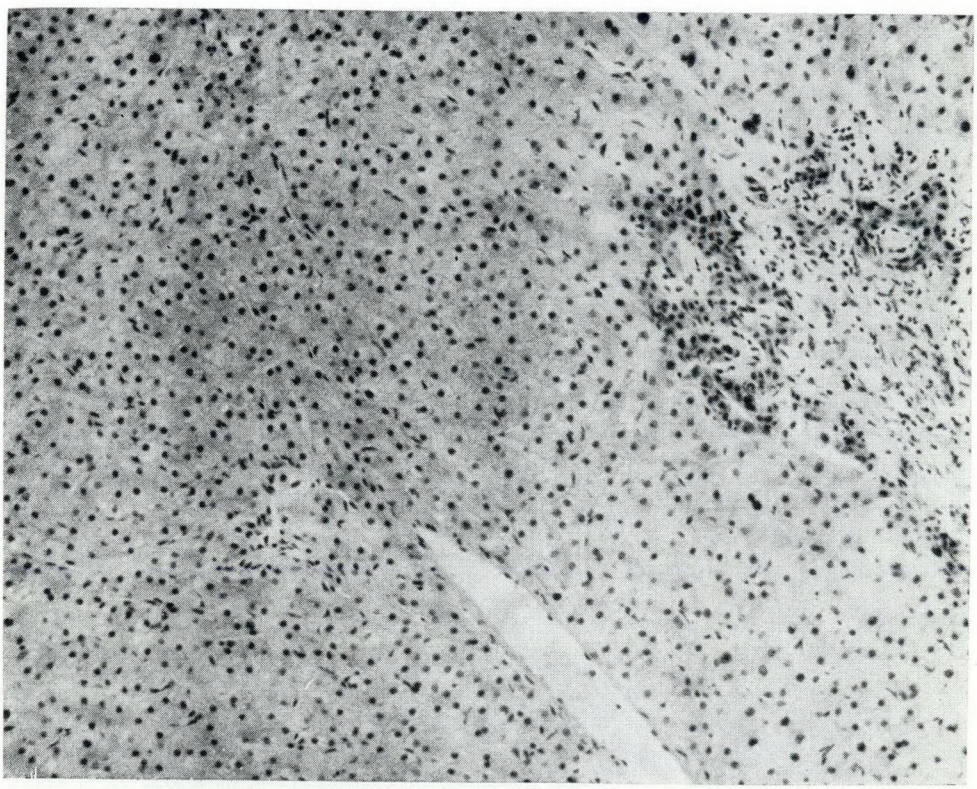


Fig. 3.—(b) Solitary liver nodule: microscopic appearance (x50).

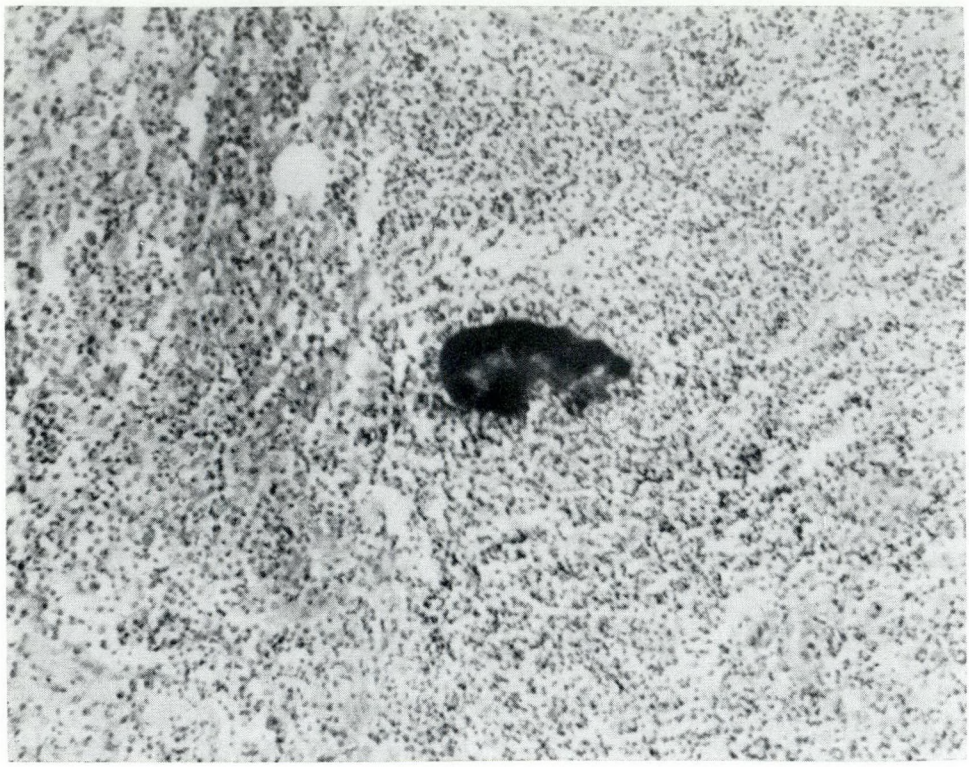


Fig. 4.—Microscopic appearance of abscess showing a colony of actinomyces (x50).

days. At this point the patient became utterly intolerant of needle therapy. She also insisted on returning home. For these reasons she was given aureomycin by mouth, 2 g. daily in divided doses initially, and after five days was discharged with a supply of this drug and instructed to take 1 g. daily in divided doses until a total of 39 g. of aureomycin had been received.

Eight weeks after her second operation the patient was asymptomatic and there were no signs of disease. Her appetite was normal and she had gained 5 lb. in weight. There had been no untoward effects from the aureomycin.

Review of Recent Literature on Antibiotic Therapy of Abdominal Actinomycosis

It is widely accepted that penicillin remains the antibiotic of choice in the treatment of actinomycosis. Its use has revolutionized the management of this disease and has produced improvement or cure in some of the most advanced and obstinate cases. Parenteral administration in the usually recommended dose, 6 to 8,000,000 units daily, presented problems in our patient which inspired us to review a few of the recent papers on the use of oral antibiotics of the broad spectrum type. None of these have yet received clinical study comparable to that afforded penicillin. There are indications, however, that some may be equally effective. Oral preparations of penicillin do not appear to have been investigated with regard to actinomycosis.

