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L'HISTOIRE DE LA CHIRURGIE CANADIENNE

UNE OPÉRATION CHIRURGICALE
à l'Hôtel-Dieu de Québec en 1700

ÉMILE GAUMOND, M.D.,* *Québec*

On était en 1700 sous le règne de celui qu'on désignait comme le grand roi: Louis le quatorzième. La colonie québecoise qui était la colonie canadienne du temps renfermait 14,000 habitants. Québec n'avait pas cent ans mais déjà depuis 1639 les religieuses de l'Hôtel-Dieu de Québec dispensaient leurs soins aux malades tant civils que militaires. Déjà, à cette époque, la spécialisation avait cours. La chirurgie était l'apanage des chirurgiens, la médecine, des médecins, et les différents remèdes étaient préparés par les apothicaires. De tous ceux-ci, un seul retiendra notre attention: Michel Sarrazin.

Michel Sarrazin,[3] fils de Claude et de Madeleine Bonnefoy, était né à Nuits-Saint-Georges, en Bourgogne, le 5 septembre 1659. D'abord étudiant au Séminaire des Missions étrangères de Paris, puis étudiant en médecine, il était venu au Canada en 1685, attaché aux troupes en qualité de chirurgien-major; il avait pratiqué son art à Montréal et à Québec. Au dire de Vallée:[2] "Comme tous du reste, il n'était encore que chirurgien, mais les efforts accomplis bientôt pour devenir médecin indiquent nettement sa mentalité supérieure".

Il passe donc trois ans en France et revient quelques années après, docteur en médecine de l'Ecole de médecine de Reims, et se fixe à l'Hôtel-Dieu. Pendant son voyage de retour, il donne ses soins sur le bateau aux passagers atteints du *Pourpre* qui devait être le typhus à forme purpurique. En 1702-1703 une épidémie de variole lui donna l'occasion de faire montre de ses connaissances médicales.

Membre correspondant de l'Académie royale des Sciences, ami de Tournefort, du Jardin des plantes appelé aussi Jardin royal, et de Réaumur, botaniste, anatomiste, naturaliste, biologiste, chirurgien, médecin et praticien remarquable, il devait faire partie du Conseil supérieur de Québec en 1707 et mourir en 1734 à l'Hôtel-Dieu d'une fièvre maligne contractée dans l'assistance des contagieux. Voilà ce qu'était celui dont nous allons raconter les prouesses.

Instruments de Chirurgie

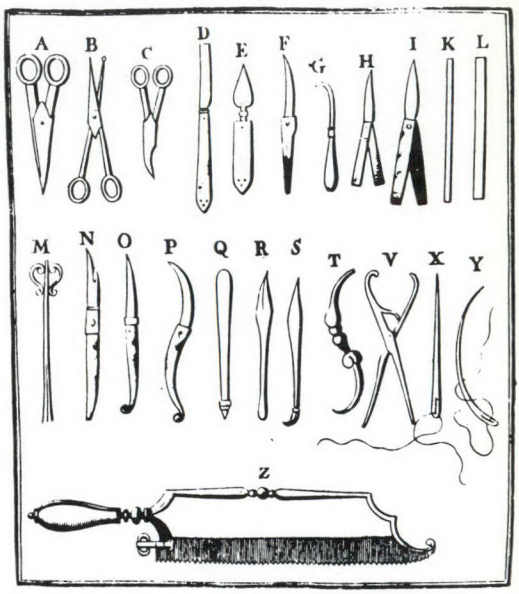

Fig. 1.—A—Ciseaux; B—Ciseaux à incisions; C—Ciseaux courbes; D—Rasoir; E—Scalpel; F—Autre scalpel; G—L'airigne; H—Lancette; I—Lancette à abscés; K—Sonde; L—Sonde platte; M—Une espèce de sonde creuse; N—Bistoury; O—Bistoury droit; P—Bistoury courbe; Q—Spatule; R—Une feüille de mirthe; S—Autre feüille de mirthe; T—Elévatoire; V—Pincettes; X—Aiguille droite; Y—Aiguille courbe; Z—Scie.

Marie Barbier de l'Assomption était fille de Gilbert Barbier, le Minime, charpentier, l'un des premiers colons de Ville Marie, où il était arrivé en 1642, avec la première recrue de Maisonneuve, et de Catherine Bellaveau. Entrée en 1678 à la Congrégation de Notre-Dame fondée à Montréal par Marguerite Bourgeoys, elle avait été envoyée en 1685 à la Mission de la Sainte-

*Président de la Société canadienne d'Histoire de la Médecine.

DES TENTES ET CANULES

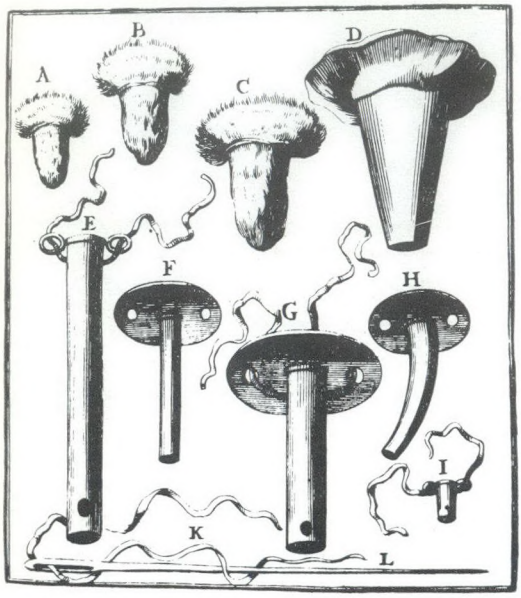

Fig. 2.—A—Petite tente de charpie; B—Moyenne tente de charpie; C—Grosse tente de charpie; D — Tente de linge; E — Grande canule; F — Canule à platine; G—Canule plate; H—Canule courbe; I—Petite canule; K—Séton; L—Aiguille à séton.

DES PLUMACEAUX

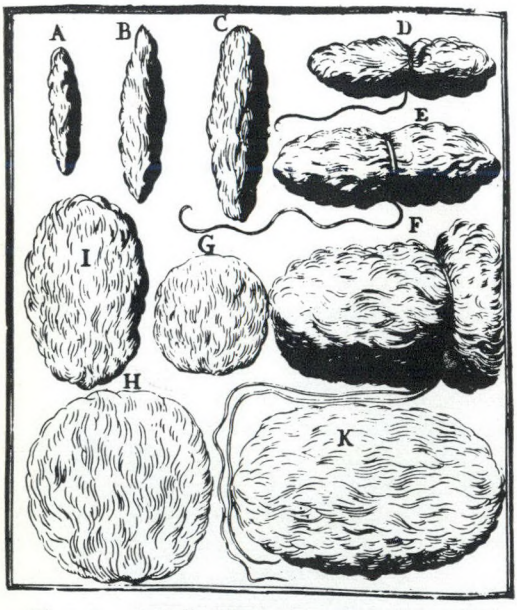

Fig. 3.—A-B-C—Trois bourdonnets; D-E—Deux plumaceaux liez; R—Gros tampon; G-H—Plumaceaux ronds et plats; I-K—Plumaceaux ovalaires.

Famille dans l'île d'Orléans, puis en 1686 mise à la tête de la Maison de la Providence, qui venait d'être établie à Québec. Rentrée quelque temps à Montréal, elle y avait été élue en 1693, supérieure de la Congrégation où elle succédait à Marguerite Bourgeoys, démissionnaire. Marie Barbier de l'Assomption était la première canadienne entrée dans le nouvel institut.

"Dès le petit printemps[3] de l'année 1700, la chère Sœur Marie Barbier de l'Assomption, Congréganiste, descendit de Montréal pour se faire guérir chez nous d'un cancer qu'elle avait au sein. Elle avait déjà demeuré quatre mois dans notre Communauté en 1698, où on la traita pour ce même mal, qui, étant depuis considérablement augmenté, l'obligea d'y revenir, et après quelques préparations, monsieur Sarrazin, aussi habile chyrurgien que sçavant médecin luy fit très heureusement l'opération le 29e de may; c'était le seul remède qui pouvoit l'empêcher de mourir". C'est en ces termes laconiques qu'est racontée dans les Annales de l'Hôtel-Dieu la première opération—paraît-il—d'un cancer du sein au Canada.

Nous analyserons ce bref exposé et à l'aide de documents de l'époque nous allons tenter de démontrer comment on faisait le diagnostic de cancer du sein, la technique chirurgicale, les soins pré- et post-opératoires, de même que toutes les choses qui accompagnaient ce genre d'opération inusité en l'an 1700.

Il est dit dans les Annales que la Sœur Barbier avait déjà passé quatre mois à l'Hôtel-Dieu deux ans auparavant. Elle avait sans doute une plaie au sein, à ce moment, plaie qui était peut être occasionnée par les mortifications que s'imposait la Sœur Barbier: port de cilices, de corsets et de ceintures hérissées de pointes. Ce traumatisme constant fut sans doute à l'origine de cette plaie que Sœur Barbier vint se faire traiter en 1698 et pour laquelle Sarrazin ne préconisa que les traitements locaux.

Apparemment cette plaie ainsi traitée par des emplâtres, onguents ou lotions ne guérit pas car en 1700 la Sœur Barbier revient pour se faire amputer le sein. C'est à ce moment que le diagnostic de cancer est porté. S'agissait-il d'un véritable cancer? Il

est à croire que oui si on se fie aux connaissances qu'avait Sarrazin de cette sorte de tumeur, connaissances qu'il avait acquises sans doute en suivant le cours de Dionis au Jardin royal à Paris.

Sarrazin toutefois est hésitant: "Quelque parti que je prenne, disait-il, je vois la Sœur de l'Assomption en danger d'une mort prochaine. Si on ne lui fait pas l'opération elle mourra certainement et sous peu de jours, son mal empirant à vue d'œil; et tenter l'opération c'est lui donner presque infailliblement le coup de mort, n'y ayant quasi pas d'espérance qu'elle la soutienne et moins encore qu'elle en puisse guérir." Sarrazin avait donc raison de diagnostiquer un cancer, les connaissances qu'il avait acquises lui en donnant toute autorité. Sarrazin avait dû suivre les cours de chirurgie du Jardin royal comme nous l'avons dit et voici ce que Dionis[1] qui donnait ces cours dit du cancer: "Le cancer est d'un consentement unanime le plus horrible de tous les maux qui attaquent l'homme; quoique la rage et la peste tuent en moins de temps, elles ne me paraissent pas si cruelles que le cancer qui mène aussi sûrement, mais plus lentement son homme au tombeau, en lui causant des douleurs qui lui font tous les jours souhaiter la mort."

Dionis divise le cancer en deux variétés: le cancer apostème et le cancer ulcéré. La description qu'il en fait correspond très bien à ce que nous connaissons du cancer aujourd'hui. Sur les causes des cancers Dionis[1] déclare: "les causes, selon quelques-uns, sont externes et internes: les premières se rapportent à une forte contusion, ou bien à une compression, comme il arrive plus ordinairement aux glandes des mamelles des femmes; ce qui donne lieu à la lymphe de s'arrêter, de s'épaissir et d'acquérir de l'âcreté par son séjour. La principale des causes internes est dans le vice des liqueurs séparées d'un sang terrestre et visqueux tout rempli d'acides coagulants qui forment des obstructions dans les glandes, y retiennent la lymphe et l'y disposent à s'aigrir jusqu'à corrompre la substance glanduleuse qui la renferme."

"De vingt femmes qui auront des cancers il y en aura quinze qui seront dans l'âge de quarante-cinq à cinquante ans, où la nature a contribué de faire cesser les évacuations

DES EMPLASTRES

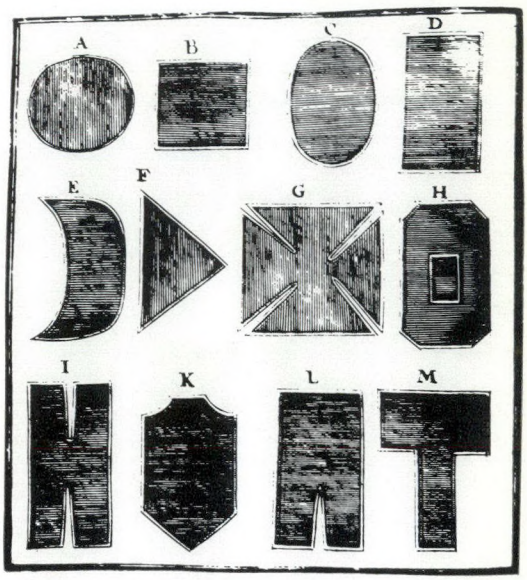

Fig. 4.—A—Emplastre rond; B—E. quarré; C—E. ovale; D—E. longitudinal; E—E. en croissant; F—E. triangulaire; G—E. en crois de Malthe; H—E. fenestré; I—E. trapezial; K—E. en écusson; L—E. ypsiloïde; M—E. en T.

DES COMPRESSES

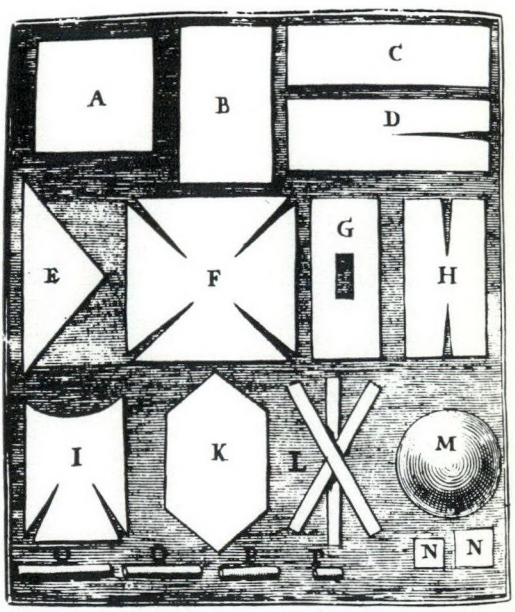

Fig. 5.—A—Compresse quarrée; B—C. splénique; C—C. longitudinale; D—C. circulaire; E—C. triangulaire; F—C. en crois de Malthe; G—C. fenestrée; H—C. trapéziale; I—C. pour épaule; K—C. lozange; L—C. oblique; M—C. ronde; N-N—Petites compresses; O-O—Longuettes; P-P—Roulées et très petites.

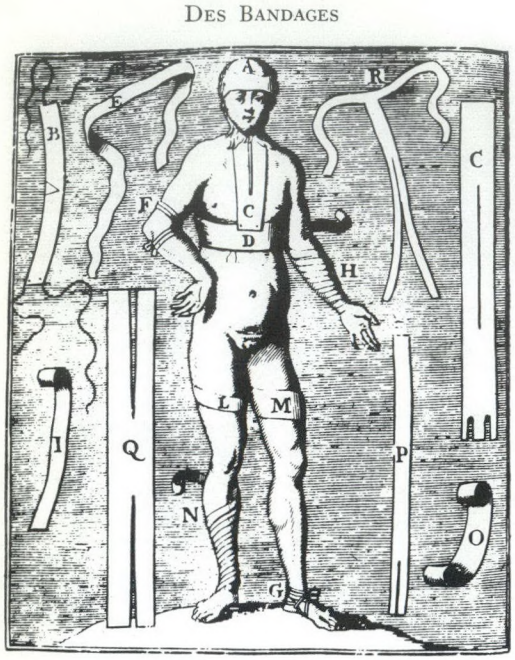

Fig. 6.—A—Le couvre-chef; B—Le bandeau; C—Le scapulaire; D—La serviette; E-F-G—Bande à saigner; H-I—Bandage rampant; L—Bandage simple; M—Autre bandage simple; N—Bandage avec des renversés; O—Bande roulée à deux chefs; P—Bandage incarnatif; Q—Bandage à quatre chefs; R—Bandage en T.

menstruelles. Ce mal est fort fréquent dans les couvents de fille" . . .

Dans le cancer ulcéré dit Dionis,[1] "le pronostic n'en peut être que fâcheux." D'après Hippocrate il ne fallait pas toucher à ces cancers ulcérés mais, comme le dit justement le même Dionis dans sa cinquième démonstration au Jardin royal, "comment résister aux persécutions d'une pauvre malade qui souffre et qui implore votre secours; un chirurgien doit chercher les moyens de la guérir et si cela n'est pas dans son pouvoir, il faut du moins qu'il travaille à adoucir son mal et à le lui rendre plus supportable." Non seulement, dans le temps, on était au courant de l'existence du cancer, de son diagnostic, d'une partie au moins de ses causes mais on en connaissait aussi le pronostic.

Revenons au thème principal de ce travail et imaginons un peu l'opération que subit la Mère Barbier. D'après Rumilly, "à Québec, les religieuses de l'Hôtel-Dieu la reçurent avec l'agrément de l'évêque, et la traitèrent comme une des leurs."

"Elle fit une neuvaine à Saint-Joseph, et toutes les religieuses de l'Hôtel-Dieu la firent avec elle comme si elles eussent prié pour une agonisante. On priait aussi à Ville-Marie. Le dernier jour de la neuvaine, le 29 mai devait être le jour de l'opération. Marie Barbier avait fait une confession générale; elle entendit la messe avec les religieuses à quatre heures et demie du matin. Elle communia après les religieuses et comme elles, à la grille. Le docteur Sarrazin communiait aussi pour renforcer le peu de chances qu'il avait de réussir une opération si exceptionnelle et si périlleuse."

On imagine facilement la sœur Barbier qui a déjeuné ou non se dirigeant d'un pas calme et en priant vers le lieu du supplice qui était la salle d'opération. Sarrazin[3] y avait fait placer sur une table les instruments dont il devait se servir: bistouri, rasoir, pinces, couteau, tentes, canules, plumaceaux, compresses, emplâtres et bandages.

Disons en passant qu'aucune anesthésie ne fut faite car selon Rumilly "Marie Barbier pria tout le temps de l'opération, qu'elle offrait en expiation de ses péchés". Sans doute lui a-t-on donné auparavant une liqueur alcoolique, du cognac par exemple afin de soutenir un courage qui aurait pu devenir chancelant. Aucune potion narcotique ne fut semble-t-il donnée. Les potions narcotiques étaient d'ailleurs prohibées en France et assimilées à la pratique de la sorcellerie. L'opium toutefois est encore utilisé au XVIII[e] siècle par Boerhaave et Sassard, mais la mère Barbier "pria tout le temps de l'opération" ce qui indique bien qu'elle était consciente. "C'est sur le stoïcisme[4] du patient aidé parfois de l'obnubilation par les fumées de l'alcool et plus encore sur une immobilisation par des entraves résistantes et des aides solides que le chirurgien doit compter pour mener à terme l'opération".

Il est à croire que la position donnée à la malade était celle adoptée au Jardin royal, à Paris. De même la technique opératoire devait être celle enseignée sans doute à Sarrazin[3] et toujours au Jardin royal. "Dans l'opération il faut situer la malade commodément pour elle et pour le

chirurgien, c'est-à-dire à demi couchée à la renverse; le bras du côté de la tumeur doit être élevé et porté en arrière, afin qu'elle paraisse davantage et que le muscle pectoral soit un peu retiré de dessous la tumeur. L'on en marque ensuite avec de l'encre toute la circonférence qui est l'endroit où l'on doit faire l'incision; puis l'on passe une aiguille courbe à travers le corps de la tumeur, elle est enfilée d'un cordonnet, dont on lie les deux bouts, et dont on fait une anse qui sert à soutenir la tumeur, et en la tirant à l'éloigner des côtes. Il serait inutile de passer l'aiguille deux fois, l'on peut épargner cette douleur, car l'on soutient aussi bien avec une anse simple qu'avec une courbe; puis, avec un rasoir ou un grand couteau que je trouve plus commode que le rasoir qui peut ployer dans l'opération, l'on coupe à l'endroit marqué et l'on enlève tout le corps de la mamelle en peu de temps; il se trouve plus de facilité dans cette opération que l'on ne s'était imaginé avant que de la faire; car la mamelle se sépare aussi aisément des côtes, que quand on lève l'épaule d'un cartier d'agneau".

La tumeur enlevée, l'on presse avec la main pour expurger le sang du pourtour. Si le saignement est trop abondant l'on met de petits boutons de vitriol sur les ouvertures des artères qui le versent. Par après la plaie est recouverte de plumaceaux faits d'étoupe et couverts de poudres astringentes incorporées avec le blanc d'œuf. On emploie aussi l'emplâtre diachalcitéos, une compresse et une serviette dont on fait une circulaire autour du corps. Dionis[1] ajoute que selon Helvetius, chirurgien hollandais réputé, on peut mettre sur la poitrine une serviette pliée en plusieurs doubles et trempée dans la bière et le beurre frais fondu battus ensemble.

Chose excessivement curieuse, dans un numéro récent du *Journal de l'Association Médicale Canadienne*,* Mustard, pratiquant des épreuves de coagulation du sang à l'aide de différentes substances graisseuses, déclare ce qui suit: "Le beurre est aussi actif que les thrombocytes ou l'extrait de cerveau dans l'épreuve de formation de thromboplastine".

INSTRUMENTS POUR L'OPERATION DU CANCER

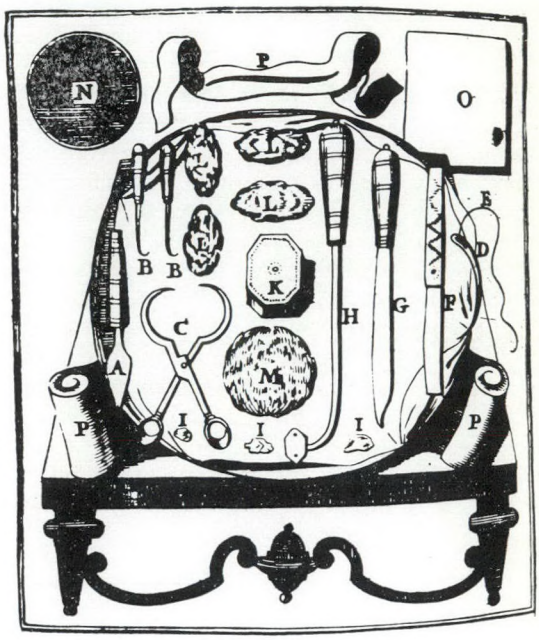

Fig. 7.

Les soins post-opératoires consistaient en une surveillance de la plaie et en applications d'onguents variés. La cicatrisation est lente à se faire, tant à cause de la figure ronde de la plaie que par la qualité de la tumeur qui a causé le mal et qui d'ordinaire est rebelle à toutes sortes de remèdes. Dionis[1] ajoute: "quand la plaie est cicatrisée, il ne faut pas discontinuer l'usage des remèdes internes pendant quelques années, de crainte qu'une nouvelle tumeur ne se jette sur quelque autre partie et ne fasse un nouveau cancer".

Comme conduite à tenir pour le malade après l'intervention on lui conseillera un bon régime de vie, on évitera les aliments acides; on conseillera plutôt une nourriture pleine de sel alcali volatil. Le malade doit respirer un air subtil. Le ventre sera tenu libre. L'on bannira tout sujet de colère, de chagrin et de tristesse; la tranquillité de l'esprit et la joie contribuent à une douce fermentation du sang, conseils qui sont encore très appropriés de nos jours.

Sarrazin[3] usa-t-il d'asepsie ou d'antisepsie? L'asepsie n'était sûrement pas connue à

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*In vitro and in vivo effect of different fat preparations on blood coagulation. J. F. Mustard—Journal de l'Association Médicale Canadienne, vol 79—Nov. 15, 1958, page 818.

ce moment (1700). Quant à l'antisepsie il est à croire qu'elle était rudimentaire. L'Hôtel-Dieu de Québec possédait depuis 1690 un aqueduc et il est vraisemblable de croire que le sein de la Mère Barbier fut lavé avant l'intervention, de même que Sarrazin,[3] qui devait opérer manches retroussées mais dans un costume ordinaire, se soit au moins lavé les mains. Les instruments employés devaient eux aussi être lavés—c'est probablement la seule antisepsie à laquelle on eut recours. Elle fut apparemment suffisante car le résultat de l'intervention fut des plus heureux malgré qu'il y eut alerte au cours de la convalescence. La sœur Barbier guérit très bien et vécut encore 19 ans après l'intervention.

Sarrazin[3] fit au moins deux autres interventions semblables pour cancer du sein l'une à la Mère Saint-Joseph, hospitalière de Montréal, l'autre à sœur Marie-Elisabeth Cheron, de Sainte-Anne.

Voici brièvement imaginé ce qui a dû se passer lors de cette première amputation du sein pour cancer au Canada.

Les illustrations qui accompagnent cet article sont tirées du "Cours d'opération de Chirurgie demonstrées au Jardin Royal" de Pierre E. Dionis, édition publiée à Bruxelles en 1708.

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6. Marie Barbier—Biographie—Rumilly, page 127.

CHIRURGIENS CANADIENS D'AUTREFOIS

CHARLES-MARIE BOISSONNAULT, *Ste-Foy, Qué.*

L'HISTOIRE DE LA CHIRURGIE en Canada se confond avec l'évolution de la civilisation dans notre pays. Dès 1640, Chomedey de Maisonneuve proclame que *celui qui était le plus nécessaire de tous,* le chirurgien qu'il amenait avec lui venait de périr; il rend un hommage éclatant à la profession. Pendant quelque cent cinquante ans, il n'y eut pas de médecins en Canada et, disent les moqueurs, on ne s'en porta pas plus mal. C'est qu'il y avait des chirurgiens. A cette époque, en effet, seuls les chirurgiens barbiers exerçaient l'art de soigner sur les rives du Saint-Laurent.

On sait que les Peaux Rouges "avaient leur chirurgie, simple et efficace, à laquelle leurs remplaçants avaient plus d'une fois forcément recours; ils traitaient les fractures et les contusions par les douches froides des sources et des ruisseaux, les blessures en suppuration et les ulcères avec l'écorce de l'orme rouge (*Ulmus fulva*), avec celle du tilleul (*Tilia Americana*) et l'écorce résineuse de l'épinette rouge (*Laryx Americana*), médicaments qui méritent bien le titre de bons émollients et de cataplasmes stimulants".[1]

C'était l'époque de l'orthopédie de fortune. On réduisait les fractures par les procédés les plus rudimentaires. Les chirurgiens, écrit le docteur Hingston, "remboitaient les membres disloqués avec force d'hommes et aussi un mouvement rotatoire, système ressemblant quelque peu à celui inauguré dans la profession par le célèbre chirurgien américain, Nathan Smith. Ils remettaient les fractures avec soin, et maintenaient des éclisses en cèdre ou genêt ingénieusement matelassées par les indiennes, avec des feuilles ou de l'herbe, solidement fixées autour de la partie malade avec des liens de jeunes bouleaux (genus *Batula*)".

Ainsi, la chirurgie existe depuis toujours sous une forme ou sous une autre. Sous la domination française, comme au cours du XIX^{e} siècle, elle se développe à Québec. Au milieu du siècle, quand s'ouvre dans la Capitale la première école de médecine, le président, le docteur Joseph Morrin, s'adresse aux étudiants en ces termes: "Mes jeunes amis, profitez de vos moments, et bientôt vous acquerrez l'expérience des années. On peut vous procurer ici tous les

moyens d'obtenir une éducation complète en médecine, qui pourront égaler et quelquefois surpasser le but de toute autre institution établie sur le continent d'Amérique". Cette idée se trouvait déjà, d'ailleurs, dans la charte de l'école.

Québec offre à cette époque de grands avantages à quiconque veut se spécialiser en chirurgie. L'Hôpital de la Marine et l'Hôtel-Dieu comptent de nombreux blessés. Des accidents de toutes sortes surviennent dans la région et le fait que les navires venus d'outre-Atlantique s'arrêtent tous dans le port de Québec contribue largement au peuplement de nos hôpitaux. Voici comment s'exprime à cet égard le docteur Joseph Morrin en inaugurant la nouvelle Ecole de Médecine: "Les progrès importants du Commerce et les avantages particuliers à Québec, comme port de mer, visité tous les ans par plus de 1,200 vaisseaux; la facilité de suivre la pratique de la Médecine et de la Chirurgie dans un hôpital qui contient plus de 300 lits, et reçoit plus de 1,500 malades pendant la durée de la navigation; l'avantage d'examiner et d'observer les cas, de connaître les maladies et d'en rechercher le siège, ainsi que les organes affectés; tout est propre à mettre cette école en état de répandre les connaissances les plus importantes dans la Chirurgie et la Médecine clinique. Il ne faut pas seulement considérer les nombreuses et importantes opérations de la chirurgie, ainsi que les moyens de les rendre profitables à l'élève; mais encore l'avantage de recevoir en français comme en anglais l'instruction clinique dans l'Hôpital de la Marine, et d'avoir accès à une bibliothèque composée des meilleurs ouvrages en Médecine, ce que semble devoir assurer l'importance et l'efficacité de l'école de Médecine de Québec."

A cette institution devait succéder en 1853 une autre école de médecine, celle de l'Université Laval fondée en 1852. Le doyen de l'école, le docteur Jean Blanchet, avait étudié la médecine et la chirurgie sous son oncle, le docteur François Blanchet, véritable savant si l'on tient compte de l'époque et de l'état des connaissances humaines en ce temps révolu. Blanchet après avoir acquis à Québec les principales sciences médicales alla à Paris parfaire ses études et se spécialisa en chirurgie sous le professeur Larrey à l'Hospice du Gros Caillou. Il fut également l'élève de Dupuytren à l'Hôtel Dieu de Paris. Revenu en Canada, il exerça son art avec compétence et succès, acquit une réputation enviable et bien méritée, c'est pourquoi il fut nommé doyen de la nouvelle Faculté de Médecine de l'Université Laval. Ce diplômé du collège royal des chirurgiens de Londres qui avait étudié sous Sir Astley Cooper, Blizard, Curry et Blundell fut un professeur remarquable et imprima à la nouvelle école de médecine une orientation qui en assura le développement et un esprit de travail qui dure encore.

Egalement ancien élève des institutions européennes, le troisième doyen de la Faculté de Médecine de Laval, le docteur James Sewell, détenait son grade du collège royal des chirurgiens de Londres. De 1833 à 1883, il exerça la chirurgie et la médecine à Québec, forma de jeunes praticiens et consacra la dernière partie de sa vie à l'œuvre de Laval, la Faculté de Médecine. Son successeur à ce poste, le docteur Alfred Jackson, détenait les mêmes titres et avait exercé son art dans la Vieille Capitale. L'Hôtel-Dieu, l'Hôpital de la Marine, l'Asile de Beauport le comptèrent au nombre de leurs médecins chirurgiens. Jackson enseignait l'obstétrique. Entre Blanchet qui professa la pathologie générale et la physiologie et le docteur Sewell se place Charles-Jacques Frémont, deuxième doyen de la Faculté de Médecine, premier professeur de chirurgie à Laval. La Faculté, l'Hôtel-Dieu et l'Hôpital de la Marine lui doivent de nombreuses améliorations de même que les différentes disciplines dont la médecine opératoire et l'enseignement clinique. Il avait été l'assistant favori de James Douglas qui réclamait son concours chaque fois qu'il entreprenait une opération. La présence du jeune Frémont lui inspire lorsqu'il exerce la chirurgie une grande confiance. Durant cette période de sa pratique, Douglas renonce à utiliser le tourniquet et affirme qu'il épargne ainsi le sang de ses patients. Le témoignage de Douglas en faveur de Frémont va encore plus loin. Il attribue au jeune chirurgien un grand nombre de ses succès et de ses cures. La vitesse avec laquelle Frémont manie le scalpel fait l'envie et l'admiration de ses

confrères. Aussi ne tarde-t-il pas à se créer une clientèle nombreuse qui dépasse les frontières de la Pointe-Lévis où il a d'abord décidé d'exercer son art. Rien d'étonnant par conséquent à ce que lors de l'établissement de l'Ecole de Médecine on fasse appel à l'expérience et à l'esprit scientifique de Frémont.

Tels sont quelques uns des chirurgiens qui ont présidé à l'établissement de l'enseignement de la médecine dans la ville de Québec. Ils avaient été précédés par un certain nombre d'autres dont François Blanchet, Zéphirin Nault, Marsden, James Douglas, Painchaud, Von Iffland. A cette époque, la médecine n'était pas aussi spécialisée que de nos jours et la piupart des médecins exerçaient la chirurgie en même temps que ce que nous appelons aujourd'hui la médecine générale. Il faut lire les comptes rendus de leurs opérations dans les rares publications médicales de l'époque pour se rendre compte de l'étendue de leur pratique, de la variété de leurs cures et des difficultés matérielles qu'ils rencontraient dans l'exercice quotidien de leur profession. Nous y reviendrons.

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UN OPÉRATEUR AMBULANT*

"Jacques Beaulieu, dit Frère Jacques (1651-1714), est un opérateur ambulant qui exerça une grande influence par sa *taille latéralisée*. Affublé d'habits religieux usagés, coiffé d'un vaste chapeau à larges bords, cet ancien soldat parcourut longtemps le Languedoc et le Roussillon, taillant pour quelques sous à peine. On le retrouve à Marseille, à Perpignan, à Besançon où il guérit un chanoine qui le recommande à un prélat parisien. Frère Jacques vient à Paris en 1697; il est alors âgé de quarante-six ans. Le prélat parle de lui à M. de Harlay; le premier président s'intéresse à l'opérateur, et les chirurgiens de l'Hôtel-Dieu sont chargés d'apprécier son talent. Il pratique son opération sur un cadavre, pauvre ambulant. On parle de notre homme à la cour; Duchesne et Fagon s'occupent de lui, et on l'autorise à opérer un malade, puis un autre, puis neuf autres, qui tous guérissent. Louis XIV apprend ces succès merveilleux et déclare qu' 'il faut avoir soin de cet hommelà'. Mais la chance de Frère Jacques eut une fin: des fistules survinrent, puis des morts. Méry fit un second rapport, aussi défavorable que le premier avait été élogieux et Frère Jacques reprit sa vie errante, en Allemagne et en Hollande. Cependant le bruit de ses nouveaux succès se répandit jusqu'à Paris. Fagon, qui avait lui-même une pierre vésicale, le fit revenir d'exil; il le logea chez lui, le fit opérer, l'aida, simplifia son instrumentation, et sa méthode fut codifiée en une brochure de huit pages. Un progrès considérable était accompli: une incision régulière, permettant d'aborder la vessie avec toute la sécurité souhaitable, remplaçait l'opération brutale du 'grand appareil'. Cependant Frère Jacques allait de succès en succès: il tailla et guérit trente-huit calculeux à Versailles et s'il n'opéra pas Fagon, il fut mandé par le maréchal de Lorges. Pour plus de sûreté, celui-ci fit opérer avant lui vingt-deux pauvres en son hôtel: tous les pauvres guérirent, mais le maréchal mourut!

"Après cet échec, que ses ennemis surent habilement exploiter, le pauvre Jacques n'avait plus qu'à reprendre sa vie vagabonde. On l'accueille triomphalement à Genève et surtout à Amsterdam. A Vienne, il est appelé par l'empereur; à Rome, le pape veut qu'on le lui présente. Il vient enfin se reposer dans son village natal et meurt chez les Bénédictins, laissant aux siens une modeste fortune. Il avait fait connaître la taille latéralisée dans toute l'Europe; mais, suivant le sort de la plupart des méthodes françaises, celle-ci fut abandonnée à Paris . . . et défendue à l'étranger."

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ORIGINAL ARTICLES

THE RECOGNITION AND MANAGEMENT OF TRAUMATIC RUPTURES OF THE TRACHEO-BRONCHIAL TREE*

EDOUARD D. GAGNON, M.D., M.S., F.R.C.S.[C], *Montreal*

ALTHOUGH TRAUMATIC TEARS of the human airway are rare in relation to the number of severe accidents today, these lesions will be of greater importance to surgeons and physicians if their early recognition permits curative treatment. I share the view of others[1] that tears and rents of the trachea and major bronchi occur much more frequently than any of us realize. Recognition of a complete tear in its early course can lead to definitive reparative surgery to the patient's benefit.[2] Early diagnosis of minor rents in bronchi can also lead to correct management and the avoidance of complications such as the so-called "wet lung syndrome"[3] or late sequelæ such as bronchiectasis, atelectasis or functionless lung.

Before developing the subject further, let us define our terms. We are speaking here of rents and tears in the tracheo-bronchial tree from closed injuries to the chest. We therefore leave aside the no less important chapter of penetrating wounds of the chest by missiles or such. Nor do we wish to discuss those cases in which peripheral lung parenchyma has been punctured by loose ends of ribs, where the cause of the localized subcutaneous emphysema is obvious.

The latter injuries, important though they may be, are less easily misdiagnosed and therefore usually better managed, especially after the work of Sanger[4] in 1945, drawing attention to the principles of their management.

As regards closed chest injuries, we also do not wish to discuss complete severance of main bronchi. These too have been written about and are being recognized today fairly early in their course. The syndrome in such cases is one of functionless lung with atelectasis preceded or not by pneumothorax.

*Lecture in Surgery presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Vancouver, January, 1959.

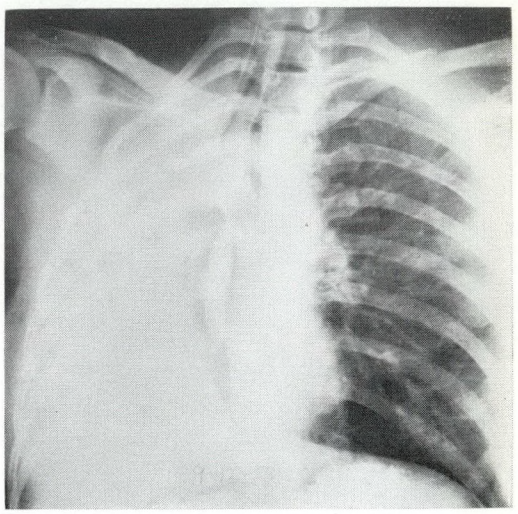

Fig. 1a.—Bronchogram 20 days after complete rupture of main bronchus, showing opaque material outside bronchus. (Figs. 1a and 1b reproduced by permission of Dr. L. R. LaFlèche—case to be published.)

An example of such an injury is one in which a young woman, involved in a car accident on November 18, 1958, showed no signs of intrathoracic lesions until 10 days later, at which time atelectasis was obvious, accompanied by progressive dyspnœa, a feeling of weight in the right chest and a hacking non-productive cough. When she was first seen by the thoracic surgeon 20 days after injury (Fig. 1a), these symptoms had increased, as had the atelectasis. Bronchoscopy at this time showed a main bronchus occluded by granulation tissue, and bronchograms showed a leak into the thoracic cavity. At operation (Fig. 1b) considerable scar tissue was found occluding the main bronchus, completely severed 1 cm. from its origin. The area was resected* and end-to-end suture with catgut of the freshened edges led to complete re-expansion and full recovery.

Such cases, we admit, are rare. More frequently, after chest injury and its concomitant increase in intrabronchial pressure against a closed glottis,[2] there occur partial

*Operation performed by Dr. L. R. LaFlèche—case to be published.

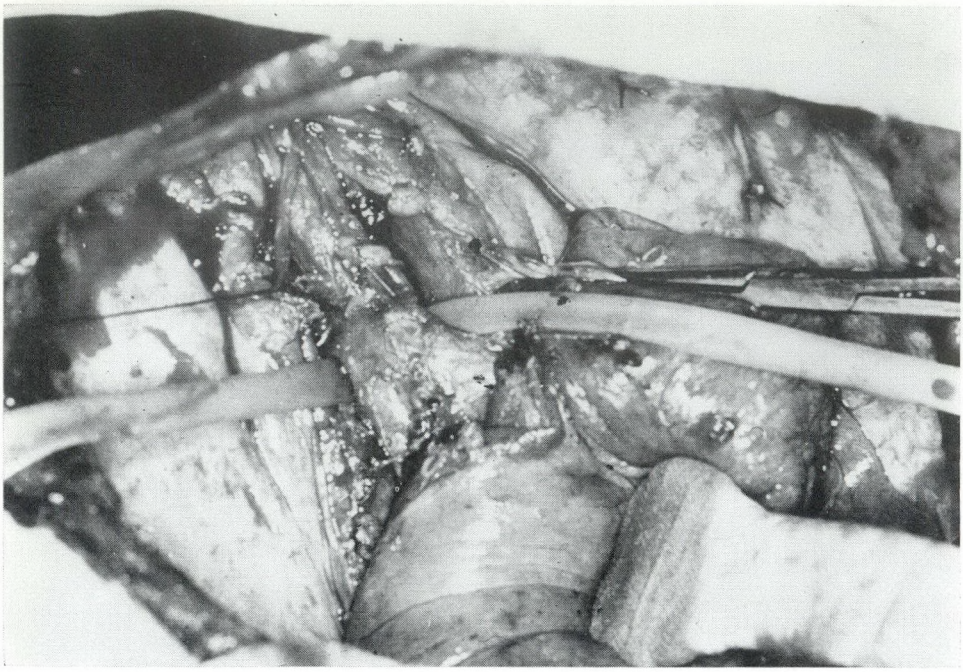

Fig. 1b.—Right main bronchus occluded by scar tissue for 1 in. (2.5 cm.) proximal to the Penrose drain. The lung is retracted downwards in the right lower corner.

tears in the human airway, and these will form the basis of our discussion from here on.

As a result of the great increase in intraluminal pressure, the point most likely to tear appears to be the thoracic trachea or a bronchus just beyond its bifurcation. The extent of the tear will of course depend on the pressure. Tremendous pressure will lead to complete severance of the bronchus, as we have just seen. Less severe pressures—and these are more common—will lead to partial tears. A gradual increase in intraluminal pressure will, according to Macklin,[7] lead to rupture, not of the bronchi but of the alveolar wall itself. This latter point was demonstrated by Macklin in his writings on the subject in 1937, 1938 and 1939.[5-7] Macklin furthermore demonstrated pathways followed by the extra-alveolar air (Fig 2a) creeping along vascular sheaths towards the hilum and bursting into the mediastinum or, secondarily, into the pleural cavity.

Whether the same path is followed when the rupture has occurred in a bronchus will be discussed below, but first let us examine the demonstration of the Macklin pathways.

Figs. 2a and 2b are taken from Macklin's work, with his kind permission. They show air from ruptured alveoli creeping along inside the vascular sheaths of the lung. Injections of carmine in a gelatin suspension demonstrated the points of rupture in the alveoli. The injury in these cases was occasioned by increasing intrabronchial pressure 2 cm. of Hg per minute over 6 minutes—by introducing air through a ureteral catheter in the lower lobe bronchus of a cat. The final pressure reported was 12 cm. of Hg. and Macklin states that at that time the cat showed air in the mediastinum, in both sides of the chest, in soft tissues, and, in some cases, in the peritoneum.

We have repeated Macklin's work in the cat, but instead of injecting air into a lobar bronchus with a ureteral catheter as he did, we injected it into the trachea through an air-tight endotracheal tube. With such a preparation, the subcutaneous emphysema starts to appear at a pressure of 3 cm. of Hg and is so pronounced at 4 cm. pressure that one feels that the cat will explode. If injection is discontinued and the animal examined, air is found everywhere includ-

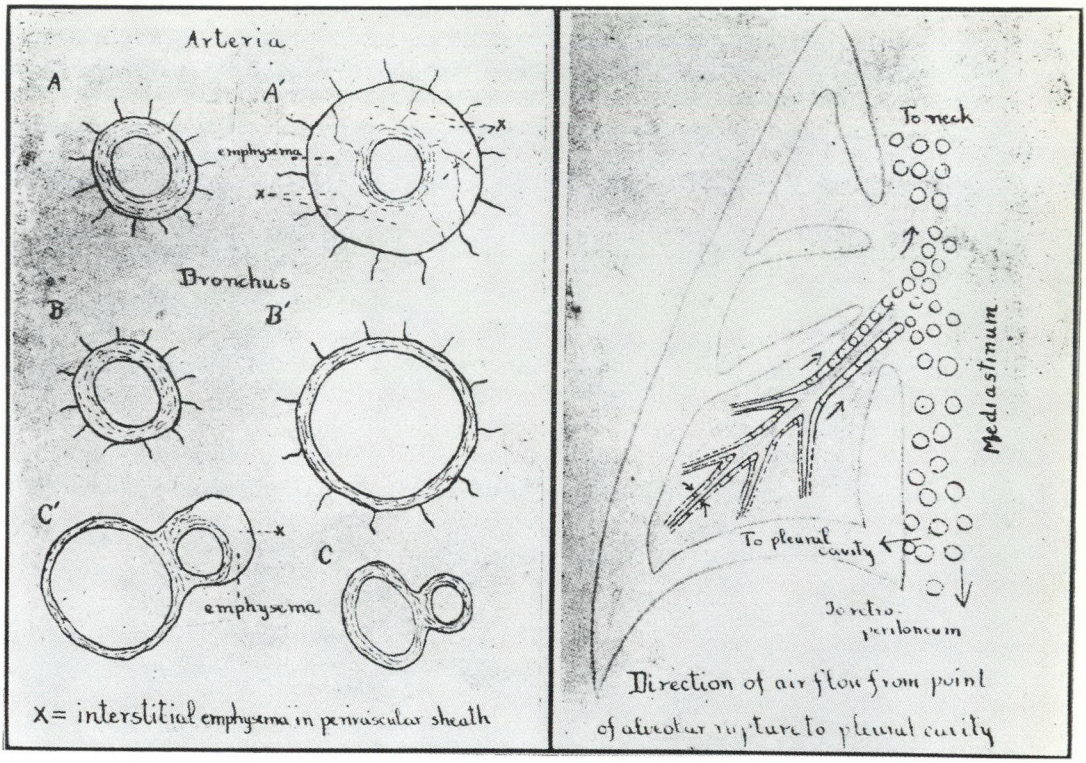

Fig. 2a.—The vascular pathways as described by Macklin, showing progression of air along bronchial vessels towards mediastinum. (Figs. 2a and 2b reproduced by permission of Dr. C. C. Macklin.)

ing the pericardial sac, peritoneum and soft tissues.

Fig. 3 is a photomicrograph of a Macklin preparation as carried out by us. It shows a bronchus with no air around it, a vessel with air in its perivascular sheath, and areas

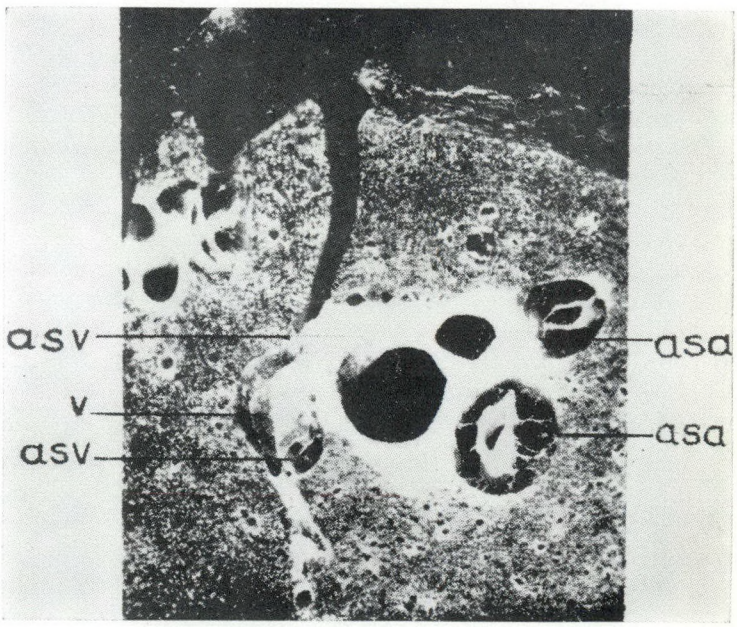

Fig. 2b.—**asa**—periarterial air in sheath of bronchial arteries; v—vein cut tangentially; **asv**—perivenous air in sheath. In centre, two bronchi with no air. Cat preparation (see text). (Macklin, C. C.: *Arch. Int. Med.*, 64: 913, 1939.)

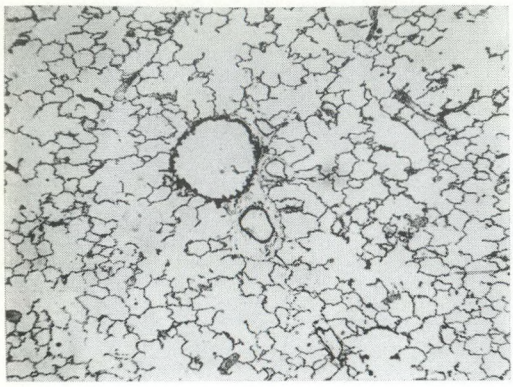

Fig. 3.—Photomicrograph of cat preparation following air introduction at 4 cm. Hg pressure into trachea with cuffed tube. *Note:* air in perivascular sheath; no air around bronchus; multiple areas of ruptured alveoli.

of emphysema with ruptured alveolar spaces. This photomicrograph therefore tends to corroborate Macklin's work, and confirm the existence of perivascular pathways towards the hilum in the cat.

It is noteworthy, however, that in our experiments air appeared subcutaneously at one-third the pressure reported by Macklin in 1937. This is probably due to the airtight closure of the tube in the trachea as opposed to Macklin's open ureteral sound in a lobar bronchus, or to the much smaller resistance of a large-bore, short endotracheal tube; possibly both these mechanisms are in play.

In the course of this study, a stillborn infant's lung was brought to our attention after intense efforts at resuscitation with a cuffed endotracheal tube had been made by the anæsthetist. We have no record of the pressures exerted, but have found that manual compression of the anæsthetic bag will give minimum pressures of 10 cm. Hg. With vigorous compression, the pressures may be 20 or 30 cm. of Hg. The infant came to autopsy with marked air extravasation in the soft tissues, peritoneum, chest, mediastinum and pericardium and bilateral collapse of the lungs, quite comparable to a Macklin cat preparation.

Using the same intrabronchial fixation method as in the cat (Fig. 4a), we could demonstrate air passing along from the visceral pleura towards the hilum, and progressing along interlobular septa, along vascular pathways and around the bronchi (Fig. 4b).

If the experiment is repeated, using pressures reported by Macklin (namely, 12 cm. of Hg) with an airtight tube in the trachea of a cat, thereby delivering the full pressure to the lungs, the microscopic picture (Fig. 5a) appears to be very similar to that in the human lung with air around the bronchi as well as around vessels. More work needs to be done at higher pressures, but it would appear reasonable to expect that a further increase in pressure would produce a comparable picture to that in the human lung.

As a control experiment, air under a pressure of 10 cm. of Hg was blown sud-

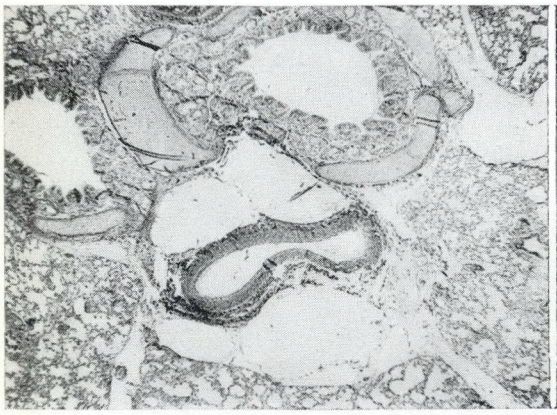

Fig. 4a.