Putman, Dockerty and Waugh[4] felt that radiation, arsenicals, potassium iodide, gold salts, etc., had obtained 16% cure rate in 75 cases. The use of sulfonamides raised this to 38.7% in 13 cases. Penicillin in doses of 500,000 to 1,200,000 units daily for four to six weeks resulted in cure or improvement in 95.6% of 24 cases. These observers reviewed 35 case reports from the literature in which the patients had been treated with penicillin. Among this group, cure was obtained in 23, improvement in eight and failure in four instances. This represents a cure or improvement rate of 88.9%.

Conant and co-authors,[2] recommend 6 to 8,000,000 units penicillin daily together with sulfadiazine and potassium iodide.

Considering Putman's results, perhaps doses in excess of 1,000,000 units daily need be employed only in selected patients i.e. with extensive, long standing disease, or those exhibiting relative drug resistance. Garrod[3] has studied the influence of penicillin on many strains. Although initial *in vitro* resistance is rare, sensitivity may decrease in unsuccessfully treated cases. In such instances another drug may be preferable to increasing the dose of penicillin.

According to the results of Garrod's *in vitro* studies on 12 strains, terramycin would appear to be the drug of choice, in place of penicillin. Chloramphenicol (Chloromycetin) was effective in a slightly higher concentration, aureomycin in a higher concentration yet, and streptomycin was least effective. He points out, however, that while terramycin and chloramphenicol are both stable, aureomycin is quite unstable under the laboratory conditions necessary for the successful growth of actinomycosis. That these results are an inaccurate assessment of aureomycin is further suggested by the clinical successes reported. Seligman[5] reported one personal case and reviewed 11 others from the literature, all of which had been treated with aureomycin with outstanding results.

Streptomycin too may be more useful than the *in vitro* studies would indicate. Here again the environment which is optimal for the growth of actinomycosis is unsuitable for the activity of streptomycin. Armitage and Smith[1] report a patient who failed to respond to "adequate penicillin" until 14 g. of streptomycin had also been administered. Although it may not be the drug of choice to be used singly, streptomycin may be useful in combination with penicillin in clinically resistant cases.

Strauss and his colleagues[7] reported *in vitro* observations on three strains of *Actinomyces bovis*, and found marked variation in their sensitivity. They concluded that chloramphenicol was superior to penicillin. Next to these came streptomycin and lastly aureomycin. Garrod[3] points out that their methods may well invalidate some of their observations. Both of these authorities recommend *in vitro* studies as a guide to therapy. It is indeed regrettable that we were unable to obtain a culture and to determine its sensitivity in our case.

Of the broad-spectrum antibiotics, aureomycin appears to have received more attention clinically than either chloramphenicol or terramycin and has proven its worth. This, plus the fact that *in vitro* they are similar in their effectiveness, led us to choose aureomycin as an alternative to penicillin. This particular case is probably not a worthy test of aureomycin because the condition was diagnosed early, the mass extirpated, and furthermore, 134,000,000 units of penicillin had already been administered when the aureomycin was begun.

Judging by the dosages which appear in the literature, it is evident that each case must receive individual consideration. Seligman[5] suggests that abdominal actinomycosis will require a larger dose of aureomycin than will the cervicofacial variety. The extent of the disease and its response to previous therapy must be taken into account. Whatever antibiotic is chosen, it must be administered in doses larger than usual and be continued for 6 to 12 weeks. During this time the patient must be observed for signs of toxicity. If relapse occurs weeks or even months after an apparently successful course of therapy, one is justified in repeating the course using the same antibiotic. Cultures should be obtained whenever possible and the sensitivity determined. If the second course should produce unsatisfactory results, these sensitivity tests can serve to guide the clinician in his choice of another drug. Considering the seriousness and chronicity of actinomycosis, treatment with two or more antibiotics may be indicated in advanced or clinically resistant cases. Putman and co-workers[4] feel that iodides and irradiation are "probably outmoded".

Successful treatment frequently requires more than antibiotics. The patient's general condition may deteriorate in the course of this disease. Negative nitrogen balance, wasting and anæmia must be prevented or adequately treated using an appropriate diet, vitamins and even intravenous infusions of whole blood. Finally, the surgeon has not been disenfranchised by the anti-

biotics, and judicious surgery still plays an important role in the management of abdominal actinomycosis.

Summary

An early case of abdominal actinomycosis has been described. It presented several unusual features, namely:

1. The location of the abscess in relation to the descending colon.

2. The fact that it occurred as a result of perforation of the colon by a spicule of bone.

3. The similarity of the lesion to a malignant tumour.

4. The presence of a solitary liver nodule (focal cirrhosis) resembling a metastasis.

Hyperplastic liver nodule is commented on briefly, and references are made to two similar reports in the literature. Solitary liver nodules should be excised and examined by frozen section or a benign nodule may be mistaken for a metastasis.

The antibiotic therapy of abdominal actinomycosis is discussed, and a limited review of recent literature concerning broad spectrum drugs is offered.

References

1. Armitage, G. and Smith, I.: Abdominal actinomycosis: case report and review of antibiotic treatment, *Brit. J. Surg.*, **42**: 77, 1954.
2. Conant, N. F. *et al.*: Manual of clinical mycology, 2nd ed., W. B. Saunders Company, 1954.
3. Garrod, L. P.: Sensitivity of *Actinomyces israeli* to antibiotics, *Brit. M. J.*, **1**: 1263, 1952.
4. Putman, H. C. Jr., Dockerty, M. B. and Waugh, J. M.: Abdominal actinomycosis: analysis of 122 cases, *Surgery*, **28**: 781, 1950.
5. Seligman, S. A.: Treatment of actinomycosis with aureomycin, *Brit M. J.*, **1**: 1421, 1954.
6. Sherry, J. B., Macdonald, I. B. and Anderson, W.: Solitary liver nodules (focal cirrhosis): two case reports, *Brit J. Surg.*, **45**: 581, 1958.
7. Strauss, R. E., Kligman, A. M. and Pillsbury, D. M.: Chemotherapy of actinomycosis and nocardiosis, *Am. Rev. Tuberc.*, **63**: 441, 1951.

Résumé

L'actinomycose abdominale se rencontre généralement dans la région iléo-cæcale. Le champignon est un hôte fréquent de la cavité buccale et des selles chez les individus normaux; son passage dans la cavité péritonéale nécessite une perforation de la paroi intestinale. Un abcès apparaît alors, qui peut donner naissance à des fistules externes et internes des plus compliquées. Du point de vue diagnostic, l'affection est souvent confondue avec une tumeur maligne, d'autant plus que des localisations hépatiques ne sont pas rares.