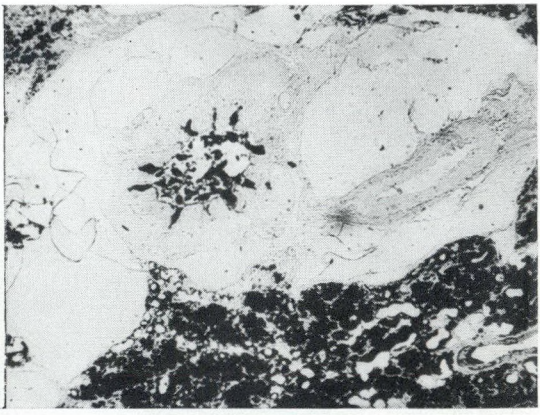

Fig. 4b.

Fig. 4a.—Infant lung preparation. Air in interlobular septa, around vessel (compressing it) and around bronchus. **Fig. 4b.**—Infant lung preparation with intrabronchial India ink injection. Demonstrates air around vessel *and* bronchus.

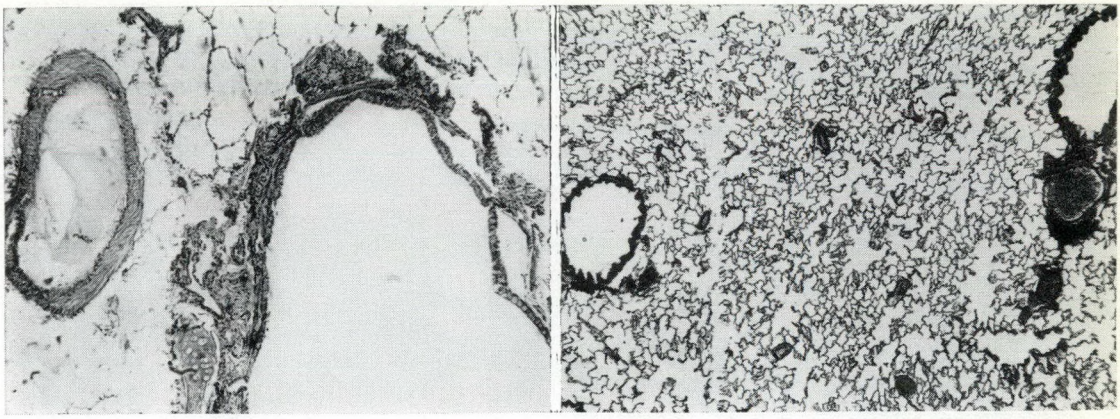

Fig. 5a. Fig. 5b.

Fig. 5a.—Cat preparation with air introduction at 12 cm. Hg. Air in perivascular sheath (left) and breaking into bronchial wall (right). **Fig. 5b.**—Cat preparation, control experiment: air under 10 cm. Hg introduced into œsophagus (see text). No perivascular or peribronchial air. Considerable congestion in vessels.

denly through a cuffed endotracheal tube into the œsophagus of a cat. This led to the same gross picture of death of the animal with marked subcutaneous, mediastinal, pleural and abdominal filling with air; on opening the chest, both lungs were collapsed. The same intrabronchial fixation technique revealed, however, no air in the perivascular sheaths or subpleurally (Fig. 5b). Instead, the blood vessels were engorged with red cells, and some red cells had extravasated into the connective tissue —evidence, we think, of extraluminal obstruction to pulmonary flow with extravasation of the fluid elements of the blood and some of the red cells.

It would appear, therefore, that ruptures of alveoli or bronchi can cause air to progress along these pathways — guided towards the hilum by the sheaths of covering tissue. This covering tissue in the human subject is the visceral pleura which runs along the secondary bronchi and vessels and delimits the segmental divisions of the lung.

With this experimental work in mind, we can now move on to clinical application of the principles involved.

Types of Injury

Many kinds of accidents can occasion tears in the human intrathoracic airway. The most important are automobile accidents, especially when the chest is suddenly and rapidly compressed, as when a driver of a car comes suddenly to rest on his steering column at the moment of impact. Another common cause for sudden thoracic compression is a fall from a height, such as a fall from a scaffolding, the victim landing upright or on his chest. A third cause is a severe glancing blow on the chest from a heavy weight. These accidental events occasion a sudden and marked rise in intrathoracic pressure, whether the compression be horizontal (as in the case of the automobile driver) or vertical (as in a fall from a height) or oblique (as in the glancing blow from a heavy object). Obviously, if the pressure is extreme, major vessels will rupture and this will cause death. Our civic morgues testify to these severe injuries after every summer weekend. Occasionally the trauma may be severe enough to split lung parenchyma neatly, as with an axe—and such cases have been encountered.

Fig. 6a, for instance, is the radiograph of a man who was brought to hospital after a glancing blow to his upper right chest by a heavy crane. His admission film shows a right pneumothorax, but a few hours later (Fig. 6b) there is a large collection of intrathoracic fluid as well. As time went on, shock deepened and immediate thoracotomy (Fig 6c) showed a partial rupture of the upper lobe bronchus at its origin, with an upper lobe split open as if with a

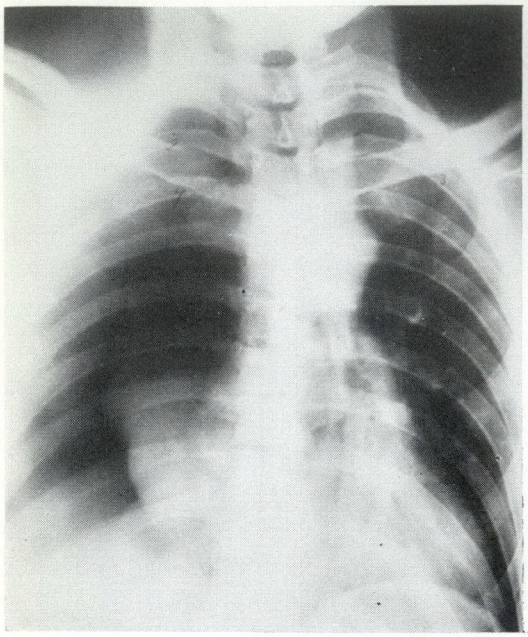

Fig. 6a.

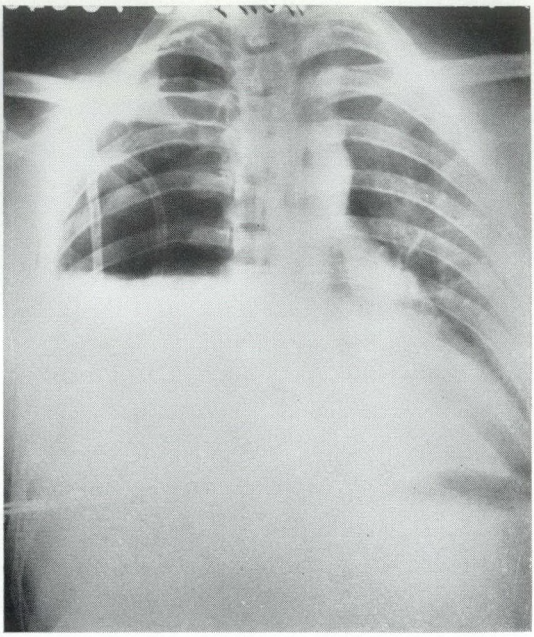

Fig. 6b.

Fig. 6a.—Admission radiograph showing tension pneumothorax on right side. **Fig. 6b.**—A few hours later, a large collection of fluid and air, as shock deepens.

meat cleaver. There was active bleeding from the lobar artery and veins. An upper lobectomy saved the patient's life and he remains well three years later (Fig. 6d).

An intercostal drainage in the presence of such a rapidly progressing fluid collec-

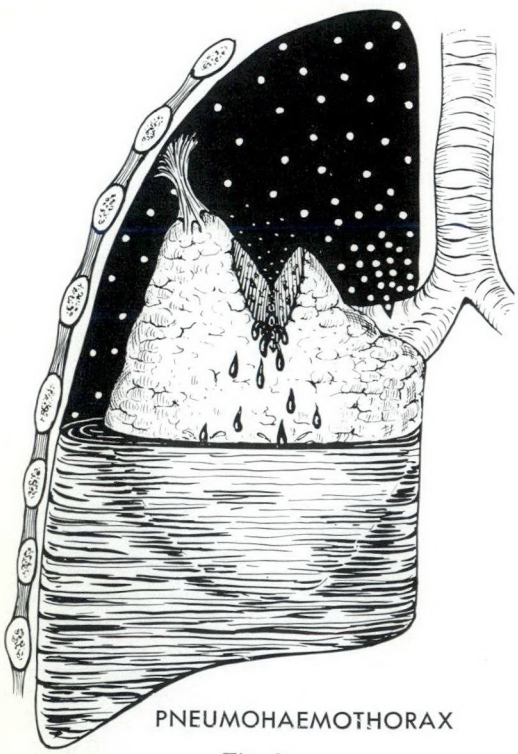

Fig. 6c.

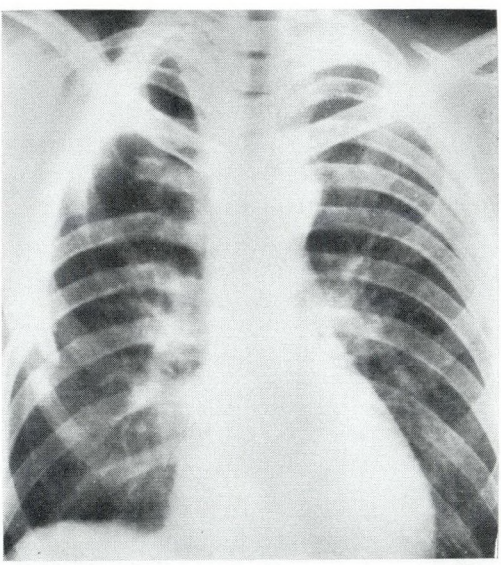

Fig. 6d.

Fig. 6c.—Artist's sketch of operative findings, showing the air leak from a partial rupture of the upper lobe bronchus, and the hæmorrhage from a rupture of parenchyma down to and including the lobar vein. **Fig. 6d.**—Chest radiograph three years after lobectomy. Remains well.

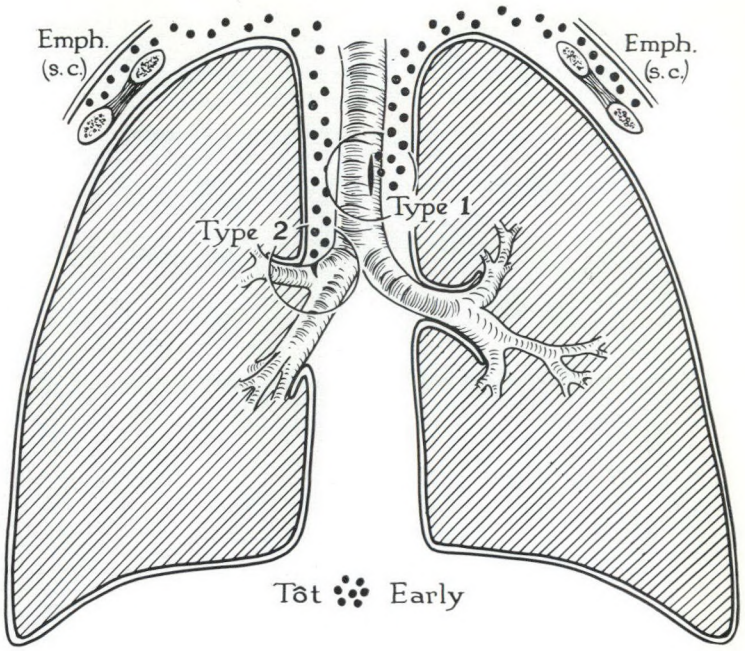

Fig. 7a.—Diagram showing tears of Types 1 and 2 into mediastinum *before* the point of pleural reflection (see text). Black dots represent early leakage of air.

tion might have led to a fatal outcome because of the active bleeding, and we have seen examples of this.

In these instances the trauma has been severe enough to occasion bursting of a bronchus as well as lung tissue, with active bleeding. It is a common enough finding in autopsies of accident victims. Those that survive long enough to reach hospital should be treated actively.

If, however, the end result of the increased pressure is a partial tear limited to the airway alone, one will be faced with two distinct clinical groups according to the site of the injury, the extent of the tear, and the state of the connective tissue coverings.

The symptoms and signs of the tears in each group can possibly be best understood by diagrammatic illustration. Fig. 7a is a diagrammatic and simplified representation of:

1. The tracheo-bronchial tree
2. The lung
3. The parietal pleura
4. The visceral pleura.

In the first group the tears will occur before the point of pleural reflection, which is that point at the hilum where the visceral becomes the parietal (or mediastinal) pleura. Ruptures here are wholly in the mediastinal space, whether the tear is in the trachea (Type 1) or in a main bronchus (Type 2).

The clinical picture is impressive, for the patient's physical features are very distorted by the rapid spread of subcutaneous emphysema. This has led to the descriptive term in the literature of "puff-ball man". There is usually no accompanying pneumothorax as the air diffuses out of the upper thoracic outlet and spreads along fascial planes, even to the toes.

Emergency therapy, in the form of mediastinal decompression by a collar of needles, is usually sufficient to allow time for moving the patient to the operating room where bronchoscopy is carried out. Endoscopy will serve to localize the side, extent and site of rupture: once this is decided, an immediate thoracotomy and suture of the defect can be carried out. In this way return of normal function is facilitated, recovery is accelerated, and no late sequelæ should occur.

Some of these patients recover without this form of therapy. However, it is our feeling that recovery is much delayed by conservative management at the expense of a grave risk of progressive mediastinitis and death, or, in the event of recovery, protracted morbidity from tracheal or bronch-

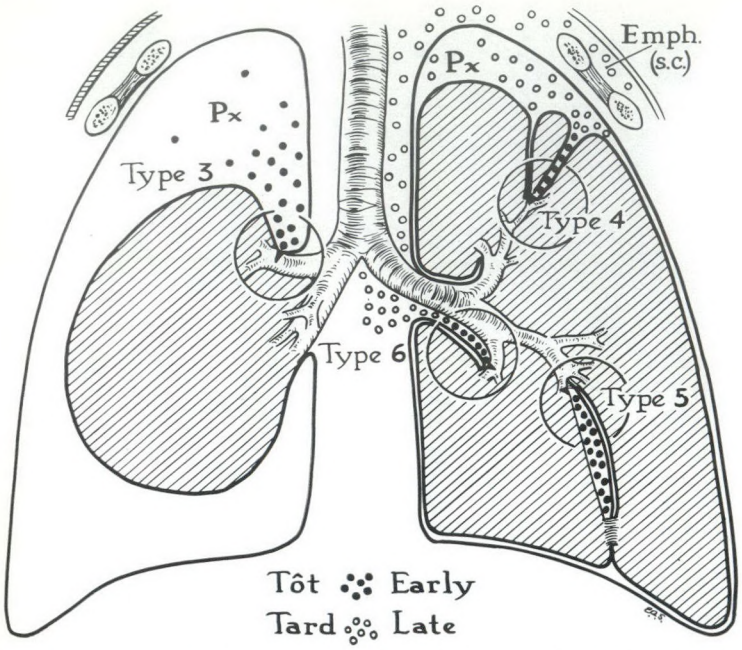

Fig. 7b.—Diagram of four types of partial bronchial tears occurring *beyond* the point of pleural reflection. Black dots represent early leakage of air, and open dots late leakage. See text for clinical picture of each type.

ial stenosis, with bronchiectasis and so on.

The second group (Fig. 7b) consists of four types, all occurring beyond the point of pleural reflection and therefore intrapleurally. The first possibility in this group (Type 3) is a partial tear of a major bronchus and its covering visceral pleura occurring at the same time. There is then an immediate pneumothorax which may become a positive pressure pneumothorax if the tear acts as a one-way valve. The symptoms therefore will be those of the pneumothorax, and emergency treatment, after bronchoscopy, is aimed at relief of this by installation of underwater-seal drainage. Thoracotomy may be delayed for a few days and is indicated *only* in the absence of re-expansion. Bronchial repair or lung excision is then performed according to the findings.

When, however, the initial tear (Type 4) has been small and in a secondary bronchus, leakage of air can continue unobserved as a subpleural accumulation under the visceral pleura. After several hours, a secondary rupture into the pleural space can lead to a *late* pneumothorax. Treatment is once again aimed at the pneumothorax, and thoracotomy is not usually required, as the repair of the minor tear is spontaneous.

Another possibility in this group is a self-limiting interlobar emphysema (Type 5). This may occur when the visceral pleura is adherent to the bronchus and the bronchial tear small—so that the visceral pleura can only be dissected away partially. This interlobar emphysema is usually reabsorbed slowly. Therapy will consist only of making sure of an adequate, dry airway by aspiration, bronchoscopy or tracheotomy if necessary, aimed at avoiding the occurrence of a "wet lung". If a rupture into free pleura takes place secondarily, the result will be a late pneumothorax as in Type 4 already discussed.

The final possible tear (Type 6) is one in which the bronchial or alveolar leak acts as a one-way valve and the visceral pleura is not adherent. The air will then dissect progressively along the air pathways and finally burst into the mediastinum after a certain number of hours. In such a case, the patient may have been considered to have a minor chest contusion or minor rib fracture until the sudden appearance of marked subcutaneous emphysema around the face, neck and body many hours after the accident. The symptoms become those of the "puff-ball man" as in Types 1 and 2, but the treatment will be different. Once the tear is recognized and localized, all that is required in most patients is tracheotomy (Table I) to maintain a clear airway, diminish the dead space and decrease the

intraluminal pressure. The bronchial lesion usually heals spontaneously and the air gradually clears.

TABLE I.—Tracheotomy in Tracheo-Bronchial Tears

Effect	*Result*
1. Decreases intraluminal pressure	(a) Prevents escape of more air out of tree (b) Prevents extension of tear
2. Decreases dead space	Increases effective ventilation
3. Makes removal of secretions easy	Prevents occurrence of "wet lung"

We have encountered examples of these different types of lesions over the past eight years, and feel sure that many more partial tears go unrecognized. The basis for their recognition is early bronchoscopy in all cases to visualize and localize the tear. Bronchoscopy has the added advantage of cleaning out the airway of blood and secretions and thereby hastening recovery. We strongly urge endobronchial examination in all cases of traumatic chest injuries, especially if any subcutaneous emphysema or pneumothorax is present. This is the only means of obtaining a true picture of the frequency of tracheo-bronchial tears.

Fig. 8, for example, is an illustration of the first type of tear. A 22 year old taxi driver, seen one hour after a head-on collision, was conscious but markedly cyanosed and dyspnœic, with a typical "puff-ball" appearance.

The x-ray revealed no fractures and no pneumothorax, but extensive air in mediastinum and soft tissues. A rupture of the airway above the pleural reflection was suspected, and a collar of multiple needles was installed. Suction on these needles stopped the progression of the emphysema. The patient was then taken to the operating room and anæsthetized. Before intubation a bronchoscopy visualized the tear, which was a Type 1 rupture, located in the distal 2 in. of trachea on the right wall. A right thoracotomy led to the lesion which was a vertical "flapping" tear at the junction of membranous and cartilaginous portions approximately 2 in. in length. It was repaired by interrupted catgut sutures and the chest was closed. The subcutaneous emphysema gradually disappeared over the next 10 days and by that time the patient could hardly be recognized as the same man. He left hospital after 18 days. He was seen again recently, and

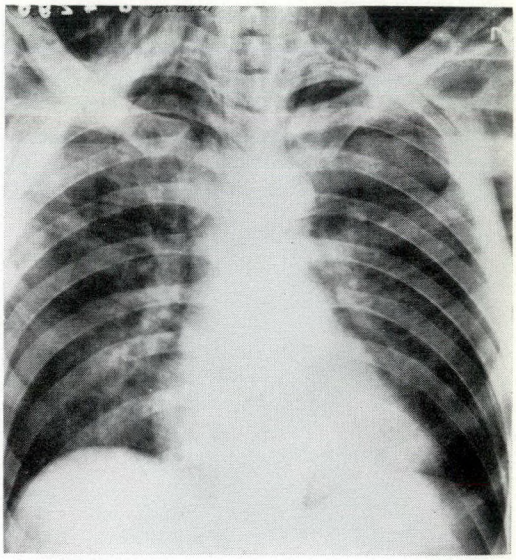

Fig. 8.—Admission radiograph of tear of Type 1 (trachea), showing marked subcutaneous emphysema and no pneumothorax.

remains well after seven years with a normal tracheo-bronchial tree.

Another example of the same type is illustrated in Fig. 9a which shows the typical "puff-ball man" appearance. We are grateful to Drs. Venne and Laurier for the details of this case, which they managed.[10] The Type 1 tear in this man was even more severe than the previous one. There was a complete "window" lesion of the right wall of the trachea. The fragment of trachea, lying loose in the mediastinum, was re-sutured in place, and prompt and full recovery took place. In Fig. 9b it is difficult to realize that this is the same man, yet it is the same patient 15 days later.

On the other hand, rupture of the bronchus just beyond the pleural reflection will give a picture of early pneumothorax (Type 3). Fig. 10 shows such a case.

A 30 year old truck helper was riding in his vehicle when it struck another large construction truck. He was thrown out of his cab and landed on the wheel of the truck ahead of him, striking his left chest. He immediately felt pain in the chest, became short of breath, and was brought to hospital.

On arrival he was markedly dyspnœic, with mild shock and rapid pulse. The x-ray revealed left pneumothorax. Continuous suction drainage was applied over the next five days. Control radiographs (Fig. 10b) having revealed no expansion, a thoracotomy was undertaken (Aug. 31, 1953) with a preoperative diagnosis of rup-

Fig. 9a.

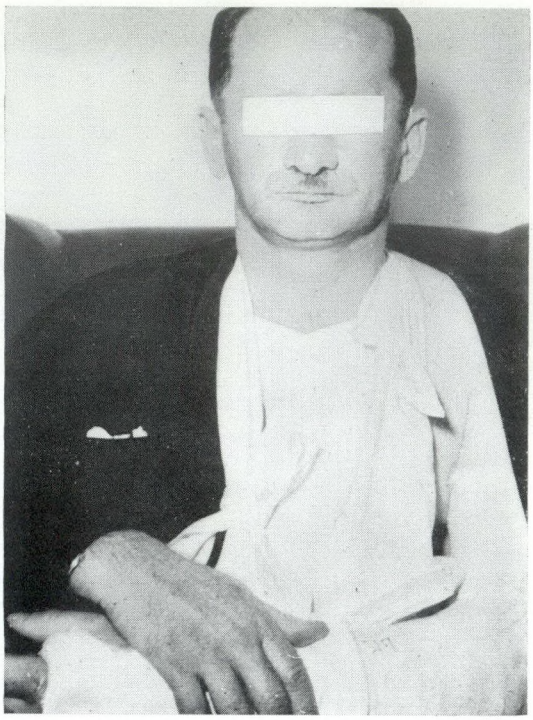

Fig. 9b.

Fig. 9a.—Typical "puff-ball man" appearance in Type 1 tear (trachea). **Fig. 9b.**—Same patient 15 days later. (Reproduced with the permission of Drs. Laurier, Messier and Venne[10].)

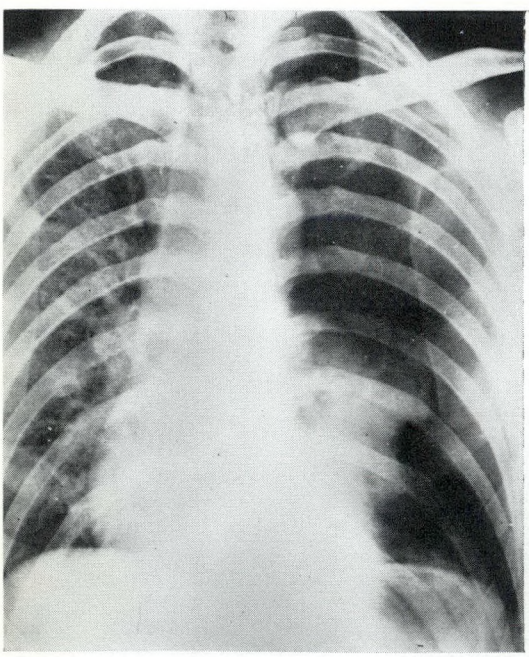

Fig. 10a.

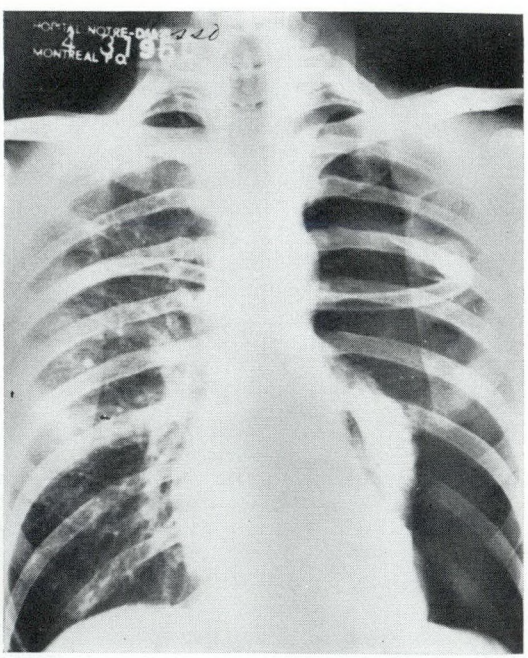

Fig. 10b.

Fig. 10a.—Left pneumothorax in Type 3 tear (see Fig. 7b) immediately after injury. **Fig. 10b.**—Same case after five days of underwater seal drainage and suction. Thoracotomy needed for repair of tear.

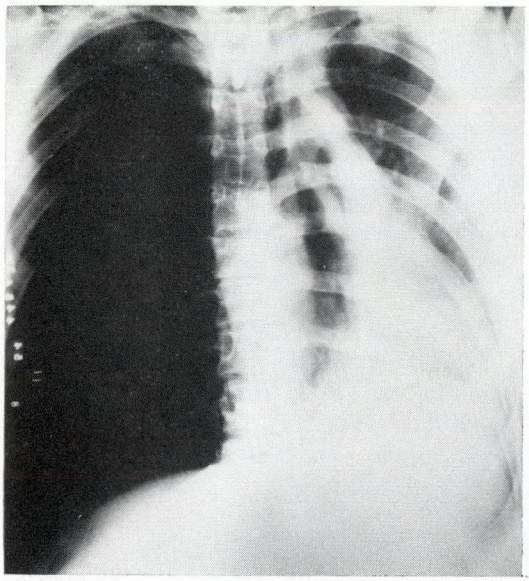

Fig. 11a.

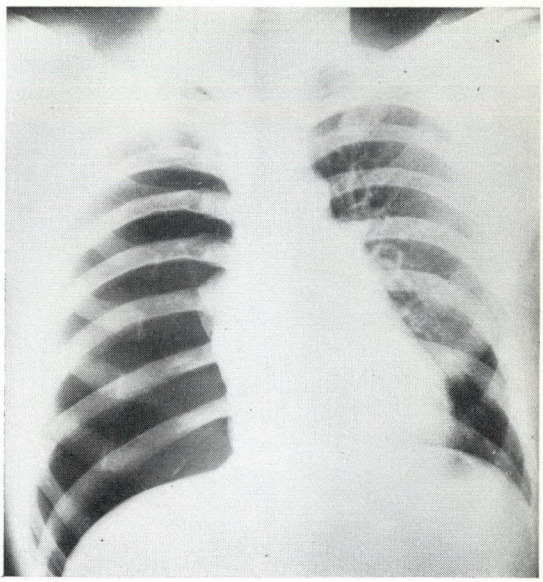

Fig. 11b.

Fig. 11a.—Late tension pneumothorax in Type 4 injury (see Fig. 7b). **Fig. 11b.**—Pneumothorax after initial aspiration.

tured left bronchus beyond the pleural reflection (Type 3 of diagram) with early persistent pneumothorax.

After suture of the bronchial leak, the lung expanded fully. The postoperative course was uncomplicated and the patient left hospital 10 days later. He has remained well for five years.

This type of partial tear gives a clinical picture different from that encountered in complete severance of the main bronchus, as pointed out earlier. It will be recalled that, in complete severance of the bronchus, the lung is completely atelectatic with an

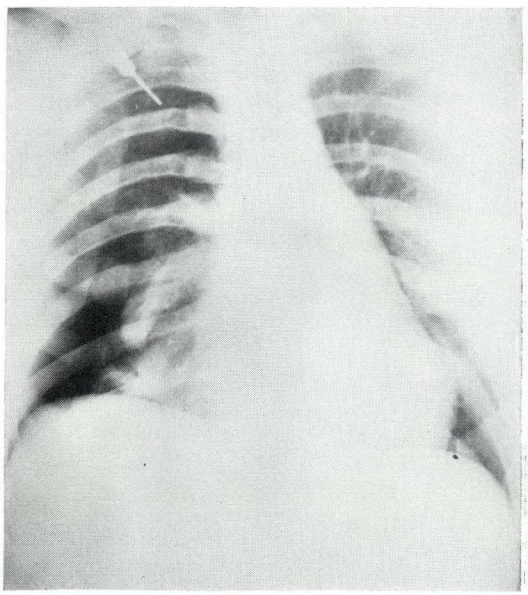

Fig. 11c.

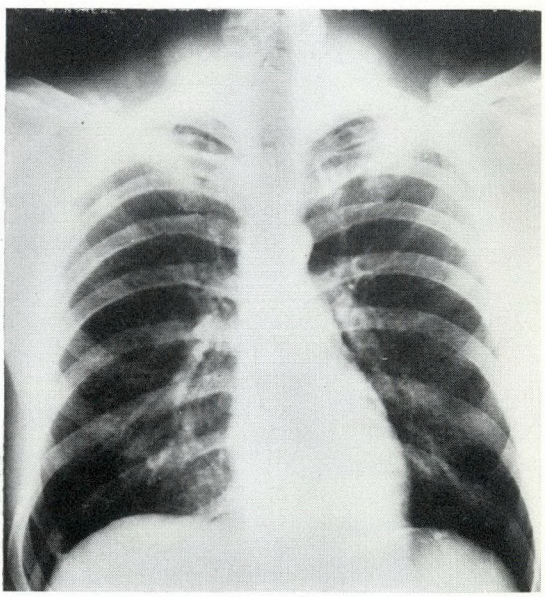

Fig. 11d.

Fig. 11c.—Beginning re-expansion with underwater seal drainage and suction. **Fig. 11d.**—Complete re-expansion after several days of suction. Remains well four and a half years later.

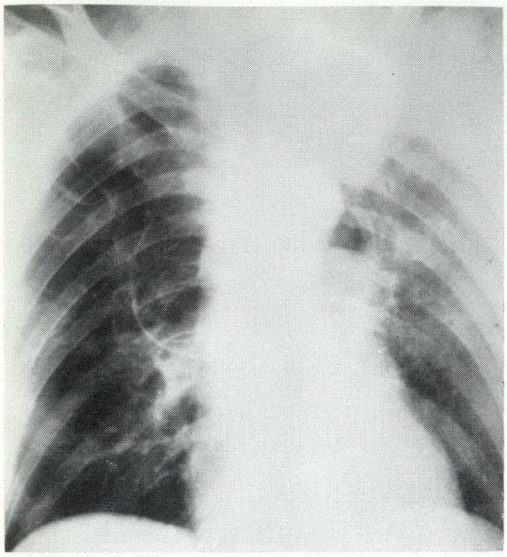

Fig. 12—Interlobar air in Type 5 tear of bronchus (see Fig. 7b). See text.

intrapleural collection of fluid and usually no persisting pneumothorax.

Type 4—or the tear with late pneumothorax—is illustrated by the following history:

On a Saturday evening, a 25 year old man appeared in our emergency department after an automobile accident. He had suffered a small laceration to an eyebrow, which was sutured, and he complained of some vague chest pain. This was not considered serious enough, after examination, to warrant x-ray examination, and he was sent home and asked to return on Monday morning for a check-up. The following day, he experienced chest pain and coughed up bloodstreaked sputum.

On Monday, while getting ready to return to hospital, he was suddenly seized with a severe pain in the right chest and immediately became short of breath. On arrival in hospital, he was breathing rapidly, with rapid pulse and cyanosis. The radiograph of the chest (Fig. 11a) showed massive tension pneumothorax.

After visualization of a partial tear in the upper lobe bronchus, and initial aspiration of air, an intercostal water-seal drainage and continuous suction were maintained for several days (Figs. 11b and 11c) and complete re-expansion was obtained (Fig 11d). No tracheotomy was required, as the airway remained dry. The patient has remained well for four and one-half years.

The fifth type of tear gives the type of picture shown in Fig. 12 with interlobar collection of air. The bronchial tear in this patient was visualized on one side only, though the x-ray picture would lead us to suspect tears on both sides. He had multiple other injuries; since the interlobar extravasation was self-limiting, no active therapy for this was required. Needless to say, some tears of this type will be missed even on bronchoscopy because the rupture has occurred in a secondary bronchus beyond the reach of the examining instrument, or in alveoli.

The last type (Type 6) is well shown by the case of a 58 year old man admitted after a fall of 17 feet from an exterior staircase. On admission, he was unconscious but recovered slowly over the next hour. On awakening he complained of severe pain in the left chest, roughly in the area of his heart. No subcutaneous emphysema was noted. The radiograph revealed fractures of ribs 4, 5, 6, 7, 8 and 9 on the left only (Fig 13a).

He was kept in hospital. Next morning early, during a coughing episode, marked subcutaneous emphysema suddenly appeared over neck, face and body, with laboured breathing and increasing cyanosis (Fig 13b). He had suddenly become a "puff-ball man".

Bronchoscopy revealed a tear in the superodorsal bronchus of the left lower lobe. Tracheotomy was performed to abolish dead space and prevent atelectasis and smooth recovery occurred

TABLE II.—Summary of Types of Tears, Usual Sites, Symptomatology and Treatment

Type	*Site*	*Symptoms*	*Treatment*
1	Trachea	"Puff-ball" early	Thoracotomy
2	Mediastinal br.	"Puff-ball" early	Thoracotomy
3	Main br.	Pneumothorax-early	(a) Water-seal drainage (b) Thoracotomy } if needed
4	Lobar br. or	Pneumothorax-late	(c) Tracheotomy } if needed
5	Segmental br.	Inter-lobar emphysema	Tracheotomy
6	Segmental br.	"Puff-ball"-late	Tracheotomy

Bronchoscopy in all cases—early.

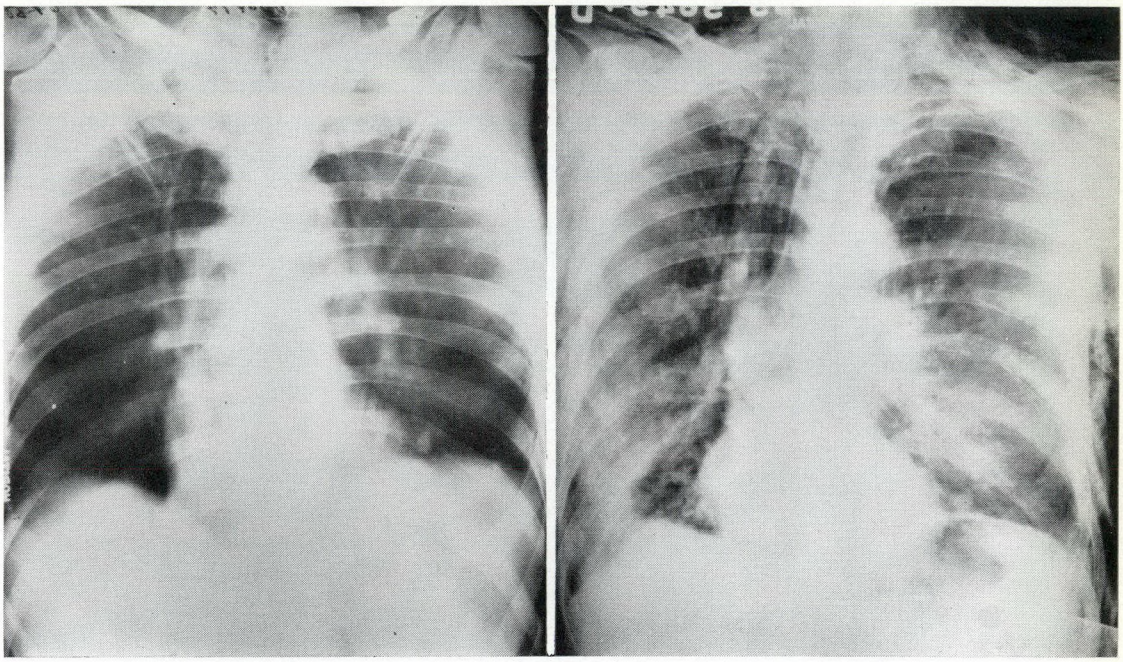

Fig. 13a. Fig. 13b.

Fig. 13a.—Admission radiograph showing only undisplaced fractures of 4th to 9th ribs on the left (poorly shown on illustration). **Fig. 13b.**—Radiographic appearance 18 hours later after sudden development of "puff-ball" appearance. No pneumothorax, but marked subcutaneous emphysema. Type 6 tear (see Fig. 7b) visualized in supero-dorsal segment of left lower lobe.

in 10 days. Discharged well 17 days later, he went back to work after two months.

Table II summarizes the six possible types of partial tear limited to the tracheobronchial tree. The two first types, before the pleural reflection, dramatic in onset and threatening life, require immediate major treatment. The four other types require more minor procedures and careful watching, but in many cases resolve spontaneously in about two weeks.

Most important is the proper recognition of the type and site of injury—based on four things: (1) the history of the injury; (2) the clinical examination; (3) the x-ray findings; (4) the bronchoscopic visualization of a tear.

Bronchoscopy is all important and must be performed as soon as possible after the accident, if necessary in the operating room prepared for thoracotomy or tracheotomy as the case may be. We feel strongly that there are no contraindications to the examination, indeed it may often be life-saving in a gravely ill patient. It has the advantage of clearing out the bronchial tree of accumulated blood and secretions; oxygen can be carried to the cleared-out lung parenchyma, and most important of all, the exact area and extent of injury can be ascertained so that adequate therapy can be carried out.

Summary

Certain aspects of accidental partial tears in the human airway are described. Pathways of air transport along vascular and bronchial sheaths are discussed. Proper recognition and localization of these tears are stressed, with reference to six different types of injury, and the modifications of treatment inherent to each type are mentioned.

All cases of chest trauma should be watched carefully. Bronchoscopy should not be withheld. A minimal amount of air in soft tissues, mediastinum or chest, and a stable clinical condition initially, can give place to quite rapid deterioration after a coughing episode.[8] Obviously, not every case of chest trauma or subcutaneous em-

physema will follow these patterns. However, when first seen, it is not possible to predict what course will be followed; for this reason we think it important to take steps to recognize the lesion, so as to manage it properly.

ACKNOWLEDGMENTS

I wish to thank Dr. S. Lauzé of the Department of Pathology, Notre Dame Hospital, Montreal, for his assistance with the histological work.

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The photomicrographs were prepared by the Department of Pathology, Notre Dame Hospital (Dr. L. C. Simard, Director).

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RÉSUMÉ

La rupture de la trachée et des bronches souches se produit plus fréquemment qu'on ne le soupçonne. La guérison dépend d'un diagnostic précoce. Le présent travail ne porte pas sur les lésions ouvertes du thorax qui sont causes évidentes d'emphysème sous-cutané mais plutôt sur celles qui peuvent passer inaperçues au début à cause de l'intégrité des téguments. La rupture complète d'une bronche produit une atélectasie, précédée ou non de pneumothorax. Lorsqu'il se produit une augmentation considérable de la pression à l'intérieur des voies respiratoires le point le plus faible et celui qui cédera le premier semble la trachée thoracique ou les bronches souches tout près de la bifurcation. L'étendue de la rupture sera en rapport avec la pression exercée. Macklin a démontré qu'une augmentation graduelle de la pression amène non pas une rupture des bronches mais de la paroi alvéolaire elle-même. Les expériences de cet auteur sur le chat ont été répétées avec une légère modification de technique portant sur l'emploi d'un cathéter endotrachéal au lieu d'une sonde insérée dans la bronche. Alors que la présence d'air dans le médiastin devient évidente à une pression de 12 cm. Hg avec une sonde intrabronchique, l'emphysème sous-cutané se manifeste à une pression de 3 cm. Hg lorsque la pression est exercée sur la trachée-artère. Au cours d'une autre expérience où l'on a insufflé rapidement une pression d'air de 10 cm. Hg à l'aide d'un cathéter dans l'œsophage d'un chat, l'auteur du présent article a constaté une infiltration d'air dans les tissus sous-cutanés, médiastinaux, pleuraux et abdominaux. L'air semble s'échapper le long de la plèvre viscérale pour atteindre le hile.

Les accidents qui peuvent causer une augmentation soudaine de la pression intra-thoracique sont les collisions d'automobiles où le conducteur est soudainement projeté sur son volant, les chutes d'une grande hauteur (la victime tombant sur ses pieds ou sur sa poitrine) ou les coups portés par un objet très lourd et effleurant la poitrine pendant un instant. Si le choc dépasse une certaine violence les gros vaisseaux seront sectionnés et la mort suivra immédiatement. Si le parenchyme pulmonaire est rompu ou broyé il y aura hémorragie plus lente. Dans les cas d'emphysème sous-cutané l'aspect du malade est impressionnant et évoque un "homme soufflé". Le traitement d'urgence consiste en une décompression par l'application d'un licou d'aiguilles. Une fois la lésion localisée grâce à l'endoscopie, une thoracotomie immédiate s'impose avec suture de la déchirure. L'attitude conservatrice dans ces cas comporte des risques graves de médiastinite ou de suites lointaines de sténose bronchique avec bronchiectasie.

Les lésions qui se produisent au-delà de la gaine pleurale du pédicule pulmonaire constituent un autre groupe. Dans le cas de rupture d'une grosse bronche avec son revêtement de plèvre viscérale il y a pneumothorax immédiat et la pression peut devenir positive si la déchirure agit à la manière d'une valve. Si la déchirure affecte une bronche plus petite, l'air peut s'accumuler sous la plèvre viscérale et ne s'échapper dans l'espace pleural que plusieurs heures plus tard donnant également lieu à un pneumothorax. Les petites déchirures guérissent spontanément. On peut aussi voir des cas d'emphysème inter-lobaire. Dans les cas où la plèvre viscérale n'adhère plus, l'air se fraie graduellement un passage le long des bronches et fait finalement irruption dans le médiastin après quelques heures. Le tableau clinique est aussi celui de l'emphysème sous-cutané mais le traitement ne consiste qu'en une trachéotomie afin de diminuer l'espace mort et de maintenir l'échange gazeux à un niveau satisfaisant. Dans la majorité de ces cas l'acte thérapeutique dépend de la bronchoscopie. Il est donc important de la pratiquer aussi souvent qu'il est nécessaire et le plus tôt possible après l'accident.

SURGICAL TREATMENT OF ACHALASIA OF THE ŒSOPHAGUS*

C. EDWARD B. ABBOTT, B.A., M.D.,
WALTER C. MacKENZIE, M.D., C.M., M.S., F.R.C.S.(C), F.A.C.S., and
COLIN A. ROSS, M.D., F.R.C.S.(C), F.A.C.S., *Edmonton, Alta.*

THE PURPOSE of this review is to evaluate the modified Heller operation in the treatment of achalasia of the œsophagus. Achalasia is a disease of unknown etiology and refers to a condition in which there is a generalized disorder of the motility of the entire œsophagus with failure of the lower œsophageal sphincter to relax on swallowing.

ETIOLOGY

The first description of this condition was by Thomas Willis[1,2,4] in 1672, who felt that there was an obstruction at the upper end of the stomach caused by a tumour or a palsy. In 1881, von Mikulicz proposed spasm of the cardiac sphincter as the etiological factor. Einhorn,[2,4] in 1888, suggested that there was an absence of relaxation at the cardiac orifice rather than actual spasm. Hurst,[3] in 1913, was impressed by the fact that even in the presence of so-called cardiospasm a bougie passed easily through the cardiac orifice. He felt that there apparently was a lack of relaxation after normal peristalsis and, following the suggestion of Sir Cooper Perry, he proposed the term "achalasia". During the years 1926 to 1930, Rake[5] together with Hurst[6] reported eleven patients with evidence of degeneration of Auerbach's plexus in the lower portion of the œsophagus associated with achalasia. They concluded that the failure of the cardiac sphincter to relax was secondary to this degeneration.

Some recent experimental work by Alnor[7] in Germany has shown that it is possible to produce achalasia in rabbits and dogs simulating that seen in human beings. This he accomplished by applying CO_2 snow to a small area of the cardia and thus injuring Auerbach's plexus. However, although most authors agree that the disease is one of neuromuscular dysfunction, the theory that degeneration of Auerbach's plexus is the primary condition is not universally accepted.

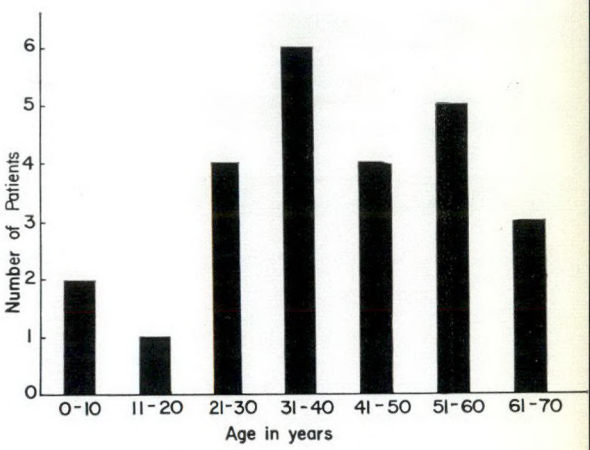


Fig. 1.—Age distribution.

STUDY

The present study concerns twenty-five patients (13 female and 12 male) operated upon for achalasia between the years 1946 and April 1958 in the teaching hospitals of the University of Alberta. The youngest patient was two and a half years old, the

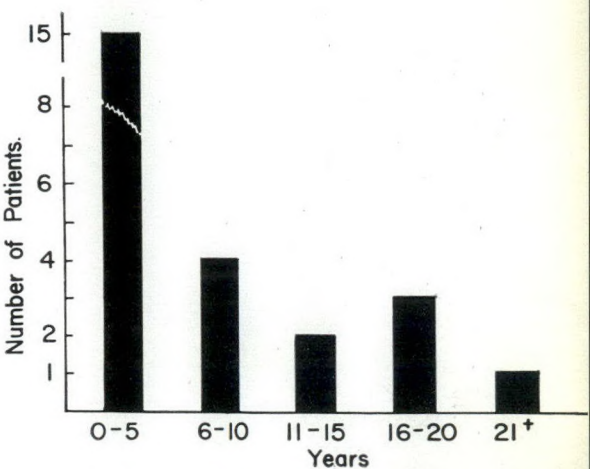


Fig. 2.—Duration of symptoms grouped into five year periods.

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

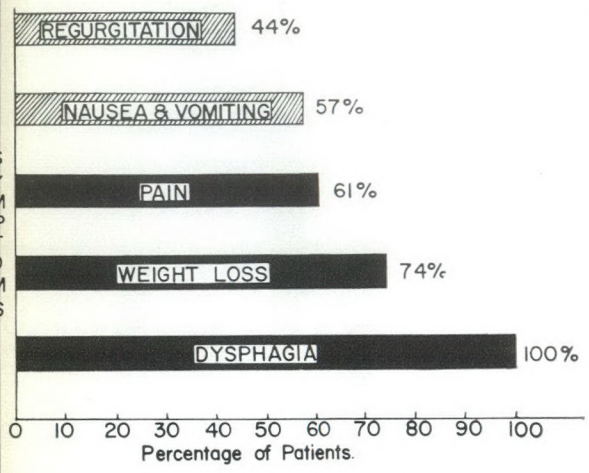


Fig. 3.—Major symptoms.

oldest was seventy years of age (Fig. 1). The duration of symptoms has been grouped into five year periods as in Figure 2, the average for the group being seven years. Sixty per cent of this group of patients had symptoms for five years or less. Dilatation of the œsophagus was carried out in 13 of the 25 cases before operation.

Symptoms

The most significant and the presenting complaint in all patients was dysphagia. In 17 patients, difficulty in swallowing was noted with both solids and liquids; in six patients it occurred only with solids, and in two patients only with liquids. Weight loss was a feature in over 70% of the patients. Retrosternal pain after meals was present in 60% of the group (Fig. 3). Physical findings apart from weight loss were not remarkable in the patients studied.

Diagnosis

The diagnosis was suspected from the history and confirmed by roentgenological and œsophagoscopic examinations. Œsophagrams were reviewed in all cases and these demonstrated various degrees of dilatation. The composite roentgenogram seen in Figure 4 demonstrates various degrees of achalasia. All patients in this series who had not undergone dilatation before operation had preoperative œsophagoscopic examination.

Differential Diagnosis

Achalasia must be differentiated from benign and malignant strictures of the œsophagus. Ellis and associates[8,16] refer to a separate entity, "diffuse spasm of the œsophagus", and point out the importance of differentiating it from achalasia. Diffuse spasm is characterized by simultaneous repetitive, forceful contractions of the

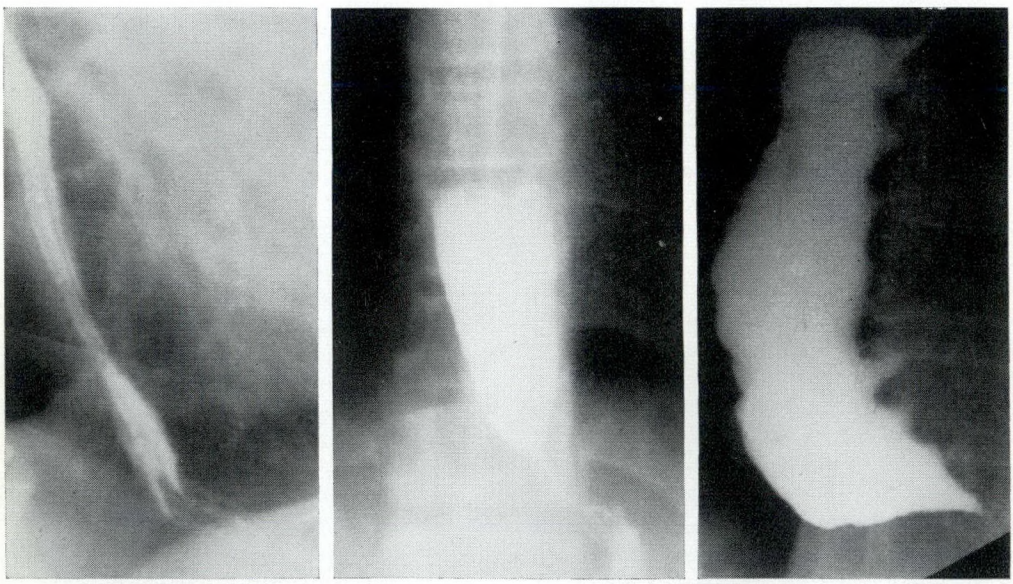


Fig. 4.—Left, a normal œsophagram. Centre, typical fusiform dilatation in the lower end of the œsophagus. Right, grossly dilated œsophagus with sigmoid-like characteristics.