Les auteurs rapportent un cas qui leur est personnel. Il s'agit d'une malade de 41 ans souffrant depuis plusieurs semaines de douleur dans le coté gauche du ventre, sans irradiation. Les antécédents ne comportaient rien d'anormal et l'examen physique ne révélait rien de pathologique dans les différents systèmes. A hauteur de l'ombilic, dans le flanc gauche, existait une masse de 6 cm. de diamètre, légèrement mobile sur le plan de la paroi postérieure, invisible sur une radiographie à vide. Un lavement baryté permit de mettre en évidence un spasme du côlon descendant, dans la partie moyenne, à hauteur de la masse palpée.

Une laparotomie fut pratiquée: la masse, de la taille d'une mandarine reliait le colon descendant aux muscles du flanc gauche. On procéda à une excision de cette masse ainsi qu'à une résection colique partielle avec anastomose bout à bout. A la révision, un nodule hépatique de 2 cm. de diamètre fut enlevé.

L'examen anatomo-pathologique montra une masse formée de tissu inflammatoire granulomateux avec des infiltrats polymorpholymphocytaires, plasmocytaires et macrophagiques; dans ces infiltrats des colonies de champignons étaient visibles. Le nodule hépatique était simplement constitué de tissu hépatique en prolifération.

Les suites post-opératoires furent compliquées: un état d'iléus se prolongeant durant une quinzaine de jours nécessita une seconde intervention: des brides d'adhérences enserrant le côlon et des anses grêles furent levées. Les suites de la seconde opération furent très satisfaisantes.

Une revue de la littérature actuelle, plus spécialement au point de vue de la thérapeutique antibiotique, termine cet article.

STRANGULATED ADNEXA IN INFANTILE HERNIA*

L. S. A. BOOTHROYD, M.B., F.R.C.S., F.R.C.S.[C] and F. J. D. SPOHN, M.D., M.C.G.P.,
Vancouver, B.C.

THE PRESENCE of uterine adnexa in the inguinal hernia of an infant female is not unusual, but strangulation in such circumstances is rare. Such a case occurred in our practice and is reported here together with a summary of the facts obtained from the literature. Two major series have been analyzed and it is interesting to note the degree of agreement between these and our small series.

The canal of Nück normally obliterates at eight months of intrauterine life, and the ovary remains an abdominal organ until its descent into the developing pelvic cavity after the age of one.[1, 2, 5] In newborn infants the distance between the middle of the Fallopian tube and the internal inguinal ring is about 14 mm.[3] These facts account for the frequency with which the adnexa present in infantile herniæ. They occur nine times more commonly in inguinal than in femoral herniæ, and in the latter the ovary is seldom accompanied by the tube, in contrast with inguinal herniæ.[4] Ten per cent of all inguinal herniæ occur in females, and of these 12% are in girls under two years of age. A simple statistical "tree" illustrates the facts (Fig. 1).

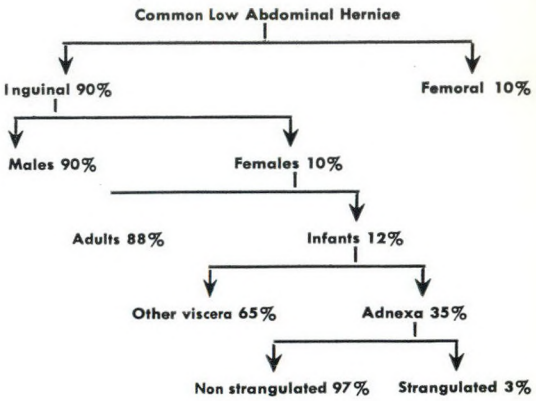


Fig. 1.

Wakeley's ten-year series (1919-29) and the ten-year series of Wiley and Chavez (1946-56) qualify the figures quoted in the table; in each, right inguinal hernia was twice as common as left inguinal hernia. In the latter series the mesosalpinx was present as a sliding component of the sac in 12 out of 52 infantile cases. Out of Wakeley's 25 collected cases only one had strangulated and out of Wiley's series of 52 cases none was strangulated. Strangulation of adnexa in the hernial sac appears therefore to be a rare complication.

In the North Vancouver General Hospital (averaging 100 beds) between the years 1948 and 1958, 34 operations were performed for inguinal herniæ on infants below the age of two years. Of these herniæ only three were in females and two of these contained uterine adnexa. In all, there were five times as many right-sided as left-sided herniæ.

In infants the ovary is painless, unless strangulation has supervened, and presents as a rounded, non-tender swelling. In adults the ovary is hypersensitive.[4]

The treatment is to perform a herniotomy after the age of one, unless complications force earlier surgery. We agree with the present trend which disapproves of exploration of the clinically unaffected opposite groin. It is well to beware of the mesosalpinx, which forms part of the wall of the sac in one-fifth of the cases.

CASE REPORT

G.L. was six weeks old at the time of admission, on December 21, 1957, to the North Vancouver General Hospital. There had been no birth trauma and she had been healthy until December 18, 1957, when she presented with a reducible left inguinal hernia for which a wool truss was provided. On December 21, 1957, the hernia became irreducible.

On admission, the lump in the left groin felt tense and tender and there was no impulse on straining. It was sausage-shaped and had many of the features of a femoral hernia without the palpable neck. It was irreducible and failed to reduce under sedation with morphine and elevation in Bryant's position with gallows traction.

At operation on December 21, 1957, the in-

*From the North Vancouver General Hospital, British Columbia.

guinal hernial sac was found to contain the left ovary and tube, which were completely strangulated, having twisted through 180° on a narrow pedicle. The medial wall of the sac was formed by the left horn of a bicornuate uterus. Reduction of these structures proved remarkably difficult from the inguinal region and could only be achieved by an intraperitoneal approach through a paramedian incision and pulling the uterus backwards. The uterine horn was viable but the left adnexa had to be removed. No inguinal repair was necessary.

The child made an uneventful recovery and has no evidence of recurrence ten months after operation.

Summary

The ovary and tube are present in one-third to one-half of inguinal herniæ in female infants, but strangulation of these structures, which occurred in the case reported, is rare. In one-fifth the mesosalpinx forms part of the wall of the sac as a sliding hernia. Two relevant series have been reviewed and a third reported, the information from each being largely in agreement.