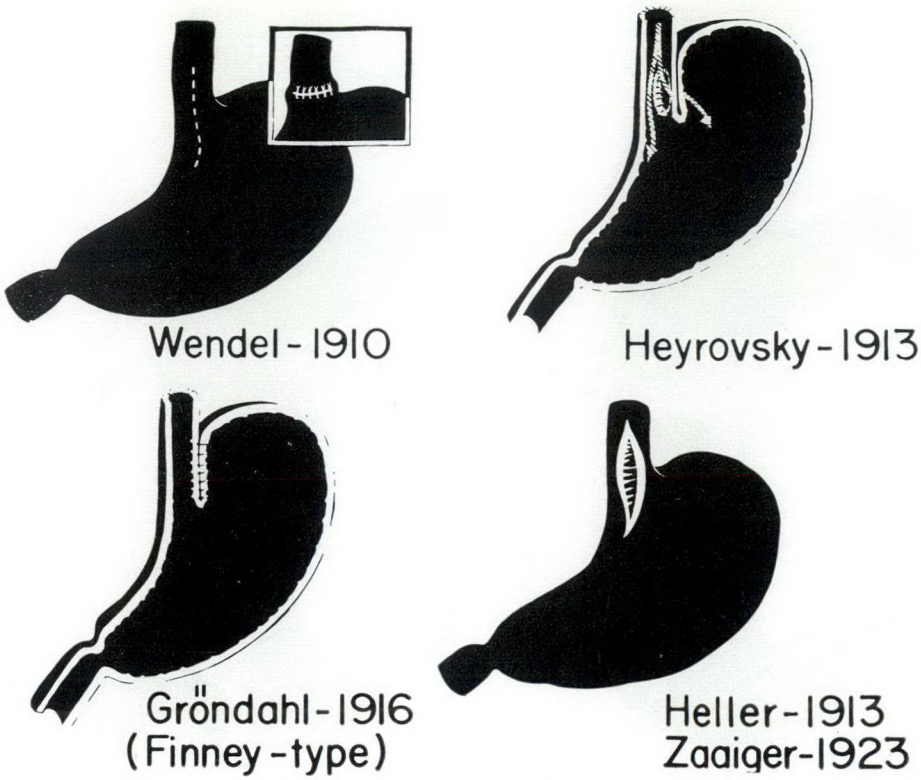


Fig. 5.—Various surgical procedures. See text.

œsophagus after swallowing, resulting in dysphagia associated with pain. Dilatation is rare in the lower part of the œsophagus, and muscular hypertrophy may be present. This entity may occur alone or with other associated gastrointestinal diseases. Scleroderma occasionally can be confused with achalasia as the œsophageal disorder precedes the dermatological manifestations. However, the wall of the œsophagus is usually stiffened and dilatation is not marked.

Development of Operative Management

Numerous surgical procedures (Fig. 5) have been advocated over the years, most of which have been discarded today. The original approach was to bypass or destroy the œsophago-gastric junction. Mikulicz, in 1904, manually dilated the narrowed segment through a gastrotomy incision. In 1910, Wendel[9] advocated longitudinal incision through all layers of the œsophago-gastric junction with transverse closure, thus increasing the diameter of the lumen at this site. This procedure has been revived as recently as 1956 by Sweet.[10] Heyrovsky,[9,18] in 1913, anastomosed the gastric fundus to the dilated œsophagus to bypass the narrowed segment at the cardia. In 1915, Girard[11] performed a modification of the Wendel type of procedure in which only the muscularis was incised and then sutured transversely. Tuttle[11] has carried this out on 36 patients since 1949 with good results. However, it is not generally agreed that this closure of the muscularis is necessary to prevent reflux œsophagitis. Gröndahl,[9] in 1916, utilized a longitudinal incision through all layers of the lower œsophagus and upper stomach with reconstruction similar to a Finney-type pyloroplasty. However, over the years the above procedures have been complicated by regurgitation and reflux œsophagitis and have fallen into disrepute.

In 1913, Heller[9,20] proposed an œsophago-gastric extramucosal myotomy anteriorly and posteriorly (Fig. 5). Heller had been impressed by the similarity of the deformity to that of hypertrophic pyloric

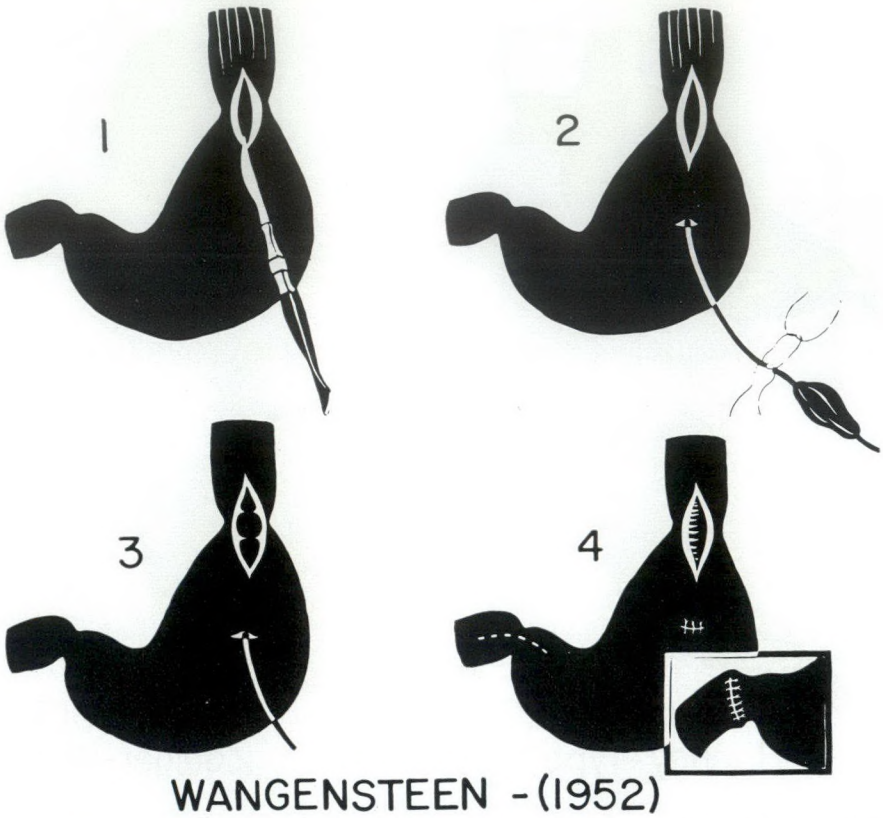


Fig. 6.—Wangensteen modification. No. 2. The Levine tube has been pulled through a gastrotomy incision and a Foley catheter tied to its end. No. 3. The Levine tube has been pulled back and the catheter bag positioned in the œsophago-gastric junction and inflated to facilitate division of the stretched fibres of the lower œsophagus and upper stomach. No. 4. Myotomy completed and the gastrotomy incision closed. The insert shows a Heinecke-Mikulicz pyloroplasty.

stenosis in infancy, and attempted to duplicate the Fredet-Ramstedt operation at the cardia. In 1923, Zaaijer[12] modified the operation by using only one incision placed anteriorly, and it is this modified Heller operation that is most commonly used today.

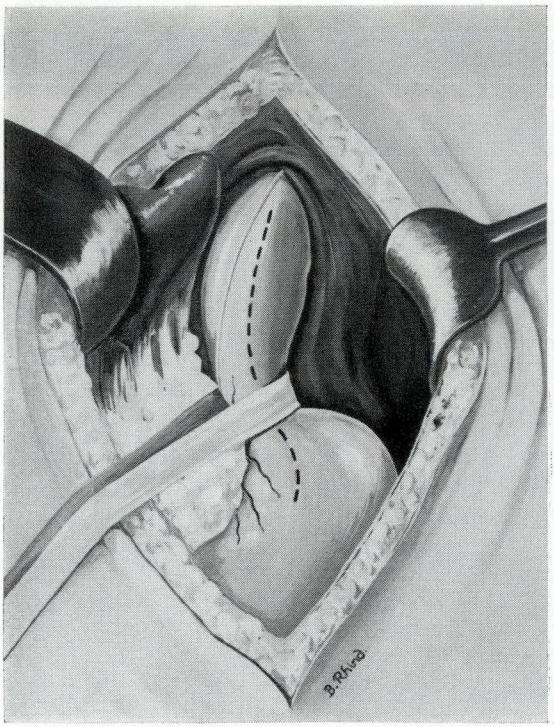
Wangensteen,[13] in 1952, performed the myotomy over an inflated Foley catheter bag introduced through a gastrotomy incision (Fig. 6). This technique facilitates complete division of all remaining œsophageal muscle fibres and upper gastric circular muscle fibres. Wangensteen also added a Heineke-Mikulicz pyloroplasty to facilitate gastric drainage. This is illustrated in Fig. 6, and consists of a longitudinal incision through all layers of the pylorus with closure transversely in one layer. Maingot[14] has also added a pyloroplasty of this type with good results, and feels that he has improved the effectiveness of the surgical procedure.

Treatment

In this series, a modified Heller operation was performed on 22 out of 25 patients, 10 through the thorax and 12 through the abdomen. Only one patient had a Heineke-Mikulicz pyloroplasty. The remaining three patients of the 25 were treated by three different cardioplastic procedures and are not included in the final results.

Operative Procedure

The abdominal approach is favoured by many surgeons because of the following advantages: (1) associated gall-bladder disease, duodenal ulcer or the presence of a hiatus hernia and other intra-abdominal



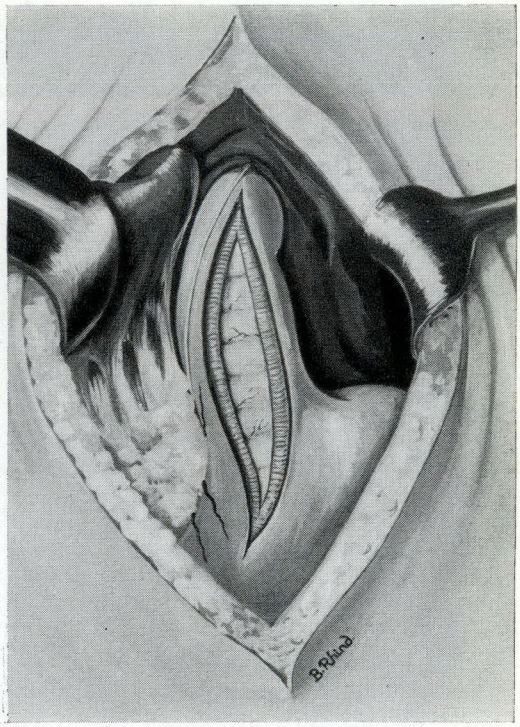


Fig. 7.—Modified from Maingot, R.: *Abdominal Operations,* 3rd. edition, 1955. Courtesy of Appleton Century-Crofts, Inc. **Fig. 8.**—Modified from Maingot, R.: *The Management of Abdominal Operations,* Volume I, 1957, 2nd edition. Courtesy of H. K. Lewis & Co., Ltd., London.

lesions can be assessed and treated at the same operation; (2) a pyloroplasty to facilitate gastric emptying can be easily added; (3) postoperative discomfort is less than with a thoracotomy. In the abdominal approach a midline incision is used and the left lobe of the liver is mobilized well to the right by dividing the left triangular hepatic ligament. The peritoneum over the œsophago-gastric junction is divided transversely and the œsophagus mobilized from its bed by blunt dissection with a finger. An encircling Penrose drain can be used to facilitate exposure of the lower end of the œsophagus (Fig. 7). The left vagus nerve is identified and preserved by retraction to the right. The broken line in Figure 7 marks the site of the incision through both muscular layers of the lower œsophagus and upper part of the stomach.

The incision should extend far enough distally to be sure that all œsophageal fibres are divided and it should be kept to the right of the midline. The total length of this incision will average 8-12 centimetres and the mucous membrane should bulge freely into view as in Figure 8, which shows the completed operation. When the procedure is complete there should be no evidence that any circular bands of muscle fibres are constricting the mucosa. Perforation of the mucosa at operation occurs most often at the junction of the gastric and œsophageal mucosa and must be recognized and carefully closed. No attempt is made to approximate the peritoneum over the defect in the muscle wall.

The addition of a pyloroplasty in all probability improves gastric drainage and decreases the incidence of reflux into the œsophagus. While this procedure was carried out in only one patient in this study, it is our feeling that the physiological basis for its addition is a sound one.

Results

All 22 patients in the Heller group have replied to correspondence, while 13 have been interviewed personally by the authors.

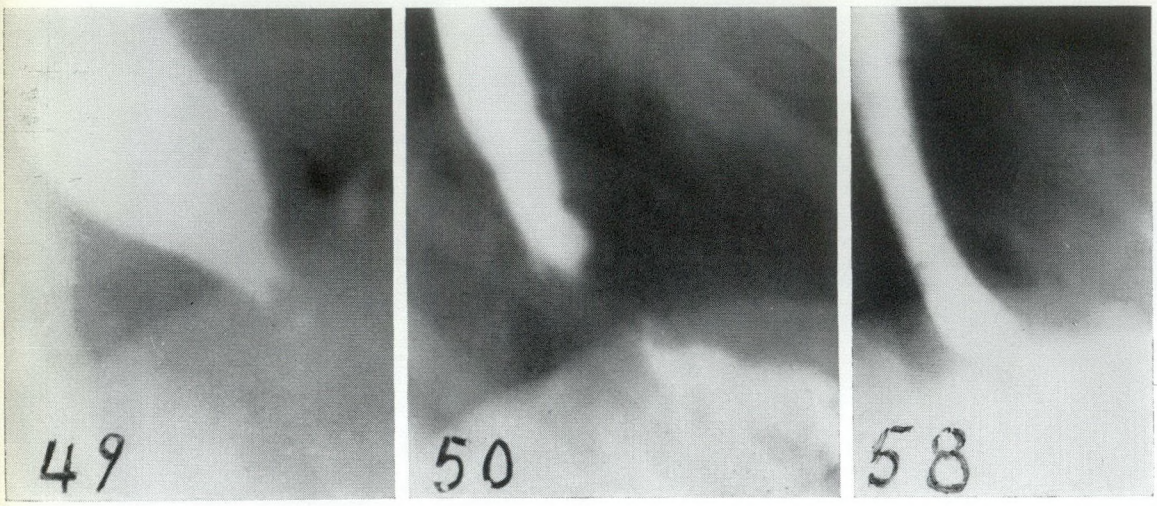


Fig. 9.

Two patients are dead from other causes. The follow-up period in this Heller group averages three years and two months. Seventy-seven per cent of the patients have had an excellent result with no recurrence of dysphagia and no clinical evidence of regurgitation or œsophagitis. Nine per cent are classified as having had a good result, experiencing an occasional bout of dysphagia, especially when eating too rapidly. The patients in both groups have gained weight and returned to normal activities. In three patients a poor result was obtained. Careful review of the records revealed that, in all three, insufficient indications were present to warrant the operative procedure. One was a child of two and one-half years of age who had muscular dystrophy; a second was a man, age 66, whose primary lesion was a stricture of the lower œsophagus; while the third poor result was in a man 58 years of age whose main symptom was dysphagia. However, the roentgenological findings did not support a diagnosis of achalasia.

The composite roentgenogram in Fig. 9 demonstrates the result in a 35 year old female with achalasia of six years' duration. She had numerous dilatations between 1943 and 1949. The preoperative œsophagram in 1949 demonstrates the moderate fusiform dilatation of the lower œsophagus, typical of many of the patients in this review. The œsophagram in 1950 was taken two months postoperatively, while the œsophagram in 1958 confirms the excellent clinical result in this patient eight years after a modified Heller operation.

Discussion

It has been previously reported[15] that between 60% and 80% of patients with achalasia will respond to periodic œsophageal dilatation and medical management. The remainder have required surgical intervention. However, it is perhaps time we re-assessed these results, remembering that patients with severe symptoms may obtain relief but not an asymptomatic state. Persistent medical management results in progressive œsophageal dilatation, inflammation and scarring which makes later cardiomyotomy difficult. We should also keep in mind the fact that bouginage or hydrostatic dilatation is simply a blind uncontrolled attempt to mimic an extramucosal œsophago-cardiomyotomy. Furthermore, it is not without its dangers, such as perforation and mediastinitis, both of which can be prevented by direct surgical approach. This has been dramatically called to our attention in a 38 year old female patient with achalasia who had a hydrostatic dilatation by an experienced, competent thoracic surgeon in November 1957. Two days later, there was clinical evidence of œsophageal perforation. A left thoracotomy confirmed the presence of a small laceration in the mucosa of the lower end of the œsophagus associated with extensive

muscle laceration similar to that seen at the conclusion of a modified Heller procedure. This patient, fortunately, did not develop mediastinitis, but has had a lengthy convalescence due to a small abscess in the left lower lobe of the lung behind the left border of the heart.

Conclusion

This study confirms the belief that extramucosal œsophago-cardiomyotomy is an excellent method of treatment for achalasia of the œsophagus when the diagnostic criteria are met. It must be recognized, however, that this operation does not abolish the deranged motility of the œsophagus but simply destroys the area of contraction at the lower end of the œsophagus.

It has been recently suggested[16,17] that the modified Heller operation should be the primary form of treatment in achalasia of the œsophagus. This can be supported by the fact that direct surgical division of the muscle fibres does away with their blind avulsion, which in turn may result in the complications mentioned earlier. The patients in this series obtained dramatic relief of their symptoms and are extremely grateful. In our opinion, an extramucosal œsophago-cardiomyotomy (better known as a modified Heller operation) is the primary treatment of choice in achalasia of the œsophagus.

Summary

Twenty-five patients with achalasia of the œsophagus have been operated upon and form the basis of this review. Twenty-two were treated by a modified Heller operation, while three had various reconstructive procedures on the gastro-œsophageal junction.

The historical aspects of the lesion and the present day theories of etiology are discussed. Typical signs and symptoms in the patients studied, the differential diagnosis, and representative preoperative and postoperative roentgenograms are illustrated and discussed.

All 25 patients have been assessed and no case has been lost to follow-up. In the group treated by a modified Heller procedure the follow-up period averaged three years and two months. In the small group treated by gastro-œsophageal reconstruction, the time since operation has averaged eleven years. The results of treatment of both groups have been carefully studied.

It has been concluded from this study that a modified Heller œsophago-cardiomyotomy is the primary treatment of choice in achalasia of the œsophagus.

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Résumé

L'achalasie de l'œsophage est un état pathologique d'étiologie inconnue dans lequel il existe un désordre du péristaltisme de l'organe accompagné d'une absence de relâchement du sphincter inférieur lors de la déglutition.

Après une revue de la littérature concernant cette question, les auteurs étudient 25 cas de cette entité, dont 13 femmes et 12 hommes. Le symptôme dominant est la dysphagie: la difficulté à avaler porte sur les solides et les liquides (17 cas), sur les solides seulement (6 cas) ou sur les liquides seulement (2 cas). La perte de poids et les douleurs rétrosternales postprandiales sont très fréquentes.

Le diagnostic fut établi d'après l'histoire de la maladie et d'après les examens radiologiques et œsophagoscopiques. Le diagnostic différentiel avec les strictures bénignes ou malignes de l'œsophage se pose.

Les auteurs exposent succintement plusieurs traitements opératoires proposés dans le passé et qu'on a maintenant abandonnés. L'intervention actuelle est une modification de l'opération de Heller, qui est essentiellement une myotomie extra-muqueuse œsophago-gastrique. En ce qui concerne le processus opératoire, la voie abdominale doit être préférée: elle permet le traitement d'un état pathologique abdominal concomitant, et l'association d'une pyloroplastie au besoin.

Des 22 malades traités de cette façon et suivis pendant environ trois ans, 77% ont eu un excellent résultat, ne souffrant plus d'aucune dysphagie, et 9%, un bon résultat, présentant seulement un peu de difficulté à avaler lorsqu'ils mangent trop vite. Trois patients non améliorés ne montraient pas d'indication opératoire bien établie.

SPLENIC INDENTATION OF THE GASTRIC FUNDUS SIMULATING NEOPLASM

Two cases are described by Brown and Dobbie (*Am. J. Roentgenol.*, 81: 599, 1959) in which a normal spleen was apparently responsible for the production of a persistent filling defect of the fundus of the stomach resembling gastric neoplasm. In each case, the filling defect was repeatedly identified on upper gastrointestinal roentgenograms.

In each case the patient had been admitted to hospital for investigation of vague but persistent upper abdominal pain, accompanied by nausea and other gastro-intestinal symptoms. In each case, the radiologist found a persistent fundal filling defect which did not however seem related to the symptoms and did not strongly suggest a malignant lesion. In one case a benign gastric tumour (leiomyoma) was diagnosed, and in the other an intramural polypoid neoplasm.

When exploration was carried out, the superior end of a normal spleen was found to be in intimate contact with the greater curvature at a site corresponding with the filling defect.

The authors suggest that, although it is not surprising that the superior tip of the spleen can cause a recognizable indentation of the gastric wall, this diagnostic trap is not well appreciated.

CYSTS AND CYSTIC TUMOURS OF THE ANTERIOR MEDIASTINUM*

MAURICE BEAULIEU, M.D., WILFRID CARON, M.D., F.R.C.S.[C] and
BERNARD PARADIS, M.D., *Quebec City, Que.*

THE MEDIASTINUM is the space between the pleural cavities extending from the upper opening of the thorax to the diaphragm. It is bounded anteriorly by the sternum, posteriorly by the thoracic vertebræ and laterally by the mediastinal layers of the pleura. A line drawn through the fourth thoracic vertebra to the junction of the second rib with the sternum divides the mediastinum into a superior and inferior portion (Fig. 1).

The most frequent tumours of the superior mediastinum are neurogenic tumours and intrathoracic goitres; lymphangiomas are rarer lesions.

Below the superior mediastinum, the mediastinal cavity may be divided into three portions: the posterior mediastinum, the middle mediastinum and the anterior mediastinum.

The posterior mediastinum is that area lying posterior to the heart and anterior to the lower eight thoracic vertebræ. In this region, enterogenous cysts and neurogenic tumours may be found. Neurogenic tumours are by far the most frequent; neurofibromas arise from the intercostal nerves and ganglioneuromas from the sympathetic ganglia of the thoracic region. About 35% of all mediastinal tumours are neurogenic; one out of five of these tumours is malignant.

The middle mediastinum includes the bifurcation of the trachea, the pericardial sac and its contents, the heart and great vessels. Masses in this region are most frequently composed of diseased lymph nodes: tuberculous, lymphomatous, metastatic or granulomatous (especially sarcoidosis).

The anterior mediastinum is that area which lies anterior to the pericardium, inferior to the superior mediastinum and posterior to the sternum. The most frequent tumours of the anterior mediastinum are bronchogenic cysts, pericardial cysts, teratoid tumours and thymomas.

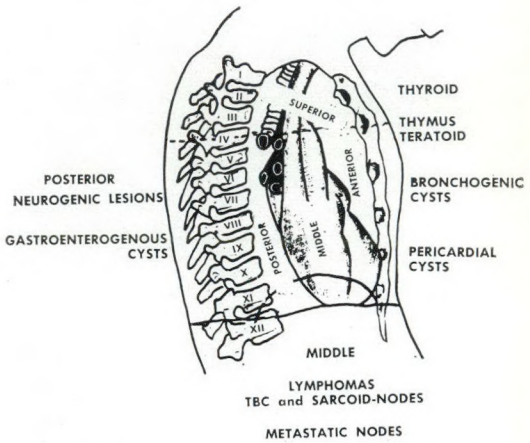

Fig. 1.—Compartments of the mediastinum and location of mediastinal tumours.

All the lesions mentioned above occur most commonly in the mediastinal area indicated (Fig. 1) but not exclusively so. For example, bronchogenic cysts are occasionally found in the posterior mediastinum. A mass may sometimes grow or slide towards more compressible or more dependent areas.

Nearly 50% of all mediastinal tumours are found in the anterior mediastinum. These tumours are frequently cystic. Bronchogenic cysts and pericardial cysts are true cystic formations; their respective incidences are about 15% and 5% of all mediastinal tumours. Teratomas may be solid but usually they are at least partly cystic; their incidence is about 10% of all mediastinal tumours. Thymomas are common and form about 15% of all mediastinal tumours. They are usually solid, thymic cysts being very rare lesions.

We will present typical cases of these cysts or cystic lesions. A brief description of the most common pathological findings in such cases will follow. Finally, we will discuss the diagnosis and the action to be taken when an abnormal shadow is discovered in the anterior mediastinum.

CASE 1.—A 56 year old man was admitted to hospital on May 1, 1958, complaining of

*From the Department of Surgery of Ste-Foy Hospital (D.V.A.), Ste-Foy, Quebec.

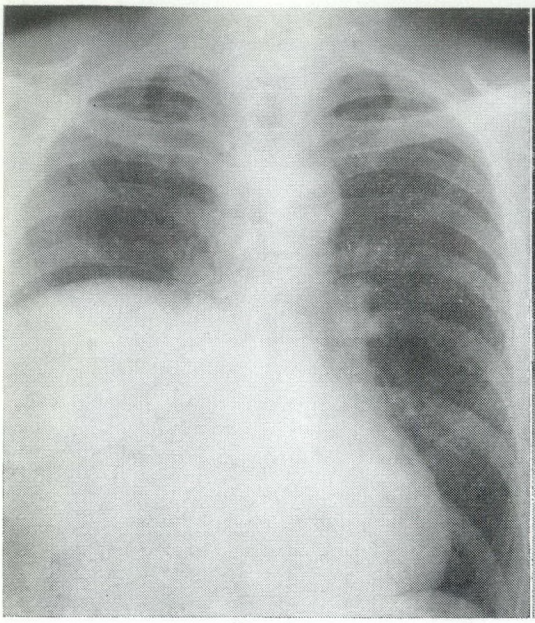

Fig. 2.

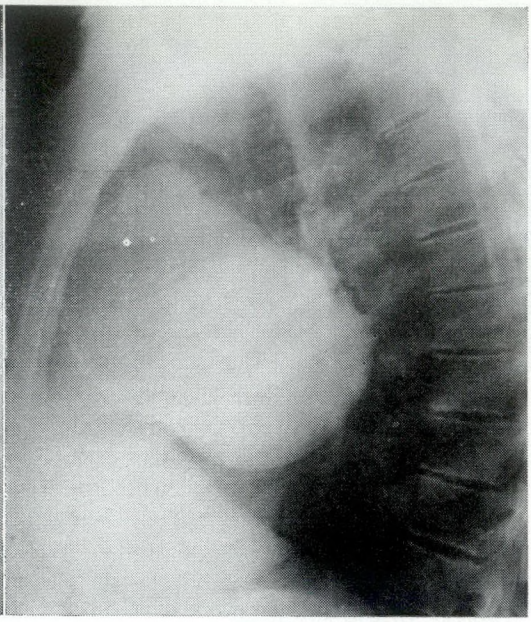

Fig. 3.

Fig. 2.—Case 1—pericardial cyst. Preoperative chest radiograph (antero-posterior view).
Fig. 3.—Case 1—pericardial cyst. Preoperative chest radiograph (lateral view).

slight dyspnœa and fatigue of six months' duration. He stated that he had had a right pleurisy at the age of 12. Radiological studies showed a large spherical mass of homogenous density located anteriorly in the right cardio-phrenic angle (Figs. 2 and 3). At fluoroscopy, there were

Fig. 4.—Case 1—pericardial cyst. Pericardial cyst as it appeared immediately after opening the right chest.

no pulsations. A barium swallow did not demonstrate herniation of abdominal viscera into the right chest. Results of physical examination, electrocardiogram and results of all laboratory examinations were within normal limits. On May 9, 1958, right thoracotomy was performed through the bed of the seventh rib. A thin-walled cyst measuring 13 by 19 cm. was easily removed, as no adhesions were present except over a limited area of the pericardium (Fig. 4). Postoperative recovery was uneventful (Fig. 5). The cyst contained clear water. Histopathological examination revealed a pericardial cyst.

Pericardial cysts are thought to be cœlomic cysts and to originate from failure of primitive mesenchymal lacunæ which form the pericardium to fuse with others; instead, they form independent cavities. They are nearly always located anteriorly and closely related to the cardio-phrenic angle, more frequently the right. Some have a pedicle attaching them to the pericardium. There may be a narrow communication through which fluid in the cyst can be expressed into the pericardium. In the case reported, the cyst was adherent to the pericardium but there was no such pedicle or communication. Pericardial cysts are frequently called "spring water cysts" because they contain crystal clear fluid. They have a thin, purely fibrous wall which is lined by a single layer of cuboidal or flat cells.

CASE 2.—A 28 year old housewife was admitted to hospital on March 30, 1954, complaining of palpitations and dyspnœa of two years' duration, and of recent episodes of dizziness on forward bending. At the time of her first symptoms, the patient had a chest radiograph taken; she was told that she had enlargement of her heart. On the admission film, it was possible to arrive at a similar conclusion (Fig. 6). However, on study of tomographic sections, a spherical mass of homogenous density was easily made out in front of the heart (Fig. 7). At fluoroscopy and kymography, the normal pulsation of the left border of the heart could not be demonstrated. Physical examination was negative except for diminished heart sounds; there was no murmur. Electrocardiographic tracings showed flattened T waves. All laboratory examinations gave results within normal limits. On April 4, 1954, a left thoracotomy was performed. A huge cyst dislocating the heart to the right and posteriorly was easily removed (Fig. 8). There was a pedicle attaching the cyst

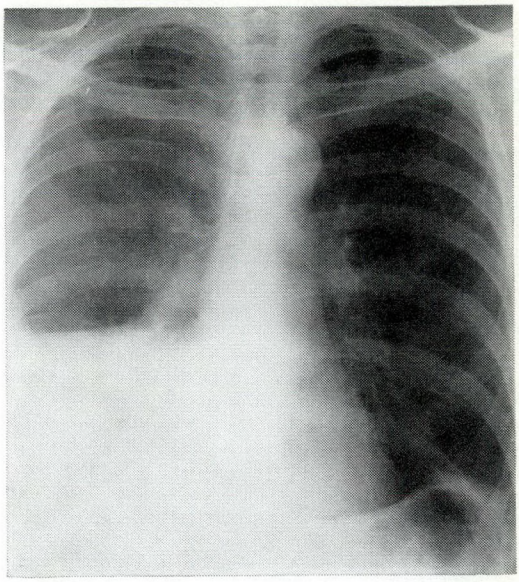

Fig. 5.—Case 1—pericardial cyst. Postoperative chest radiograph.

to the left main bronchus but no free communication with the bronchial tree could be demonstrated (Fig. 9). The anatomical diagnosis was that of a typical bronchogenic cyst. The cyst was unilocular and contained about 1500 c.c. of brownish thin fluid. Four years after operation, the patient remains well and free of her previous symptoms.

Bronchogenic cysts originate essentially in a developmental fault, probably by a "pinching off" process from one of the out-

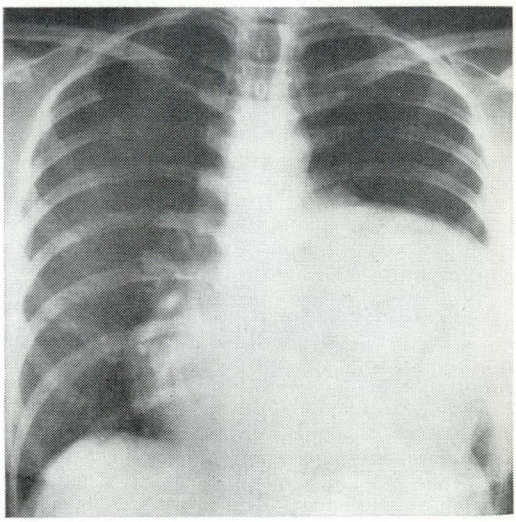

Fig. 6.—Case 2—bronchogenic cyst. Preoperative chest radiograph.

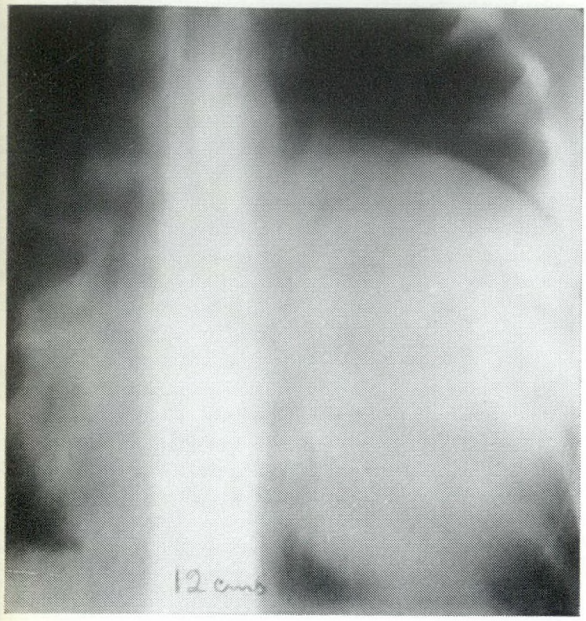

Fig. 7.—Case 2—bronchogenic cyst. Anterior tomographic section (12 cm. from the posterior chest wall).

growing lung buds. They are closely related to the carina or a major bronchus, to which they may be attached by a pedicle. Free communication with the bronchial tree is rarely present; in the few cases reported, there was occasional evacuation of fluid contents into the air passages, and on x-ray films the cyst showed a fluid level. Bronchogenic cysts are usually unilocular and the contents may be either clear and mucinous or brownish. Microscopically, the wall contains bronchial elements such as cartilage, smooth muscle, mucous glands and elastic tissue in disorderly arrangement; it is lined with bronchial epithelium which may or may not be ciliated. In the case reported, these tissues were present; the bronchial epithelium was not ciliated. Bronchogenic cysts are benign lesions, but infection is a serious complication; it may well lead to rupture of the cyst, mediastinal abscess and empyema.

Case 3.—A 22 year old soldier was admitted to Ste-Foy Hospital on March 6, 1957, complaining of slight left upper chest pain. The left pleural cavity had been drained for empyema at the age of three.

Radiological studies showed an abnormal mediastinal shadow at the level of the aortic knob (Fig. 10). This shadow was best visualized

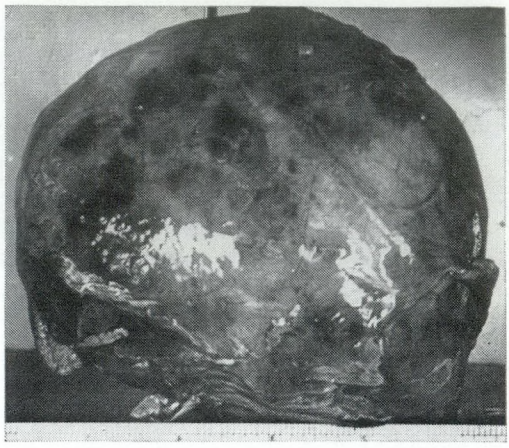

Fig. 8.—Case 2—bronchogenic cyst.

on anterior tomographic sections but its contour remained partly obscured (Fig. 11). At fluoroscopy, there was no evidence of pulsation and the mass did not rise on swallowing. Physical examination was negative. Blood pressure was taken in the four limbs with normal findings. Serological and all laboratory tests were negative. On April 4, 1957, a left thoracotomy was performed. The lung was intimately adherent to the chest wall, probably because of the previous pleural sepsis. An irregular and embossed cyst measuring 16 by 7 cm. was found in front of the great vessels and their divisional branches. It was markedly adherent to these structures. During separation, the cyst was accidentally ruptured, and brownish sebaceous fluid and hair spilled out. Careful sharp dissection was required for complete removal. The postoperative course

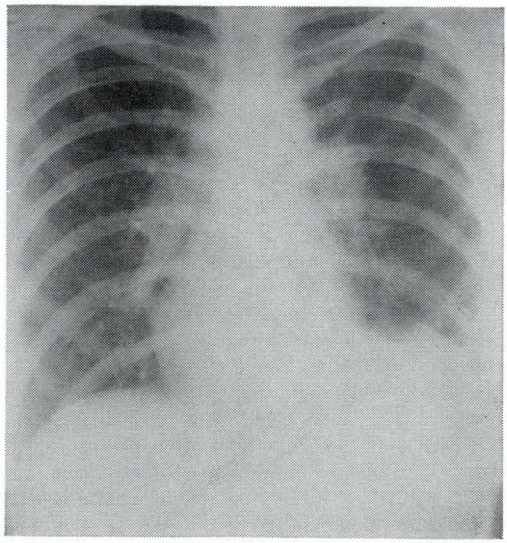

Fig. 9.—Case 2—bronchogenic cyst. Postoperative chest radiograph.

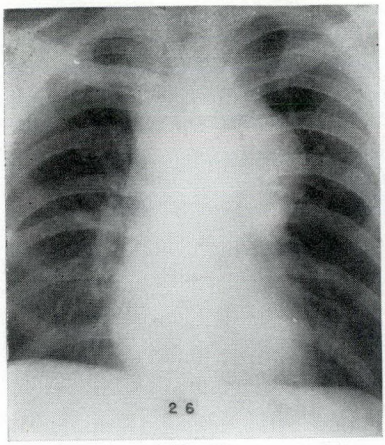

Fig. 10.

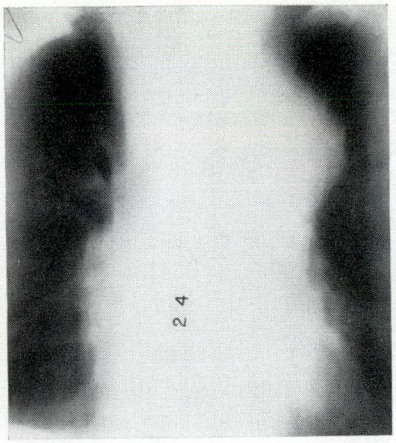

Fig. 11.

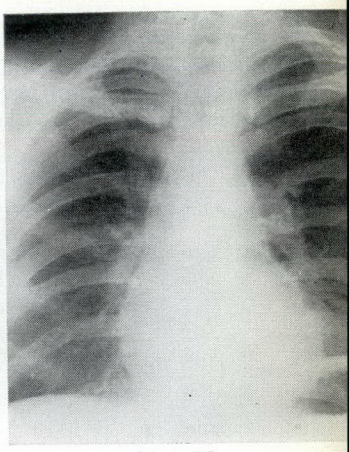

Fig. 12.

Fig. 10.—Case 3—"dermoid" cyst. Preoperative chest radiograph. **Fig. 11.**—Case 3—"dermoid" cyst. Anterior tomographic section. **Fig. 12.**—Case 3—"dermoid" cyst. Postoperative chest radiograph.

was uneventful (Fig. 12). At examination, the cyst was unilocular and its wall was 0.5 cm. thick and firm. The inner lining was irregular and showed a protruding and pediculated sphere (Fig. 13). Histological studies revealed well differentiated tissues of all three germinal layers, but ectodermal derivatives were markedly predominant. The pathologist's diagnosis was of benign teratoid tumour of the "dermoid" type. Two months after operation, the patient was back to full-time work.

Case 4.—A 45 year old veteran was admitted to Ste-Foy Hospital on September 25, 1956, complaining of long-standing palpitations and dyspnœa. He had a history of chronic alcoholism and low-grade hypertension. Chest radiography on admission demonstrated a large mass at the level of the right border of the heart (Fig. 14). Lateral views showed that the tumour was located in the antero-inferior mediastinum. Tomographic sections distinctly showed its smooth margins and its homogenous density (Fig. 15). Physical examination was negative except for diminished breath sounds in the lower right chest. Blood pressure was 160/80 mm. Hg. Electrocardiographic tracings were within normal limits. Serological and all laboratory tests were negative. A right thoracotomy was performed on October 2, 1956. Removal of the mass was made difficult because of the presence of dense adhesions. Postoperatively, the patient made an uneventful recovery (Fig. 16). At examination, the tumour was found to be polycystic and to contain thick brownish fluid. It weighed 1040 g. and measured 17 by 18 by 9 cm. (Fig. 17). Histological examination revealed well differentiated tissues of all three germinal layers, present in fairly even distribution. The pathologist's diagnosis was of benign cystic teratoma. At the follow-up clinic, the patient still complained of dyspnœa and palpitations. Slightly elevated blood pressure (175/90 mm. Hg) seemed responsible for this. Chest x-ray appearances have remained unchanged for the last two years.

Case 5.—A 56 year old woman was admitted to hospital on May 7, 1957, complaining of dyspnœa and pain in the right lower chest of six months' duration. The patient was obese and there was no history of weight loss. Temperature was slightly elevated. Breath sounds were diminished at the base of the right lung anteriorly. The heart rate was 82 and there was no murmur. Erythrocyte sedimentation rate was 108 mm.

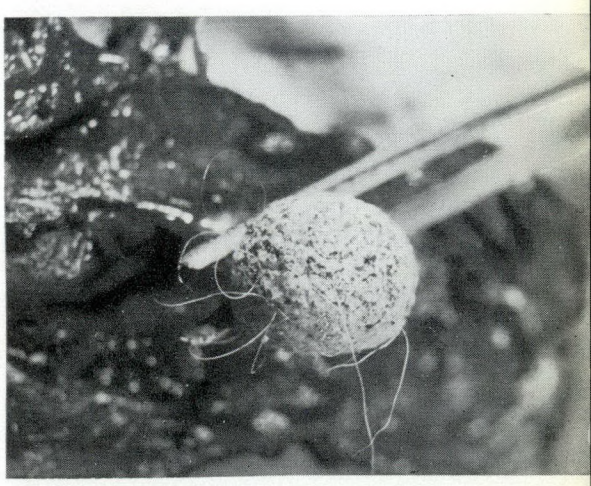

Fig. 13.—Case 3—benign teratoid tumour of the "dermoid" type.

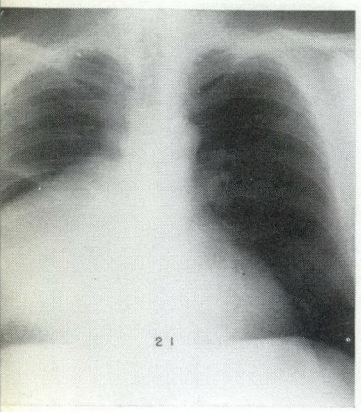

Fig. 14.

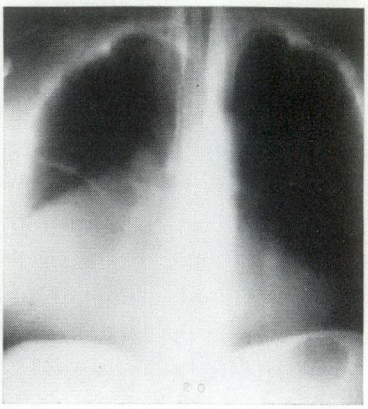

Fig. 15.

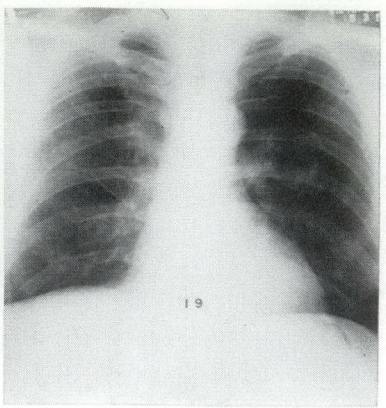

Fig. 16.

Fig. 14.—Case 4—benign teratoid cyst. Preoperative chest radiograph. **Fig. 15.**—Case 4—benign teratoid cyst. Anterior tomographic section. **Fig. 16.**—Case 4—benign teratoid cyst. Postoperative chest radiograph.

in one hour. Radiological studies demonstrated a mass obscuring the lower half of the right lung. The tumour appeared to be connected with the mediastinum by a neck-like prolongation (Fig. 18). At fluoroscopy, the anterior position of the mass was established; pulsation was absent. Bronchoscopy revealed a normal bronchial tree. Urinalysis was negative. Examination of the blood revealed a hæmoglobin value of 12.1 g. % and a white cell count of 22,660 per c.mm., with 75% neutrophils. A right thoracotomy was performed on April 19, 1957. A very adherent tumour was removed with great difficulty. The pericardial sac had to be extensively excised along with the mass, as no separation was possible at this level (Fig. 19). The specimen was a unilocular cyst measuring 15 by 10 by 10 cm. (Fig. 20), and containing brownish fluid. The wall thickness varied from 1 to 5 mm. The cyst wall was composed of round cells with large hyperchromatic nuclei and of multinucleated giant cells with occasional mitoses, and lined by disorderly arranged epithelial cells. The pathologist's diagnosis was of malignant cystic teratoma. Two weeks after operation the patient was started on radiation therapy. Metastatic dissemination occurred with death two months after operation.

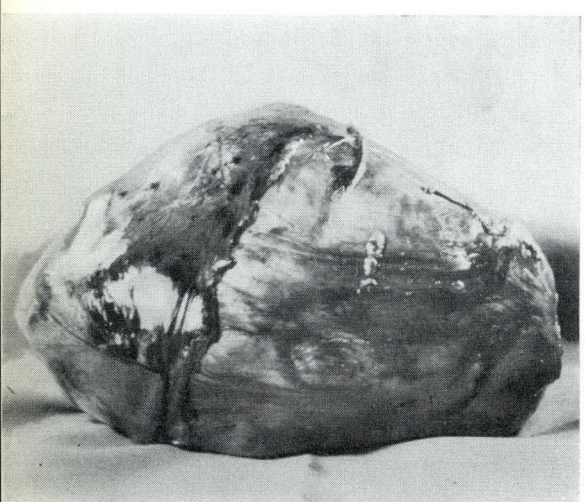

Fig. 17.—Case 4—benign cystic tumour.

Teratoid tumours are the commonest tumours of the anterior mediastinum. They are thought to arise as abnormal outgrowths of the third and fourth branchial arches, descending into the mediastinum during embryonic development, along with the great vessels. These tumours vary greatly in their make-up, but in each type all three germinal layers are ordinarily present. When such ectodermal structures as hair, teeth or sebaceous glands predominate, they are commonly referred to as "dermoid" tumours. These are generally cystic and contain thick gelatinous or sebaceous material. Mesodermal structures (such as bone, cartilage, smooth muscle) and entodermal elements (of glandular and enteric appearance) may predominate. These teratomas may be solid but usually they are at least partly cystic. The surgical removal of teratoid tumours often presents great difficulty owing to dense and tough adhesions to the underlying pericardium, great vessels and their first divisional branches. Infection and malignancy may add considerably to these hazards. Cystic teratoid lesions may become infected, with possible rupture and forma-

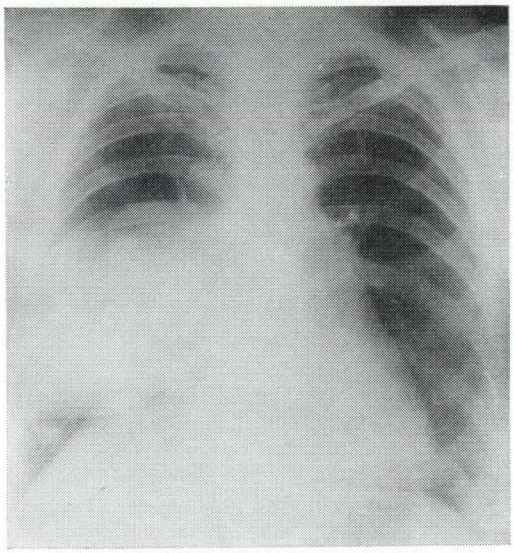

Fig. 18.—Case 5—malignant teratoid cyst. Preoperative chest radiograph.

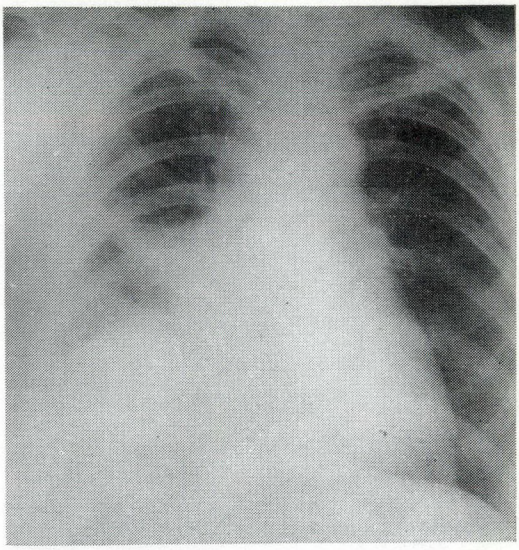

Fig. 19.—Case 5—malignant teratoid cyst. Postoperative chest radiograph.

tion of a mediastinal abscess or empyema. On rare occasions, these cysts have ruptured into a bronchus with expectoration of sebaceous fluid and hair, a surprising experience which establishes the diagnosis. Solid teratomas are more likely to become malignant than cystic ones. From 10% to 20% of teratoid tumours undergo malignant change. Malignant teratomas usually lack ectodermal structures and their epithelial component is predominantly adenocarcinomatous.

Case 6.—A 37 year old veteran was admitted to Ste-Foy Hospital on January 7, 1956, complaining of low back pain. The patient had suffered a traumatic fracture of the lumbar spine (L1 and L2) in early 1955. There was also a history of precordial pain of two years' duration. The pain radiated to both shoulders and was considerably relieved by coronary vasodilators. Standard x-ray films of the chest showed enlargement of the heart shadow (Fig. 21). Tomography was most informative, as the heart could be distinguished from a right-sided adjacent mass. This tumour was best visualized in anterior sections, which also showed its connection with the mediastinum by a large pedicle (Fig. 22). Physical examination was negative except for some muscular rigidity in the lumbar region. Electrocardiographic tracings and results of all laboratory tests were within normal limits. On February 23, 1956, a right thoracotomy was performed. A tumour of apparent cystic nature was found in front of the right auricle, superior vena cava and ascending aorta. It could be easily freed from these structures. A neck-like prolongation of the cyst extended upward to a level about two inches (5 cm.) above the left innominate vein where it seemed to originate. Postoperatively, the patient made an uneventful recovery (Fig. 23). Examination of the specimen revealed a unilocular cyst (Fig. 24). Its inner surface was embossed with small yellowish nodules. It contained yellowish serous fluid rich in cholesterol. Histologically, it was a thymic cyst. The patient is seen periodically, and has gained 15 lb. and his precordial pains have disappeared.

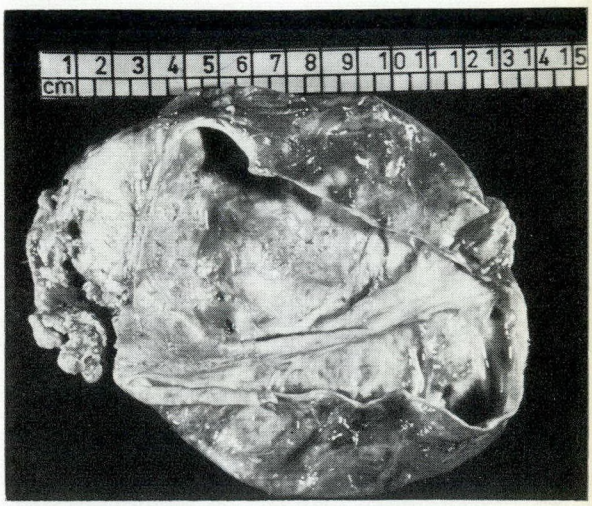

Fig. 20.—Case 5—malignant cystic tumour.