References

1. Arey, L. B.: Developmental anatomy: a textbook and laboratory manual of embryology, W. B. Saunders Company, Philadelphia, 5th ed., 1946, chap. 14.
2. Chamberlain, J. M.: Anomalies and accidents complicating repair of inguinal hernias in infancy and childhood, *Boston M. Quart.*, **7:** 23, 1956.
3. Kousmine, B. J.: Tumeur volumineuse de la région iléofémorale gauche constituée par une hernie de la trompe uterine, *Rev. de chir.*, **15:** 313, 1895.
4. Wakeley, C. P. G.: Hernia of the ovary and Fallopian tube; 25 cases, *Surg. Gynec & Obst.*, **51:** 256, 1930.
5. Watson, L. F.: Hernia, C. V. Mosby Company, St. Louis, Mo., 1948.
6. Wiley, J. and Chavez, H. A.: Uterine adnexa in inguinal hernia in infant females, *West. J. Surg.*, **65:** 283, 1957.

Résumé

Un ovaire et une trompe sont inclus dans le tiers ou même la moitié des hernies inguinales des bébés du sexe féminin. L'étranglement cependant est rare. Les auteurs en rapportent un cas. Une fois sur cinq le mésosalpinx forme une paroi du sac d'une hernie par glissement. Le rappel de deux séries de cas ainsi que le rapport d'une troisième offre des renseignements qui s'accordent.

ROYAL COLLEGE OF PHYSICIANS AND SURGEONS OF CANADA

To the Editor:

On November 29, 1958, a meeting was held at CMA House in Toronto of representatives of medical organizations to discuss matters of medical ethics. This meeting was called on the invitation of The Royal College of Physicians and Surgeons of Canada.

The Royal College representatives reported on this meeting to the Council of The Royal College at the meeting of the Council held January 21, 1959. Arising out of the discussion, a statement relating to itinerant surgery and medical ethics in general was approved by the Council. The Council directed me to transmit to you this statement and indicate that the Council would appreciate your co-operation in publishing the statement in the *Canadian Medical Association Journal* and in the *Canadian Journal of Surgery*.

"In general The Royal College of Physicians and Surgeons disapproves of itinerant surgery. It is permissible only where there is need of skill in the art of surgery not possessed by those practising in the local area. The surgeon is morally and perhaps legally responsible for the diagnosis of the condition for which he operates, and for the post-operative care of the patient. If this principle is violated in the practice of itinerant surgery it is difficult to insist on its observance under other circumstances.

"When the welfare of the patient makes necessary the employment of a visiting surgeon rather than one resident in the area the arrangement regarding fees should be such that there is no improper financial inducement to the referring doctor or the surgeon.

"The Council of The Royal College declares its intention to continue its activities against unethical professional practices. When the College receives information which seems to make it necessary to investigate the professional practices of a Fellow or Certified Specialist it proposes to do so. Should the allegations of unethical conduct be substantiated, the College intends to take action under Article VII of its By-laws. Should the name of the Fellow or the Certified Specialist be removed from the College Register, notification of such action will be sent to the local provincial licensing body and to the Director of the Canadian Council on Hospital Accreditation.

"The Council of The Royal College desires to express its appreciation for the support it has received from the Canadian Medical Association, the College of General Practice of Canada and the medical organizations in the province of Ontario. Council is desirous that further discussions may make possible the solution of these ethical problems which concern the profession throughout the country."

James H. Graham, F.R.C.P.[C],
Secretary.

CANADIAN JOURNAL OF SURGERY

VOLUME 1 AND VOLUME 2

Box binders, which have been designed to supply both the protection and the pleasing appearance of a permanent binding, are available for both volumes of the Journal.

They are made in two sections so that the Journals may be easily slipped in and out. Both sections are covered in an embossed, leather-grained paper. The title, volume number and year are gold-stamped on the spine of the outer section.

The price of each binder is $1.50 postpaid.

Orders may still be placed for either or both of the binders by returning the form below to:

Canadian Journal of Surgery,
150 St. George Street,
Toronto 5, Ontario.

I enclose the sum of $.....

for.....binder(s)

Volume 1, 1957-58 ☐

Volume 2, 1958-59 ☐

Name.....

(Please print)

Address.....

.....

.....

.....

JOURNAL CANADIEN DE CHIRURGIE

TOME 1 ET TOME 2

Si vous désirez conserver vos exemplaires du Journal tout en garnissant votre bibliothèque, procurez-vous un de nos emboîtages de carton recouvert de papier simili-cuir. Ils se composent de deux boîtes avec titre, tome, année et numéro imprimés en or sur le dos.

Les différents numéros seront ainsi groupés, faciles d'accès et protégés de la poussière.

Ces emboîtages se vendent $1.50 l'exemplaire, franco de port. Servez-vous du bon de commande ci-dessous, que vous devez envoyer au

Journal Canadien de Chirurgie
150 rue St-George,
Toronto 5, Ont.

Ci-joint la somme de $.....pour.....emboîtage(s)

du Journal Canadien de Chirurgie.

Tome 1, 1957-58 ☐

Tome 2, 1958-59 ☐

Nom.....

lettres moulées s.v.p.

Adresse .....

.....

.....

.....

BOOK REVIEWS

(See also page 286)

ATLAS OF TECHNICS IN SURGERY. John L. Madden, New York Medical College, New York. 648 pp. Illust. Appleton-Century-Crofts, Inc., New York, 1958.

Surgery has now become so divided into subspecialties that it is rare indeed that one author undertakes the writing of a large volume to encompass almost all and more of the procedures undertaken by a general surgeon. Many surgical specialties are not touched, but this Atlas includes much, from the evacuation of a thrombosed hæmorrhoid and appendectomy to tendon graft and end-to-end splenorenal shunt. The author has written descriptions of standard operations to go with fine illustrations drawn by Alfred Feinberg. Criticisms of the procedures by sixty-two outstanding surgeons are added lest the reader get the impression that there is only one correct technique for any major operation.

This Atlas is an excellent reference work for residents, occasional surgeons, fellows in surgery, interns, and for practising surgeons who are about to undertake an operation not often on his list. And it is well worth browsing through to see how one great New York surgeon performs one's favourite operation.

The illustrations are beautifully clear and precise and suitable for all levels of surgical learning. The explanatory text, the articles on preoperative and postoperative care, fluid and electrolyte balance, and anæsthesia, the comments by the distinguished critics are concise and well-written.