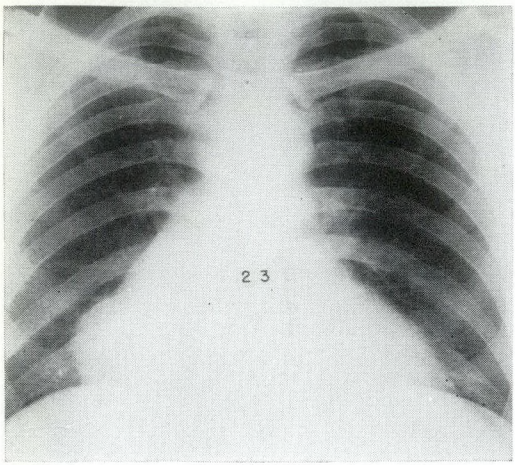

Fig. 21.—Case 6—thymic cyst. Preoperative chest radiograph.

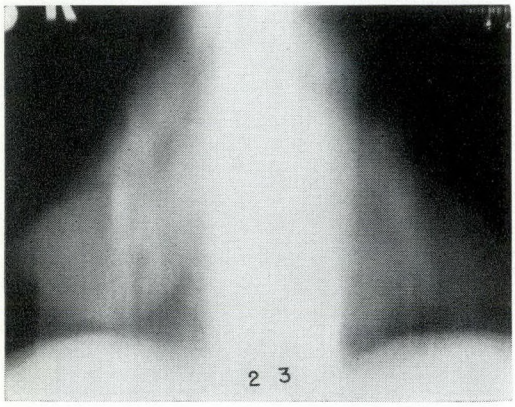

Fig. 22.—Case 6—thymic cyst. Anterior tomographic section.

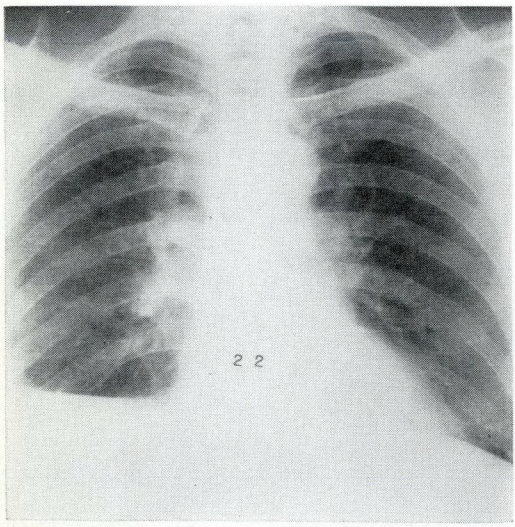

Fig. 23.—Case 6—thymic cyst. Postoperative chest radiograph.

About 15% of all mediastinal tumours are of thymic origin, but thymic cysts are very rarely encountered. In the early literature, thymic cysts were reported in syphilitic patients, especially in children with congenital syphilis. Nowadays, such lesions are rarely reported. On the other hand, a few cysts claimed to have arisen from remnants of the thymic duct have been described in recent years. Krech and associates, in 1954, collected 13 cases from the literature and added two of their own. Thymic cysts, which appear to be related to branchial and cervical cysts, are usually derived from a remnant of the third branchial pouch. They may be located in any position along a line extending from the angle of the jaw medially to the midline of the neck, thence descending into the anterior mediastinum to the pericardium as far distant as the diaphragm. The anterior mediastinum is the most frequent location. Histologically, these cysts are usually lined by low cuboidal or pavement epithelium. Thymic tissue elements, lymphocytes and thymo-epithelial cells are immediately adjacent to this lining. Hassall's corpuscles may or may not be present. Scattered foci of cholesterol clefts are a common finding. In the case presented, these pathological features were all present, including Hassall's corpuscles and cholesterol deposits.

Before resorting to surgery for an abnormal shadow of the anterior mediastinum, a differential diagnosis of primary tumour should be attempted. Intrathoracic goitre, aneurysm of the great vessels, primary bronchogenic carcinoma, enlarged lymph nodes, and diaphragmatic hernia through the foramen of Morgagni are all possibilities that must be borne in mind. Some of these require a special surgical approach and technique; in others, thoracotomy is not indicated at all. When each in turn has been eliminated, the wide field of primary cysts and tumours remains for differentiation.

Tumours of the anterior thoracic inlet which move on swallowing are attached to the larynx or upper trachea and are probably of thyroid origin. The use of radioactive iodine isotopes is of great help in identifying intrathoracic thyroid masses.

Most of the intrathoracic goitres can be removed through a cervical incision.

Aneurysms of the great vessels may be distinguished by radiological evidence of pulsation, by demonstrating their continuity with the vascular structures, if necessary by angiography, by serological reactions and characteristic physical signs.

About 75% of all bronchogenic carcinomas arise in the major bronchi. They may cast a shadow simulating a mediastinal tumour. In these cases, bronchoscopy with biopsy and cytological examination of the bronchial secretions can establish the correct diagnosis.

Lymphatic tissue is richly disposed in the mediastinum. Chains of lymph nodes are found clustered on either side of the trachea, at the carina and about the hila. It is not surprising that these mediastinal nodes are involved both in localized tumours and granulomas, and in generalized disorders of lymphatic tissue.

The two most frequently encountered causes of benign enlargement of mediastinal lymph nodes are tuberculosis and sarcoidosis. In both conditions, the clinical, roentgenographic and laboratory findings yield sufficient clues to arrive at a diagnosis. Biopsy of superficial or prescalenic lymph nodes is often rewarding.

Hodgkin's disease and lymphosarcoma are common causes of massive enlargement of mediastinal lymph nodes. X-ray examination will reveal multiple lesions in the mediastinum and often also at the hilar lymph nodes. The cervical lymph node chains become involved eventually in most cases. Biopsy of accessible lymph nodes or of nodes of the anterior scalene region and the blood picture may considerably assist diagnosis. The "therapeutic test" of radiation therapy may be employed in a limited number of cases when node biopsy does not establish the diagnosis. Lymph node enlargement in the mediastinum is noted roentgenographically in about 20% of cases of leukæmia.

Finally because of the richness of its lymphatic connections, the mediastinum is a frequent site for metastatic deposits, in particular from the lung (bronchogenic carcinoma). It happens occasionally that the enlarged nodes are more prominent than is the primary tumour. Bronchoscopy, cytological examination of bronchial secretions or biopsy of accessible lymph nodes usually establishes the correct diagnosis. Carcinoma of the breast, carcinoma of the gastrointestinal tract, or other malignant disease originating within the abdominal cavity or elsewhere may spread to the mediastinal lymph nodes. In such circumstances, the physician is only rarely misled if a complete physical examination has been carried out.

Fig. 24.—Case 6—thymic cyst.

Diaphragmatic hernia through the foramen of Morgagni is a condition which inevitably features in the differential diagnosis of primary tumours of the anterior mediastinum when the abnormal shadow is immediately above the diaphragm. Gastrointestinal series and the induction of an artificial pneumoperitoneum may assist diagnosis.

When an abnormal shadow in the anterior mediastinum is thought to be a primary lesion, its precise nature and its innocence or malignancy should be established before operation. For this purpose, radiological studies are most rewarding. The comparison of current and previous films, if available, may help in determining the age of the lesion and its rate of growth. Benign tumours grow very slowly and malignant tumours may or may not grow slowly. Tomography with antero-posterior and lateral views is a valuable tool for exploration of the anterior mediastinum, which is away from heavy bony structures. These radiological studies can accurately outline the margins of a lesion and determine its relationship to adjacent structures.

A well demarcated and spherical border is usually associated with a benign tumour. This finding coupled with homogenous density on all laminar section films suggests a cystic lesion. A nodular or embossed contour suggests not only a solid tumour rather than a cyst but also the possibility that it is malignant.

Cystic lesions of the anterior mediastinum, as well as partly cystic or solid tumours, should be removed as soon as a definite diagnosis of primary tumour has been established. These lesions are malignant or potentially malignant. With progressive enlargement, they will cause more and more severe pressure symptoms and perhaps greater technical problems when operation is decided upon. Tumours of the anterior mediastinum are frequently cystic; they may become infected, with the danger of rupture with mediastinitis or empyema.

SUMMARY

The cystic lesions encountered in the anterior mediastinum are pericardial cysts, bronchogenic cysts, teratoid cysts and thymic cysts.

Differing in their histological character, these cysts owe their origin to a common factor, the developmental complexity of the region.

Complete radiological studies are usually more rewarding than physical examination as regards accurate diagnosis of an abnormal shadow in the anterior mediastinum. Tomography in particular is often useful in determining its origin and in fixing the exact relationships.

Cysts and cystic lesions of the anterior mediastinum should be surgically removed because of the possibility of pressure symptoms, malignancy or infection.

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RÉSUMÉ

Les tumeurs les plus fréquentes du médiastin antérieur sont les kystes bronchogènes et péricardiques, les tumeurs tératoïdes et les thymomes. Près de 50% des tumeurs du médiastin se trouvent dans la portion antérieure. Elles sont souvent kystiques; les thymomes cependant sont presque toujours solides et les tératomes participent aux deux formes.

Le diagnostic différentiel comprend les lésions suivantes: le goitre intra-thoracique qui cliniquement devrait bouger à la déglutition et dont on peut déceler la présence par l'emploi de radio-iode. L'anévrysme des gros vaisseaux qui devrait montrer une certaine pulsation à la fluoroscopie et que l'angiographie met en relief. La sérologie peut donner des indications utiles dans ces cas. Le carcinome bronchogène primaire, dont la présence peut être révélée à la bronchoscopie et confirmée par l'étude cytologique de frottis de sécrétions obtenues des deux côtés. Les ganglions lymphatiques hypertrophiés peuvent se trouver dans les cas de tuberculose et de sarcoïdose. Il faut donc dans ces circonstances faire la recherche méthodique des causes de lymphadénopathie et procéder à une biopsie des ganglions préscaléniques ou d'autres ganglions d'accès plus facile. On doit se rappeller que des dépôts métastatiques de foyers éloignés peuvent quelquefois donner naissance à des masses ganglionnaires thoraciques. La hernie diaphragmatique au travers du foramen de Morgagni peut être dépistée à l'aide du repas baryté ou du pneumopéritoine.

Le chirurgien doit faire toutes les recherches nécessaires pour déterminer la nature exacte des ombres radiologiques avant de procédér à l'opération. A cette fin il doit avoir recours à la comparaison avec les films antérieurs s'ils existent ou à la tomographie si les plaques simples sont insuffisantes. Une ombre offrant une démarcation nette est souvent causée par une tumeur bénigne. S'il existe une densité homogène sur chaque coupe tomographique on peut soupçonner la présence d'un kyste. Une apparence nodulaire avec des bords flous suggère la présence d'une tumeur maligne.

Toutes les tumeurs bénignes doivent être réséquées. Elles peuvent causer des symptômes par la pression qu'elles exercent sur d'autres organes et un grand nombre d'entre elles renferment des possibilités cancéreuses. Les kystes peuvent s'infecter et donner lieu à une médiastinite.

THE RECOVERY OF TUMOUR CELLS FROM VENOUS BLOOD DRAINING CEREBRAL GLIOMAS

A Preliminary Report*

T. P. MORLEY, F.R.C.S., *Toronto*

As part of a larger investigation by the departments of surgery and surgical pathology, Toronto General Hospital, into the presence of tumour cells in the peripheral blood of patients with cancer, it was decided to submit samples of blood drawn from the superior sagittal sinus in cases of cerebral tumour. The fact that all of the first three cases so investigated had tumour cells in the venous blood draining the area of the tumour, and that two of these tumours were gliomas, seems of sufficient interest to be reported.

Material

One metastasis from a bronchogenic carcinoma and two gliomas were studied. All three tumours were selected in this preliminary investigation with particular regard for their anatomical position. It was necessary for the venous blood draining the tumour to be easily accessible at the time of operation, therefore tumours in the frontal and frontoparietal parasagittal area were chosen, so that the superior sagittal sinus immediately posterior to the tumour could be conveniently exposed at the same time. Blood samples (5 ml.) were taken from the sinus through a No. 20 hypodermic needle and transferred immediately to heparinized tubes. After centrifugation, films were made from the buffy layer and stained with a modified Wright's stain.

Case 1. N/S 5566/15/59. — Bronchogenic carcinoma. Tumour cells were seen in samples taken from the sinus (a) immediately after the bone flap was turned but before the dura was opened, (b) during manipulation of the tumour, and (c) after the tumour had been removed by suction. No cells were recovered from the antecubital vein after operation.

Case 2. N/S 5515/66/58.—Mixed oligodendroglioma-glioblastoma. Blood samples from the sinus were negative for tumour cells immediately after the bone flap was turned, but were present after a brain needle had been passed into the tumour through the dura mater (Fig 1). Further samples removed during the excision of the tumour were negative, and blood taken some hours later from the antecubital vein was negative for tumour cells.

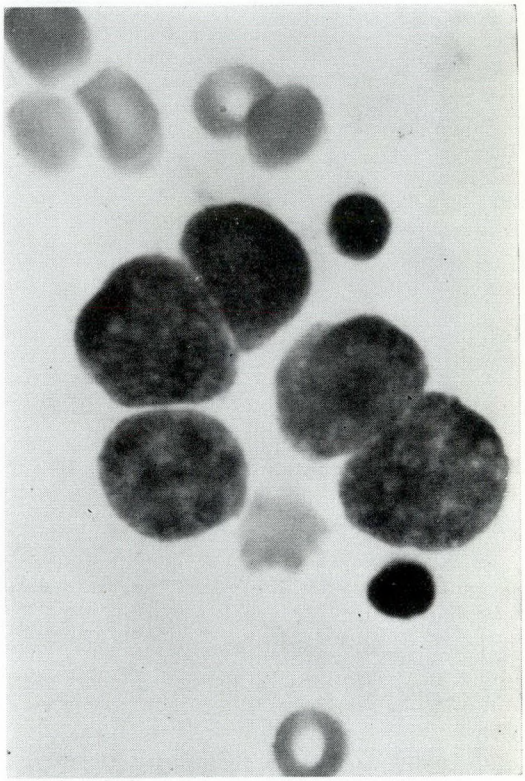

Fig. 1.—Case 2. Oligodendroglioma-glioblastoma. A clump of tumour nuclei in sagittal sinus blood. Wright's stain (x 2000).

Case 3. N/S 5595/31/59. — Glioblastoma multiforme. Positive samples were obtained from sinus blood after the bone flap had been turned but before the dura or tumour had been disturbed (Figs. 2 and 3). A second sample taken during enucleation of the tumour was also positive. No specimen was taken from the antecubital vein.

Discussion

This is not the place to discuss in general the phenomenon of tumour cells in

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

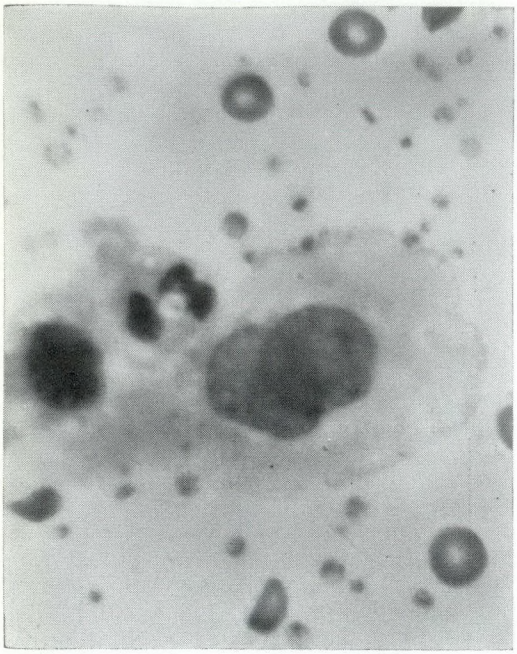

Fig. 2.—Case 3. Glioblastoma. Tumour cells in sagittal sinus blood. Wright's stain (x 1414).

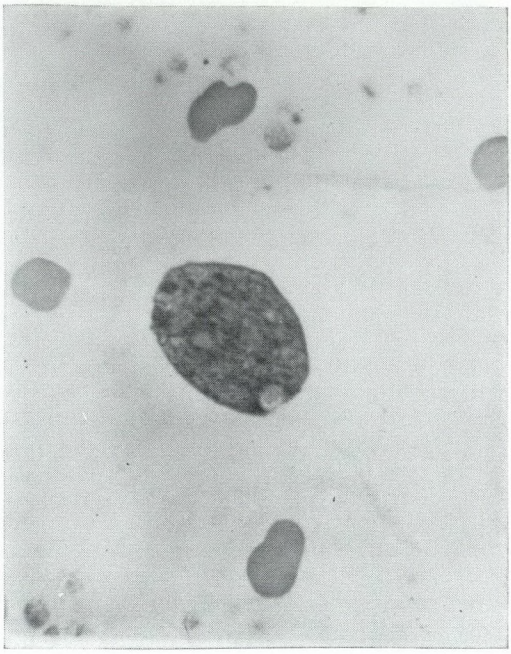

Fig. 3.—Case 3. Glioblastoma. Tumour cell nucleus in sagittal sinus blood. Wright's stain (x 1414).

circulating blood. For that the reader is referred to Engell,[1] Moore, Sandberg and Schubarg,[2] and Sandberg and Moore.[3] Furthermore, no useful general conclusions can be drawn from such a meagre series. The presence of tumour cells in the sagittal sinus in the case of the metastatic carcinoma may merely have been part of a systemic invasion from the primary growth or other metastases in spite of the absence of tumour cells in the sample of blood from the antecubital vein. The presence of tumour cells in the blood draining the two gliomas, however, is a different matter, for the occurrence of gross metastases outside the central nervous system from this type of tumour is very rare indeed; many deny that it ever takes place (e.g. Zulch,[4] Tom[5]). It has never been known why malignant gliomas do not metastasize, since from their microscopical appearance there is every reason to expect them to do so. To say that the conditions for growth outside the central nervous system are unfavourable is to beg the question. The absence of metastases still leaves the gliomas a group apart, although even in carcinoma and sarcoma only a very small proportion of tumour cells released into the circulation give rise to tumours.

ACKNOWLEDGMENT

The blood specimens were examined and reported by Dr. William Anderson; I am grateful to him for placing them at my disposal.

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RÉSUMÉ

Travaillant à un projet sur le dépistage des cellules tumorales dans le sang circulant, l'auteur a étudié ce qui se passait au niveau du sinus longitudinal supérieur dans des cas de tumeurs cérébrales. Dans chacun des trois premiers essais ainsi entrepris, des cellules cancéreuses furent trouvées.

Le sang fut prélevé par ponction à la seringue (5 c.c.) et immédiatement transféré dans des tubes avec héparine; après centrifugation, des étalements furent faits sur lames et colorés au Wright.

Cas 1.—Métastase cérébrale d'un carcinome bronchique: les cellules cancéreuses sont trouvées dans les échantillons de sang prélevés pendant

l'ouverture de la calotte, pendant la manipulation de la tumeur et même après la fin de l'ablation. Cas 2.—Oligodendrogliome-glioblastome: les échantillons sanguins prélevés lors de l'ouverture de la calotte sont négatifs; ils deviennent positifs au moment où l'on passe une aiguille dans la tumeur à travers la dure-mère; ils redeviennent négatifs ultérieurement. Cas. 3.—Glioblastome multiforme: les échantillons sont tous positifs. L'auteur termine par quelques rappels de littérature.

THE EFFECTS OF GRADUAL COMPLETE OCCLUSION OF THE HEPATIC VEINS*

P. B. R. ALLEN, M.D. and S. KLING, M.D., F.R.C.S.[C], *Edmonton, Alta.*

PHYSICIANS have long been frustrated in their attempts to understand and treat the problem of portal cirrhosis and its sequelæ of portal hypertension and ascites. It was a desire to better understand the basic physiology of this problem which prompted the present study.

In 1954, Madden *et al.*[1] working with corrosion specimens of the liver postulated that "the primary factor in the formation of ascites is an obstruction of the outflow tract of the liver, namely, the hepatic veins. In cirrhosis of the liver, with irreversible ascites the obstruction is due to an obliterative fibrosis of the intrahepatic systemic venous bed." These findings greatly interested us and stimulated speculation on what results would be produced by a gradual occlusion of the hepatic veins. A survey of the literature revealed that there were established techniques for producing portal cirrhosis; the injection of substances toxic to the liver such as carbon tetrachloride[2, 3] and the use of liver irritants such as silicon dioxide as described by Volwiler,[4] but there was little evidence of any work on the production of hepatic venous occlusion. Earlier workers, such as Brandes[5] and Armstrong and Richards,[6] have reported on the results of acute occlusion of the hepatic veins, but no reference could be found to techniques for the gradual occlusion of these veins.

As a preliminary study, the hepatic veins in experimental animal cadavers were dissected, and this demonstrated that these veins are too inaccessible to permit a direct surgical attack. In view of these findings, considerable doubt was felt that the techniques described by previous investigators for the production of acute occlusion of the hepatic veins had indeed resulted in such occlusion. It was apparent that an indirect approach would have to be utilized to produce occlusion of the hepatic veins. We have finally developed a technique which is consistent, relatively simple and, to the best of our knowledge, original. The method is as follows:

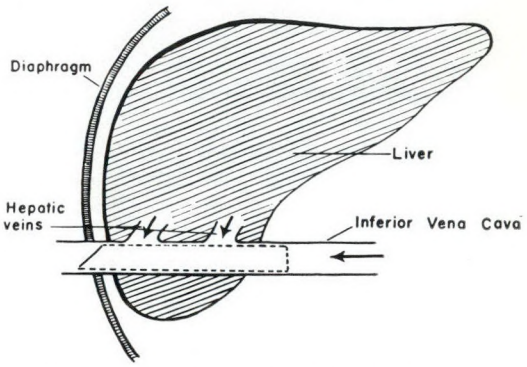

Fig 1.—Diagram depicting lateral projection of liver and inferior vena cava. Dotted lines illustrate position of plastic cannula. The wall of the cannula is in apposition to the ostia of the hepatic veins, thus interfering with the drainage of blood from the liver.

METHOD

Twenty adult mongrel dogs ranging in weight from 12.5 to 20.0 kilograms were selected for this experiment. The animals are anæsthetized with sodium pentobarbital (30 mg. per kilogram of body weight)

*From the Department of Surgery and the McEachern Cancer Research Laboratory, University of Alberta Faculty of Medicine, Edmonton, Alberta 1958 Medallist Paper—Royal College of Surgeons of Canada.

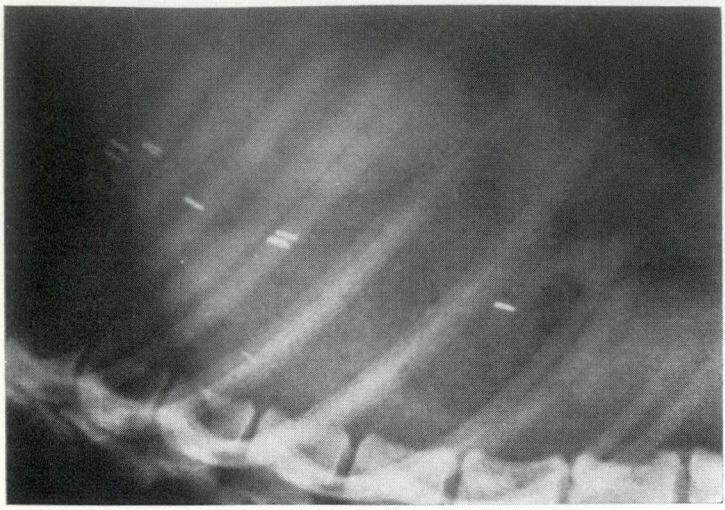

Fig. 2.—Lateral radiogram of dog. Silver clips have been placed along the cannula which has been inserted into the inferior vena cava. The most cephalad end of the cannula lies at the level of the diaphragm. The most caudad end of the cannula lies just caudal to the liver.

intravenously, and the anterior abdominal wall is prepared by shaving, washing with soap and water and double application of 2.5% tincture of iodine.

The abdomen is opened through a midline incision. The portal vein and inferior vena cava are identified and venous pressures in the vessels are taken. To take these pressures a thin-walled 15-gauge needle is introduced into the lumen of the vessel through an area of vein wall enclosed by a purse-string suture of 4-0 vascular silk. The needle is connected by a short segment of rubber tubing to a simple water manometer, both of which have been previously filled with an heparin-saline solution. The base level of the manometer is judged to be at the level of the right atrium of the

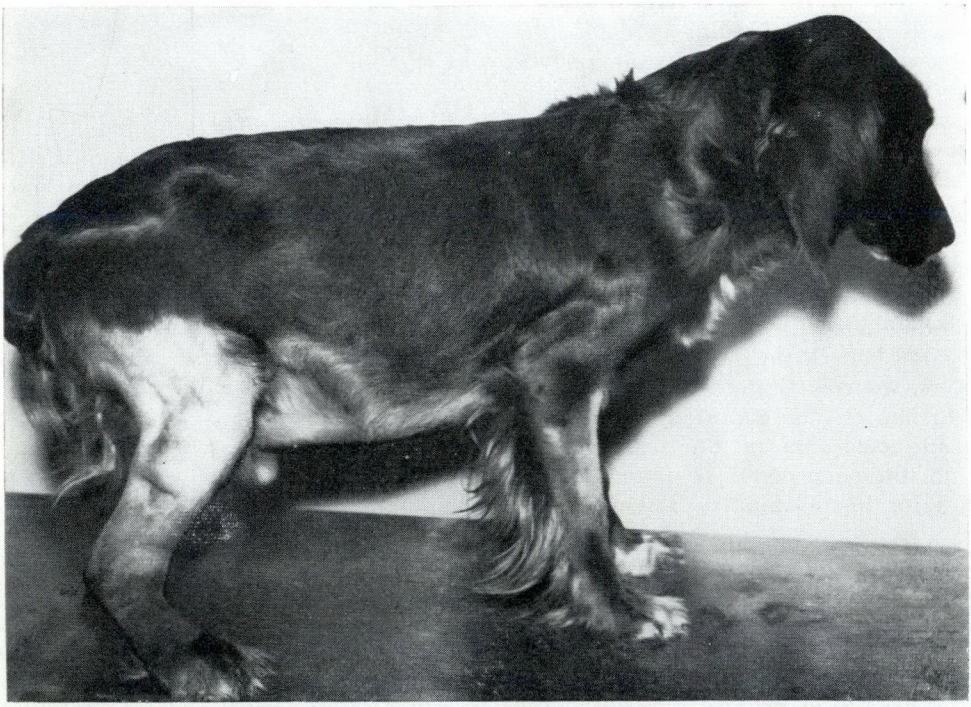

Fig. 3.—A dog eight weeks following introduction of plastic cannula into the inferior vena cava. Marked abdominal distension and pitting œdema over the hind legs is evident.

heart and all pressure readings are taken with this feature constant. Before withdrawing the needle from the portal vein, 20 c.c. of 70% diodrast (Iodopyracet-Winthrop) is injected and a roentgenogram taken. The needle is then withdrawn and the purse-string suture made taut so as to prevent an extravasation of blood from the puncture site.