"Atlas of Technics in Surgery" is a valuable aid in the almost impossible task of teaching surgery.

TREATMENT OF CANCER AND ALLIED DISEASES. Vol. 1. Principles of Treatment. Edited by George T. Pack and Irving M. Ariel. 646 pp. Illust. 2nd ed. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1958. $22.50.

The new edition of the classical work by Pack and Ariel is now available after an interval of 18 years. It is not surprising, therefore, that this volume is virtually a new book. The writing is catholic in style and the subject matter ranges from the organization of cancer programs through diagnosis and pathology to aspects of treatment by surgical, radiation and chemotherapeutic methods, ending on the very sound note of the methods used in reporting results of cancer treatment. The book is lavishly illustrated with radiographs, charts and photographs many of which are well known. (Figure 20-10 would be much more valuable if it were the right way up.)

It is difficult to be sure for whom this volume was prepared, for although the title is "Principles of Treatment" remarkably few principles are enunciated. On the other hand, there is a wealth of detailed information, particularly in Chapters 13 to 30 dealing with radiotherapy.

In the chapters on irradiation, the advances in physics during and since the war are embodied in the emphasis on supervoltage irradiation and the use of artificial radioisotopes. Each eminent authority has written a lucid and valuable chapter on his own field. The introductory chapters on irradiation covering the physical basis, the pathological response and the biological effects are an excellent summary of the present position. It is refreshing to see in print the statement that "three months after irradiation tumour cells may be found to look viable under the microscope, and yet the tumour subsequently disappears and the patient remains well for years." In the past, too often the histologist's comment that "the tumour has not been sterilized" has led to early and unnecessary surgical intervention.

In several of the chapters on radiotherapy, implantation of small radioactive sources such as radioactive gold seeds, cobalt beads, iridium or tantalum wire, is advocated for the treatment of metastatic lymph node disease. This would seem to confirm that there is a place for radiotherapy in the management of lymph node spread. This is in accord with certain current concepts.

The chapters on hormone therapy and chemotherapy are brief and to the point. In particular, Dr. Gelhorn's remarks on the unsuccessful attempts at chemotherapy in the terminal patient should be read by all who deal with malignant disease. In essence, the humane desire "to do something for a patient" is often best served by making that something nothing.

On the reporting of results of cancer therapy, the information in Chapters 36 and 37 is invaluable. It shows the many fallacies inherent in statistical analysis, and how easy it is to draw false conclusions from certain data. One cannot help but agree with Caskey (*Canad. M. A. J.*, 80: 251, 1959) that a national statistical group should be formed. The function of such a group would be to assess the statistics in papers relating to the results of cancer treatment—and either issue or withhold a seal of authority—after the style of *Good Housekeeping*. Some degree of validity would thus be ensured.

In summary, this volume is another milestone in medical literature. "Pack and Ariel" will find its place on the shelves of most medical libraries.

INTERNATIONAL SYMPOSIUM ON MAMMARY CANCER: Proceedings of 11th International Symposium on Mammary Cancer held at the University of Perugia, July 24-29, 1957. Edited by L. Severi. 871 pp. Illust. Division of Cancer Research, Perugia, 1959.

This volume contains the papers presented at the second International Symposium on Mammary Cancer held in the University of Perugia July 24-29, 1957. It would be quite impossible to do justice to this publication in a short review. Professor A. Haddow attempted to give a summing up of the conference in the final seven pages.

(Continued on page 317)

CANADIAN JOURNAL OF SURGERY

All communications concerning this Journal should be marked "Canadian Journal of Surgery" and addressed to the Editor, C.M.A. Publications, at C.M.A. House, 150 St. George St., Toronto 5.

The Journal is published quarterly. Subscription is $10 per year. (It would be greatly appreciated if subscribers would please add bank exchange to their cheques.)

INSTRUCTIONS TO CONTRIBUTORS

Manuscripts

Manuscripts of original articles, case reports, and other contributions should be forwarded with a covering letter requesting consideration for publication in the *Canadian Journal of Surgery*. Acceptance is subject to the understanding that they are submitted solely to this Journal, and will not be reprinted without the consent of the author and the publishers. Acceptance or rejection of contributions will be determined by the Editorial Board. As space is available, a limited number of case reports will be published. Articles should be typed on one side only of unruled paper, double-spaced, and with wide margins. Carbon copies cannot be accepted. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents. The Concise Oxford Dictionary will be followed for spelling. Dorland's American Medical Dictionary will be followed for scientific terminology. The Editorial Board reserves the right to make the usual editorial changes in manuscripts, including such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity with the style of the *Canadian Journal of Surgery*. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints

Reprints may be ordered on a form which will be supplied with galley proofs. It is important to order these before publication of the article, otherwise an extra charge for additional type-setting will be made.

References

References should be referred to by numerals in the text and should be set out in accordance with the *Cumulative Index Medicus* abbreviation of journal name and general style. They should include in order: the author's name and initials in capitals; title of the article; abbreviated journal name; volume number, page number and year. References to books should include in order: author's name; title of book; title of publishing house; city of publication; number of edition (e.g., 2nd ed.); year of publication.

Illustrations

A reasonable number of black-and-white illustrations will be reproduced free with the articles. Colour work can be published only at the author's expense. Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10″ x 8″. Prints of radiographs are required and *not the originals*. The magnification of photomicrographs must always be given. Photographs must not be written on or typed on. An identifying legend may be attached to the back. Patients must not be recognizable in illustrations, unless the written consent of the subject for publication has been obtained. Graphs and diagrams should be drawn in India ink on suitable white paper. Lettering should be sufficiently large that after reduction to fit the size of the Journal page it can still be read. Legends to all illustrations should be typed separately from the text and submitted on a separate sheet of paper. Illustrations should not be rolled or folded.

Language

It should be clearly understood that contributors are at full liberty to submit articles in either English or French, as they please. Acceptance will be quite independent of the language of submission. If the contributor wishes, he may submit an informative summary of not more than 300 words in the language other than that in which he has submitted the article. For example, an article in English must carry an English summary and may, if the author wishes, carry a more detailed summary in French.

BOOK REVIEWS

(Continued from page 315)

Perhaps some idea of the contents of the volume can be given by quoting the first paragraph of this summary.

"Inevitably this summary must be regarded as incomplete and selective, and there is obvious difficulty in attaining completeness in any survey of some eighty papers from fifteen countries—especially when these cover as wide a range as pathogenesis and hormonal studies, morphology and histogenesis, clinical diagnosis and therapy of human breast cancer, carcinogenesis, hormone and endocrine gland studies, cellular pathology, genes and virus studies, attempts at prophylaxis and therapy of experimental breast cancer and lastly, comparative studies between human and experimental breast cancer."

There is something of interest in the volume to anyone interested in any aspect of the problem of breast cancer. Many of the papers are, of course, highly technical and not conclusive. One gains from perusing this publication some notion of the tremendous amount of work that is being done upon this problem and the great variety of the approaches being made. Dr. Lucio Severi, who was responsible for the organization of the conference and the publication of its proceedings, is entitled to our congratulations and gratitude.

TREATMENT OF BREAST TUMORS. R. S. Pollack, Stanford University School of Medicine, Stanford, California. 147 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1958. $6.00

This is a remarkably well written and illustrated book with chapters by contributors on pathology, radiotherapy, hormone therapy, hypophysectomy and extended radical mastectomy. The aims of brevity and conciseness have been admirably accomplished, giving a clear and rapid review of the breast tumour problem and its management.

BREAST CANCER. The Second Biennial Louisiana Cancer Conference, New Orleans, January 22-23, 1958. Edited by Albert Segaloff. 257 pp. Illust. The C. V. Mosby Company, St. Louis, Mo., 1958. $5.00.

This volume on breast cancer may not at first appear to come up to other more complete texts on the subject, but in fact it is much more satisfying and informative to the clinician. This book is a record of the Second Cancer Conference in Louisiana and includes presentations by authorities in their fields.

It is divided roughly into four parts. There are two sections on basic biology, one on definitive therapy, and one on hormonal therapy. Summation by the editor ends the book.

One of the attractions of this most interesting and authoritative book is a panel discussion at the end of each section which acts as a "breather" from the preceding heavier reading, and answers questions which naturally arise out of this reading.

This is a book which can be perused rewardingly in one evening, yet remain on one's shelf for later reference.

MODERN TRENDS IN ANÆSTHESIA. Edited by F. T. Evans and T. C. Gray. 318 pp. Illust. Butterworth & Co. Limited, London and Toronto, 1958. $13.50.

Anæsthesia as it is practised today (1958) in leading teaching centres is discussed in this volume of 22 easily read chapters. Each chapter deals with a specific subject and has been written by a separate author, each a recognized authority in his field. The authors are predominantly British, but some contributions are made from Canada, the United States and Scandinavia.

Among the subjects discussed are the relaxant drugs; new conceptions of consciousness; hypothermia; cardio-respiratory pumps; anoxic states and their treatment; trends in the mode of investigation of anæsthetic problems. A nice balance has been struck between theory and practice. The chapters are compact, with ample references.

This book will be read with interest and profit by all practising anæsthetists, particularly those engaged in teaching. It will also be a source of ready reference to all who desire the latest thinking in anæsthesia.

SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM. Sir John Paterson Ross, St. Bartholomew's Hospital, London (England). 170 pp. Illust. 3rd ed. Baillière, Tindall and Cox, London; The Macmillan Company of Canada Limited, Toronto, 1958. $6.00.

The wave of enthusiasm for sympathetic surgery has passed and the rationale behind the various procedures and the results achieved can now be more calmly contemplated. Sir James Paterson Ross has always combined thoroughness with a certain objectivity and detachment in his writings on this and other subjects in which he is an acknowledged authority. The third edition of his book (the first appeared in 1933) has been largely rewritten to express his final conclusions on a topic that has occupied a prominent place in his thoughts and surgical practice during the last thirty years. This is therefore one man's creed rather than a comprehensive work on surgery of the sympathetic nervous system, and as such it has a singular value.

OPERATIVE SURGERY. Volume 8. Neurosurgery, Eyes, Ear, Nose and Throat. Edited by Charles Rob and Rodney Smith, London, England, 196 pp. Illust. Butterworth & Co. (Canada) Limited, Toronto, 1958. $21.50.

In the January 1959 issue of this Journal (page 218) the price of Operative Surgery Volume 8

(Continued overleaf)

was incorrectly quoted as $19.50. The correct price is $21.50.

TUMORS AND TUMOROUS CONDITIONS OF THE BONES AND JOINTS. H. L. Jaffe, Hospital for Joint Diseases, New York, 629 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1958. $18.50.

From a modern authority on bone tumours has come one of the finest contributions to orthopædic pathology in recent times. "Tumors and Tumorous Conditions of the Bones and Joints" by Jaffe is an up to date presentation of the accepted facts pertinent to the whole field of bone tumours. By correlation of clinical and radiological features with pathological characteristics, a text valuable to orthopædist, radiologist and pathologist has been presented.

Particular attention is to be paid to the author's approach to classification and diagnosis. Some previously accepted neoplastic identifications are discussed and the limitations that have appeared with the advent of newer knowledge are pointed out. Dr. Jaffe dwells on description of the lesions rather than marshalling reasons for a personally preferred classification.

The accepted facts related to the standard lesions are presented in workmanlike fashion. Clinical considerations, gross and microscopic pathological appearances, x-ray picture, differential diagnosis and current treatment are documented for each entity.

Certain more recently demarcated lesions like aneurysmal cyst of bone, glomus tumour of the bone and others are fully described. The author has wisely included disturbances related to bone-like tumours from overlying soft parts and some tumour-like conditions such as villonodular synovitis that add to the completeness of the book. This work may be recommended as the most complete presentation of the subject at the present time.

ANATOMY FOR SURGEONS. Volume 3 – The Back and Limbs. W. Henry Hollinshead. Illust. Paul B. Hoeber Inc., Medical Book Department of Harper & Brothers, New York, 1958. $23.50.

This is the third and last volume of Hollinshead's *Anatomy for Surgeons*. The first two volumes cover the head and neck, and the thorax, abdomen and pelvis. Following an introductory section, the rest of the book is divided into sections in which anatomy is discussed by regions, e.g. pectoral region, axilla and shoulder; the wrist and hand. This general approach will be familiar to Canadian graduates of recent years who were brought up on Grant's *Method of Anatomy* and who have been taught to consider all of the structures in a region instead of learning each system separately (angiology, myology, osteology, etc.) as is the vogue in most texts. For this reason, Dr. Hollinshead's book will doubtless have rather more appeal for what might be termed lesser or non-Grantian breeds of surgeon!

The author says a little about many things, with the result that detail is lacking. For this reason the practical value of the book may be questioned; those who expect hints on surgical exposures will be disappointed. Perhaps the greatest value of the book is that is gathers together in one place the wealth of material on "normal variations" in anatomy which has been the surgeon's contribution to this field. Thus the variations in innervation to the intrinsic muscles of the hand are clearly discussed, and under the shoulder one finds a clear summary of de Palma's work on the normal occurrence of separation of the glenoid labrum and of spontaneous "rupture" of the rotator cuff.

After reading this book, one cannot help but feel that the best "anatomy for surgeons" remains one of the great standard texts of anatomy, but that there is room for a smaller reference volume devoted to describing the variations from the normal—a field of endeavour in which the surgeon is in a very advantageous position for making contributions.

TRACHEOTOMY: A CLINICAL AND EXPERIMENTAL STUDY. Thomas G. Nelson. 102 pp. Illust. Williams & Wilkins Co., Baltimore, Md., 1958.

This is a first-class monograph on the expanding value of tracheotomy. It is short (102 pages) yet comprehensive, and illustrated with pictures, sketches and case reports. Particularly useful is the section on indications for operation; secretional respiratory obstruction with its sequelæ is well outlined. The author gives experimental reasons for preferring a collar incision, division of thyroid isthmus and excision of a piece of tracheal wall. One might wish that more emphasis was laid on the place of tracheotomy with mechanical respirators.

ANALYSE, DIAGNOSTIC, CLINIQUE, PRONOSTIC ET TRAITEMENT DES TUMEURS BENIGNES ET MALIGNES. LEUR DIAGNOSTIC PRECOCE EN CONSULTATION. C. H. A. Perret. 892 pp. Illust. G. Doin et Cie, Paris, 1958.

Les "Tumeurs benignes et malignes" par le Professeur Charles A. Perret de l'Université de Lausanne est un volume de 892 pages comprenant 184 figures dans le texte.

Le plan de l'ouvrage est ordonné selon les différentes régions du corps. Les tumeurs de chaque organe sont étudiées séparément selon leur diagnostic clinique, leur pronostic et leur traitement. L'auteur a omis intentionnellement toute description de pathologie microscopique. Par contre, il attire souvent l'attention ou lecteur sur l'importance d'un diagnostic précoce et il expose très clairement les moyens d'y arriver. Les références sont surtout tirées de la littérature suisse, allemande, française et scandinave. Le traitement de certaines tumeurs est parfois dif-

férent ici en Amérique de celui indiqué par l'auteur.

Dans l'ensemble, cet ouvrage est remarquable et il est à conseiller aux médecins et étudiants en médecine, à cause surtout de sa valeur pour le diagnostic clinique des tumeurs.

THE COMPARATIVE ANATOMY AND PHYSIOLOGY OF THE NOSE AND PARANASAL SINUSES. Sir Victor Negus, King's College Hospital, London. 402 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto. 1958. $12.00.

Specialists in rhinology, anatomists, physiologists and biologists will read this new book with pleasure and enthusiasm. Those in various other fields of medicine and science will find it an easy and enjoyable book. The reason for this is twofold: first, the book is comprehensive and authoritative, and second, it is written by the author with unusual clarity, wit, and a patent love of the subject.

Sir Victor Negus has studied the ear, nose and throat in health and disease for many years, a fact that is attested to by his long series of publications over several decades. In addition to his clinical interest, he has been extremely active in the study of the normal respiratory tract. For the last eight years he has been investigating the comparative anatomy and physiology of the nose. During this work he has rebuilt a special museum collection of the Royal College of Surgeons in London and he has also used the facilities of the Ferens Institute of the Middlesex Hospital Medical School.

Undoubtedly anyone looking at this book will recognize it as a masterpiece. It is hard for the reviewer to imagine that a better book will ever be written on this subject. Profusely illustrated, it contains almost 200 illustrations, mostly original and almost all extremely useful.

This book can be recommended without reservation to anyone having the slightest interest in the anatomy and physiology of the nose.

CLINICAL OBSTETRICS AND GYNECOLOGY, Vol. 1, No. 3. Symposium on Abnormal Uterine Bleeding. Edited by J. I. Brewer: Symposium on Special Diagnostic Aids. Edited by C. P. Hodgkinson. 852 pp. Illust. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1958. $18.00 per volume.

The book contains two symposia—the first on "Special Diagnostic Aids" and the second on "Abnormal Uterine Bleeding".

The section which deals with aids in diagnosis does not provide very stimulating reading material but the subject makes this largely unavoidable. Five of the 17 chapters are devoted to the application of cytological techniques to gynæcology, and this fact gives justifiable emphasis to the growing recognition of the value of this aid. Two of the sections are devoted to reviews of methods of diagnosis which have not been used very widely. These include "The Gynogram" and "Urethrocystograms; Metallic Bead Chain Technique".

The authors of three of the reviews, "Culdoscopy" by Albert Decker, "Hypofibrinogenæmia" by Duncan E. Reid and Charles C. Roby, and "Rh and ABO Incompatibility" by Milton L. McCall, deserve special praise for their conciseness and clarity.

The second symposium concerning abnormal uterine bleeding is, if anything, a little too exhaustive. Schauffler's "Bleeding during Infancy and Childhood" is excellent, and Lock's — "Psychosomatic Aspects of Uterine Bleeding" provides some shrewdly pointed examples of this often neglected phase of these problems.

In the main, this volume covers its subject matter adequately but may be received somewhat less enthusiastically by the specialist than the preceding numbers.

HEY GROVES' A SYNOPSIS OF SURGERY. Edited by Sir Cecil P. G. Wakeley. 650 pp. Illust. 15th ed. revised. John Wright & Sons Limited, Bristol; The Macmillan Company of Canada Limited, Toronto, 1958. $6.35.

It is 50 years since Hey Groves expanded his lecture notes to students and produced the first edition of this synopsis, which appears still to enjoy popularity among hard-pressed examination candidates. In the present edition, Sir Cecil Wakeley bows out as author, leaving a younger London surgeon to continue the task of revamping this work to reflect advances in surgery. The book remains strictly what it claims to be—a synopsis containing the maximum number of facts and opinions on all branches of surgery in the minimum number of words.

HEAD INJURIES. Mechanisms, Diagnosis and Management. E. S. Gurdjian and J. E. Webster, Wayne State University College of Medicine, Detroit, Mich. 482 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1958. $14.00.

This nicely published volume begins with a résumé of the history of head injuries and their treatment. This portion of the book is a fascinating summary of cranial cerebral trauma beginning in the prehistoric period and extending to modern times. The chapter on anatomy is insufficient in some details but on the whole provides an adequate account of the structures germane to the subject. The third chapter on the mechanisms of injury to the skull and to the brain is the most original and worthwhile section of the book. It comprises a concise review of present theories and knowledge and an exposition of the authors' important clinical and experimental research in this field.

Although the illustrations of roentgenograms are disappointing, the description of diagnostic techniques is complete. In the reviewer's opinion,

the significance of electroencephalographic changes is somewhat overemphasized.

In the subsequent seven chapters descriptions are given of the various clinical and pathological manifestations of different types of head trauma. Although much of it is repetitive, the plan of the book makes it easy for one to look up any particular topic of interest. There are very few original observations or conclusions drawn and these portions of the work do not differ significantly from the writing of other good authors. The chapter on the medico-legal aspect of head injury warns of pitfalls to avoid in the presentation of evidence.

The section on the general care and management of non-surgical conditions emphasizes the use of tracheotomy and the general nursing care of the unconscious patient. In the chapter on surgical management and techniques, detailed descriptions of the various procedures are given. In such matters most surgeons have their own preference but all would subscribe to the principle expounded.

The authors tend to write a great deal in the subjunctive tense. Presumably in an effort to achieve coverage of their subject, they merely mention a number of topics which might be better omitted or discussed in a more informative manner. Many sections are written in a halting, awkward literary style. Others flow smoothly and are easily read.

This book should be in the library of persons specifically interested in the care of head injuries.

Books Received

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

Modern Trends in Surgical Materials. Edited by Leon Gillis, London, England. 266 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1958. $14.50.

Klinische Methoden der Blutgerinnungsanalyse (Clinical Methods of Blood Coagulation Analysis). J. Jürgens, Frankfurt am Main, and F. K. Beller, Tübingen. 391 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1959. $13.35.

Obstetric and Gynecologic Milestones: Essays in Eponymy. Harold Speert, Columbia University College of Physicians and Surgeons, New York. 700 pp. Illust. The Macmillan Company, New York; Brett-Macmillan Ltd., South Galt, Ontario, 1959. $15.00.

Nouvelle Pratique Chirurgicale Illustrée: Fascicule XII. (New Surgical Techniques Illustrated). Edited by Jean Quénu. 296 pp. Illust. G. Doin et Cie, Paris, 1958. 3.500 fr.

A Practice of Thoracic Surgery. A. L. d'Abreu, Birmingham, England. 619 pp. Illust. 2nd ed. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1959. $17.00.

Neoplastic Disease at Various Sites. General Editor, D. W. Smithers. **Volume II—Tumours of the Bladder.** Edited by David M. Wallace, London, England. 352 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1959. $10.25.

Surgery of the Sympathetic Nervous System. Sir James Paterson Ross, Director of the Surgical Professorial Unit, St. Bartholomew's Hospital, London, England. 170 pp. Illust. 3rd ed. Baillière, Tindall and Cox, London; The Macmillan Company of Canada Limited, Toronto, 1958. $6.00. (Reviewed in this issue.)

Fracture Surgery. A Textbook of Common Fractures. Henry Milch, Hospital for Joint Diseases, New York, and Robert Austin Milch, Peter Bent Brigham Hospital, Boston. 470 pp. Illust. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1959. $17.50.

Fractures et Luxations (Fractures and Dislocations). Paul Mathieu. 211 pp. Illust. L'Expansion Scientifique Française, Paris, 1959. 1.500 fr.

Modern Trends in Diseases of the Vertebral Column. Edited by Reginald Nassim and H. Jackson Burrows. 292 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1959. $15.00.

Atlas de Techniques Chirurgicales: Les grandes techniques: cou, thorax, abdomen, chirurgie pelvienne. R. Michel-Bechet. 580 pp. Illust. G. Doin et Cie, Paris, 1958. 25.000 fr.

Breast Cancer: Factors Modifying Prognosis. A. J. Delario. 208 pp. The Macmillan Company, New York; Brett-Macmillan Ltd., South Galt, Ontario, 1959. $7.50.

Urology in Outline. T. L. Chapman, University of Glasgow, Scotland. 176 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1959. $4.70.

Cancer of the Pharynx, Larynx and Œsophagus and its Surgical Treatment. Ronald W. Raven, University of London, England. 292 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1958. $13.50.

Treatment of Cancer and Allied Diseases. Volume 2: Tumors of the Nervous System. Edited by George T. Pack and Irving M. Ariel. 316 pp. Illust. 2nd ed. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1958. $15.00.

Les Traumatismes du Thorax (Thoracic Injuries). Jean-Charles Sournia, Beyrouth. 354 pp. Illust. G. Doin et Cie, Paris, 1958. 4.800 fr.

Clinical Orthopedics No. 12. Rehabilitation. Anthony F. dePalma, Editor-in-Chief. 327 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1958.

Principles of Peripheral Vascular Surgery. S. Thomas Glasser, New York Medical College, Metropolitan Medical Center, New York. 410 pp. Illust. F. A. Davis Company, Philadelphia; The Ryerson Press, Toronto, 1959. $13.75.

The Effect of Advancing Age Upon the Human Spinal Cord. L. Raymond Morrison, with the collaboration of Stanley Cobb and Walter Bauer. 127 pp. Illust. Published for The Commonwealth Fund by Harvard University Press, Combridge, Mass.; S. J. Reginald Saunders and Company Limited, Toronto, 1959. $6.60.

Les Dechirures Obstetricales Compliquées du Perinée: Traitement Chirurgical (Complicated Obstetrical Tears of the Perineum: Surgical Treatment). René Musset, Marcel Cottrel and Maurice Dubost, Paris, 120 pp. Illust. Masson et Cie, Paris, 1958. 1.400 fr.

Cadres de Suspension et Attelles de Sustentation (Suspension Frames and Sustaining Braces). G. Rieunau, Toulouse. 54 pp. Illust. G. Doin et Cie, Paris, 1959. 600 fr.

CHANGE OF ADDRESS

Subscribers should notify the Canadian Journal of Surgery of their change of address *two* months before the date on which it becomes effective, in order that they may receive their Journal without interruption. The coupon on page 33 is for your convenience.