A segment of the inferior vena cava cephalad to the renal veins is mobilized for a distance of three to four centimetres. Blalock vascular clamps are placed at the upper and lower ends of this segment of vein. A piece of thin-walled, wide-bore plastic cannula is then selected of such length that it reaches from just caudad to the liver to a point just cephalad to the diaphragm (Fig. 1). A suture of 4-0 vascular silk is then placed on the caudal end of the cannula to act subsequently as an anchor suture for the cannula. The cephalad end of the cannula is tapered to facilitate its introduction into the inferior vena cava. Several small silver neurosurgical clips are placed on both ends of the cannula to permit radiological localization of the cannula when it is in place (Fig. 2). The Blalock clamps are closed and an incision 1.5 cm. long is made in the anterior wall of this isolated segment of the vena cava. The bevelled end of the cannula is introduced into the phlebotomy incision while an assistant releases the cephalad Blalock clamp so that the cannula is fully inserted into the lumen of the vessel. The anchor suture of the cannula, referred to above and which has the needle still attached, is brought out through the vein wall at the caudal end of the incision. This same suture is then utilized to close the incision in the wall of the vena cava. A simple running stitch is employed for this purpose. In earlier cases we had not anchored the cannula in this fashion, with the result that it was rapidly carried to the right atrium of the heart and led to the death of the animals. A neurosurgical type of suction is found to be the best method for achieving visualization of the wound during closure of the opening in the vena cava. The plastic cannulæ are made of polythene, and in each operation we select one with the largest calibre which can be conveniently

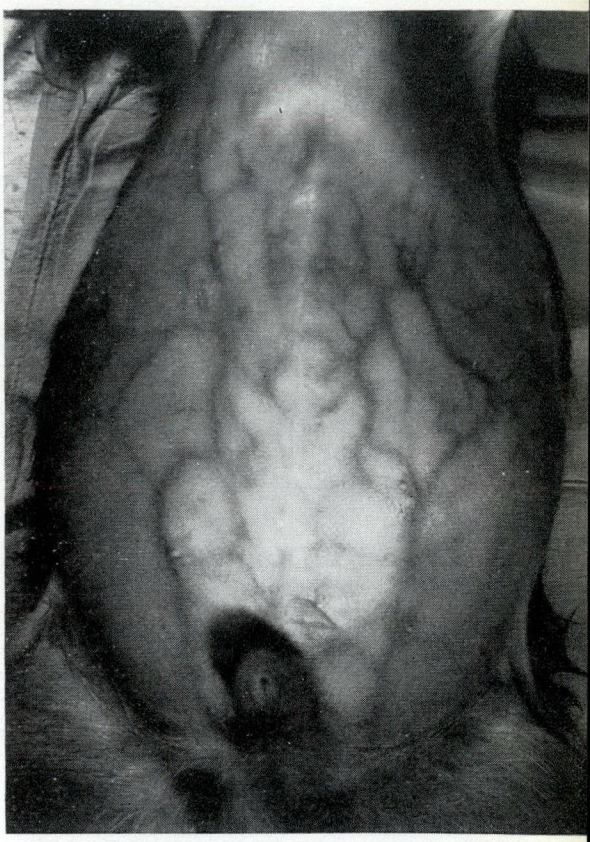

Fig. 4.—Fig. 4 shows marked abdominal distension by acitic fluid and evidence of prominent collateral venous channels in the abdominal wall.

inserted into the vena cava. The abdomen is closed in layers without drainage. As experience increased, we found that the entire operative procedure could be carried out in approximately 45 minutes with negligible mortality. The blood loss is usually small. It is apparent from the above technique that besides any effect the presence of the cannula might have upon the hepatic veins, it could well influence venous return to the heart through the vena cava. To help us assess this factor, a control experiment was devised. Four dogs were selected. The operation was repeated employing a technique identical to the one first described, with the following exception. In this group a short cannula of the same material was used, being placed in the vena cava just cephalad to the renal veins but well caudad to the point of entry of the hepatic veins into the vena cava. The same biochemical

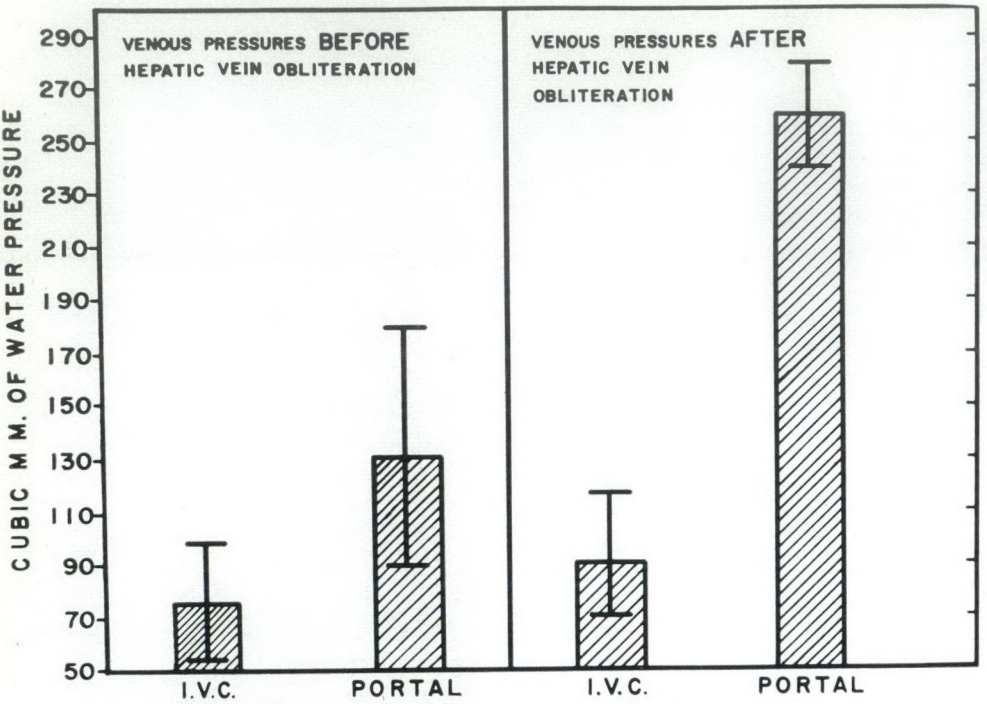

Fig. 5.—This chart demonstrates the pressure means and ranges taken in the inferior vena cava and portal veins before and after obliteration of the hepatic veins.

studies were made as in the main experiment.

Biochemical Assessment

Preoperatively the following studies are done on each animal:

1. Total serum proteins and fractionation. The constituent proteins are analyzed in a Spinco paper electrophoresis cell after the method described by Jencks, Jetton and Durrum.[7]

2. Serum sodium and potassium levels are determined by the Baird flame photometer.

3. Serum chloride is determined by the method of Schales and Schales.[8]

4. Bromsulphalein dye retention is measured after the technique of Rosenthal and White[9] as described and modified by Helm, Mateer, and Machella.[10-12]

These studies are repeated at two-week intervals following the operation.

When ascites became clinically evident the animals were re-operated upon. Direct pressure readings were again taken from the inferior vena cava and portal vein. In a certain number of cases portal venography was repeated. When the animals were sacrificed, necropsy specimens were taken from the liver, spleen, kidney and the inferior vena cava in the region of the hepatic veins.

Results

A. *Clinical Findings*

Four of the animals survived the operative procedure but died within a few days, and before developing any evidence of ascites. One of these animals died of pulmonary infection and one died of generalized peritonitis. In the other two cases the cannula broke free and caused death by impacting in the right atrium of the heart.

Ten of the animals developed clinically demonstrable ascites within six to eight weeks of the operation. In these the abdomen became markedly distended and tortuous venous collateral channels became prominent in the abdominal wall (Figs. 3 and 4). When the ascites reached full clinical proportions, the animals developed a pitting œdema over the hind legs. The amount of ascitic fluid present varied with the size of the animal, but ranged from 3500 to 5500 c.c.

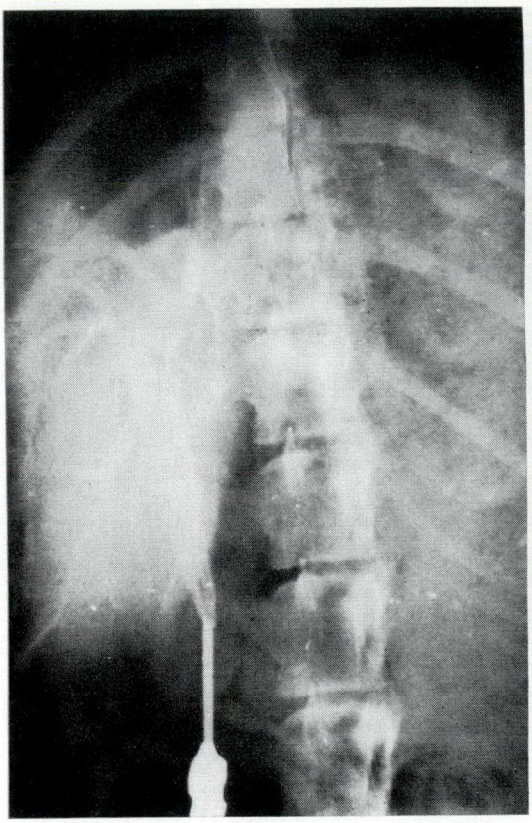

Fig. 6a.

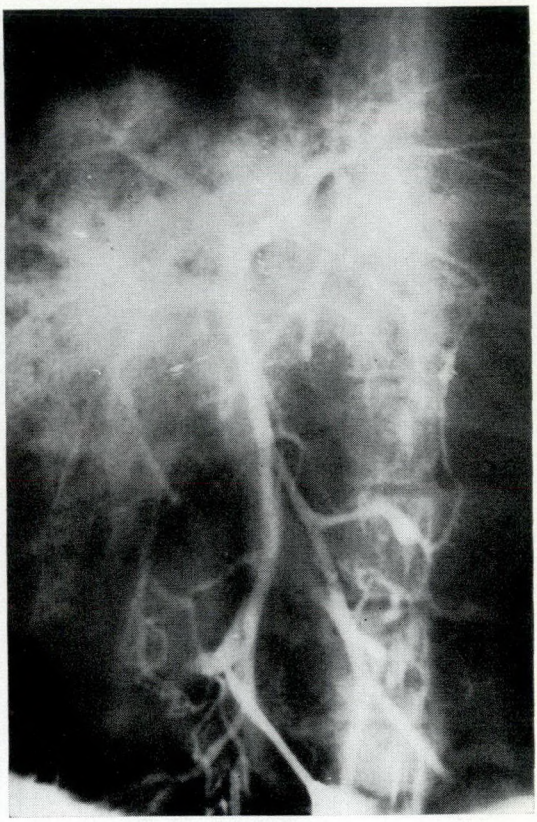

Fig. 6b.

Fig. 6a.—Portal venogram taken at initial operation showing the normal picture of the portal system in the dog. **Fig. 6b.**—The portal venogram taken after the establishment of ascites and portal hypertension. Tortuosity and pooling in the portal system are evident and multiple collateral portal-systemic shunts can be seen.

In the remaining two dogs which survived the operation no demonstrable ascites developed nor was there any appreciable alteration in the biochemical studies. Both were re-operated upon to discover the cause. In each of these two cases it was found that the cannula was too short and that patent hepatic veins, of good size, were emptying into the vena cava cephalad to the cannula.

The 10 animals which survived the operative procedure and did develop clinical ascites showed the following changes: The inferior vena caval pressures taken at the time of the original operation in this group averaged 75.3 mm. of water with a range as indicated in Fig. 5. The comparable portal vein pressures averaged 130.5 mm. of water with a range as indicated in Fig. 5. When the animals were re-operated upon after establishment of ascites, the inferior vena cava pressures averaged 90.5 mm. of water and the portal vein pressures averaged 260 mm. of water (see Fig. 5). It was interesting to note that in this group the portal-systemic collateral venous channels were very obvious and troublesome when the anterior abdominal wall was incised. There was marked dilatation and tortuosity of the mesenteric veins, as further evidenced by the portal venograms. Fig. 6a shows a typical portal venogram taken at the time of the primary operation. Venograms taken at the time of the second operation (Fig. 6b) show marked tortuosity of the entire portal system with prominent collateral channels particularly between the portal and lumbar veins. Blood was pooled in the portal regions of the liver giving a radiological picture similar to that described by Atkinson *et al.*[13] in cases of established portal hypertension.

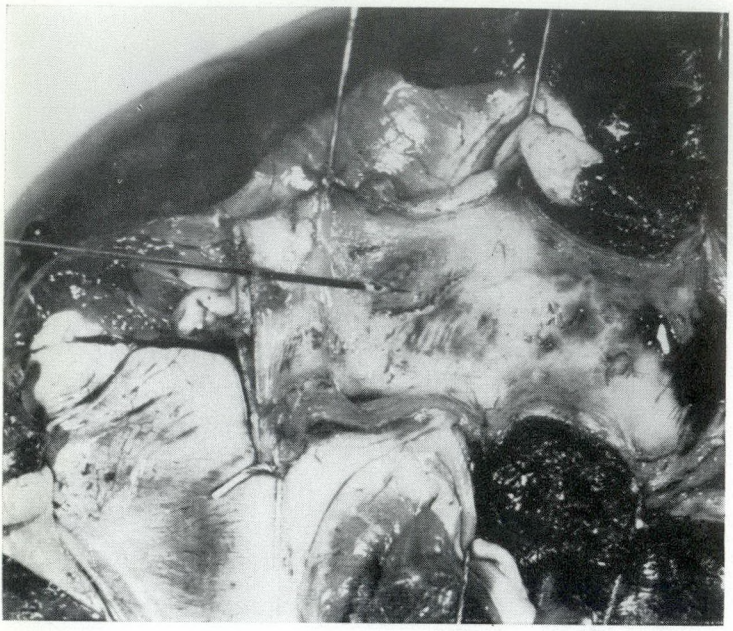

Fig. 7.—The inferior vena cava has been opened at autopsy in the region of the hepatic vein ostia. The scarred area at the tip of the pointer marks the position of an obliterated hepatic vein ostium.

In each of the 10 animals the peritoneal cavity was filled with a clear yellow fluid of high specific gravity which had a protein composition very similar to that of the plasma of the animal.

Striking changes were noted in the wall of that portion of the vena cava which contained the plastic cannula. The wall was markedly thickened, and examination of its intima showed that the ostia of the hepatic veins had been obliterated by organized thrombi in the lumen of each vein. Various stages of thrombus formation and organization could be recognized in serial sections of the hepatic veins (see Figs. 7 and 8).

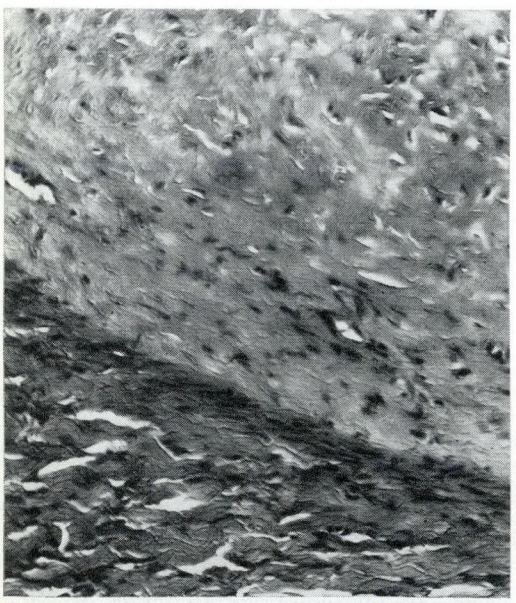

Fig. 8.—Photomicrograph of thrombus organization in the lumen of a hepatic vein. The darker tissue represents the original wall of the hepatic vein, the lighter tissue being the organized thrombus.

Sections of the liver taken at necropsy revealed marked congestion in the central region of the liver lobules. Fibrosis of the liver substance was not a prominent feature in any of our animals. Sections of the spleen and kidneys revealed a moderate degree of congestion.

The four animals used in the special control experiment all remained healthy for a six-month period following the operation. There was no evidence of clinical ascites and the biochemical findings remained unchanged. When they were sacrificed, the cannula was patent in two of the animals and in the other two it was completely thrombosed, indicating that in the latter two cases, the factor of vena caval obstruction had been reproduced. In none of these animals was there any ascites or any evidence of portal hypertension. In none of these animals was there any evidence of occlusion of the hepatic veins. This would indicate that the pathological changes in the original series were due to occlusion

of the hepatic veins and not to occlusion of the inferior vena cava.

B. *Biochemical Findings*

As mentioned above, the biochemical studies were repeated at two-week intervals. These demonstrated several interesting trends. The animals showed an overall decrease in total serum protein with a reversal of the albumin-globulin ratio. There was marked fall of albumin and beta globulin values with relative increase in the gamma globulin fraction (Table I).

The bromsulphalein dye retention increased as the ascites progressed (Fig. 9), reflecting increasing functional impairment of liver cells.

Serum sodium, potassium and chloride values showed little fluctuation and remained essentially normal throughout the experiment.

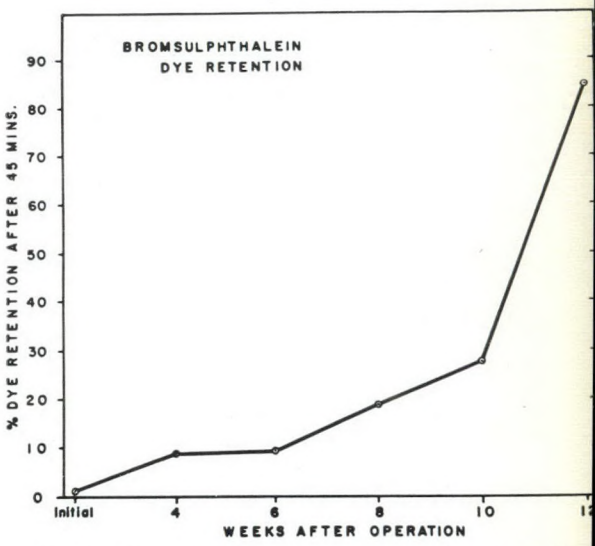

Fig. 9.—demonstrates the progressive increase of r tension of bromsulphthalein dye over a period of 12 week This reflects the increasing liver cellular function impai ment which occurred as ascites and portal hypertensi became established.

Discussion

The preceding data would appear to show that marked ascites and a degree of portal hypertension are produced in all animals in which this procedure is correctly employed. It is necessary to stress the importance of accurately placing the cannula so that fibrosis of the ostia of all the hepatic vein is produced. Failure to obliterate even one large hepatic vein may permit enough venous outflow from the liver to prevent the formation of ascites and portal hypertension. We feel that the control experiment described demonstrates that the important factor in the production of portal hypertension and ascites is the occlusion of the hepatic outflow tract, and that the inferior vena caval obstruction which occurs in some of the animals is not an important factor.

TABLE I.—Serum Proteins Values in Dogs Before Initial Operation (B), and after Ascites Formation (A).
Components Expressed as Percentage of Total Planimeter Areas.

Dog		Albumin	Globulins			
			α_1	α_2	β	γ
KA1	B	28.7	4.1	13.3	32.7	21.2
	A	18.1	9.0	7.8	23.1	42.0
KA5	B	35.0	5.7	10.2	31.0	18.1
	A	28.5	9.6	18.6	17.8	25.6
KA6	B	42.3	5.1	12.2	19.6	20.8
	A	30.9	9.2	20.6	14.0	25.5
KA8	B	39.8	12.4	10.4	24.1	13.3
	A	28.7	6.2	28.7	14.6	21.8
KA9	B	38.1	6.7	15.1	30.1	10.0
	A	32.5	10.3	23.3	15.0	18.9
KA15	B	33.5	10.8	6.9	34.2	14.6
	A	25.6	11.9	10.2	24.4	27.9

Although the amount of ascites produced is very striking, the degree of portal hypertension is not nearly so marked. It is our opinion, as well as that of other workers in this field, that the portal pressure is not strikingly elevated because of the formation of abundant collateral channels in the dog.

This procedure is relatively simple technically and produces uniform results. With such an aid we think that we are now able to produce portal hypertension and ascites in the experimental animal with comparative ease, and thus provide a source of experimental material by which we may assess the relative merits of the various types of therapy currently prescribed for these conditions.

Summary

A new method for producing a gradual, complete occlusion of the hepatic veins is described. Associated with this is the successful production of ascites and a degree of portal hypertension.

ACKNOWLEDGMENT

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RÉSUMÉ

Les nombreuses tentatives expérimentales en vue de reproduire le syndrome de l'hypertension porte dans la cirrhose n'ont pas connu grand succès jusqu'à présent. C'est avec l'idée d'étudier la cirrhose hépatique et l'ascite de façon physiologique, que les auteurs ont mis au point un technique d'occlusion du système sus-hépatique.

Leurs expériences furent faites sur des chiens de 12 à 20 kg. Après avoir procédé à une laparotomie médiane, on prépare la veine cave inférieure sur 3 à 4 cm. de long du côté caudal; on y introduit ensuite un cathéter de plastique de calibre assez gros pour occuper toute la lumière veineuse, et de longueur telle que son extrémité supérieure soit au niveau du diaphragme et son extrémité inférieure juste à hauteur de la phlébotomie. Ce catheter est enfoncé en totalité et l'incision veineuse est réparée. Le retour du sang hépatique vers l'oreillette droite se trouve donc totalement bloqué.

Une vingtaine de chiens furent ainsi opérés, dont 10 développèrent un syndrome ascitique. Chez ces derniers, l'abdomen devint très tendu en huit semaines environ, et une circulation collatérale s'installa. Ces chiens ont été étudiés au point de vue physiopathologique. Les pressions veineuses dans les systèmes cave et porte furent mesurés, et des angiographies pratiquées. Différents dosages et épreuves de laboratoire démontrèrent bien l'apparition progressive d'un trouble hépatique. Cette progressivité semble liée à la création d'un thrombus qui vient compléter l'occlusion autour du cathéter.

Cette nouvelle technique présente l'avantage de n'occasionner qu'une faible mortalité opératoire des animaux d'expérience.

AN EXPERIMENTAL STUDY OF TENDON SUTURING TECHNIQUES*

ROBERT J. COWAN, M.D., F.R.C.S.[C], and A. DOUGLAS COURTEMANCHE, M.D.,
Vancouver, B.C.

THE PURPOSE of this work was to compare the relative merits of six tendon suturing techniques commonly used in hand surgery.

These are:

1. Modified Bunnell method No. 2 with 4-0 silk;[1]
2. Modified Bunnell method No. 2 with 34 gauge stainless steel wire;[1]
3. Mason and Allen suture with 4-0 silk;[2]
4. Yaeger wire-and-barb pull-out suture;[3]
5. Pulvertaft's interlacing method with 40 gauge stainless steel wire;[4]
6. End-through end-to-side interlacing method with 4-0 silk.[5]

Successful tendon surgery depends on accurate diagnosis, asepsis, atraumatic technique, hæmostasis and careful rehabilitation. How much is demanded of the tendon suture itself? Bunnell stated that the suture should not strangle tissue, that the suture material should be reduced to a minimum, and that a buried suture is preferable to a surface stitch; he also indicated that less tissue reaction was created by stainless steel wire.[5] For Mason, the ideal tendon suture should anchor firmly in the tendon, produce a minimum of trauma, use a minimum of material, and avoid knots between the tendon stumps.[6, 7]

By experimental methods we have attempted to determine the validity of these statements. The main points used for comparison were: the relative simplicity of technique; the degree of reaction; the separation of the tendon stumps, and the effect on the healing process as measured by tensile strength.

EXPERIMENTAL METHODS

Rabbits weighing between 5 and 10 lb. were used. Anæsthe ia with intravenous pentobarbital (Nembutal) followed by ether was found most satisfactory. Aseptic technique was observed. The tendon which served our purpose best was the flexor digitorum longus in the hind limb. In addition the tendon of the tibialis posterior was used for both of the interlacing methods. To measure the separation after suture, wire sutures, placed 1¼ in. (3 cm.) apart, were used as markers. The tendon was divided midway between these markers. With the accordion effect produced, in particular by the Bunnell and Yaeger sutures, the distance between the markers after suture was often reduced to ⅞ in. (2.2 cm.). Suturing was done proximal to the tendon sheath where the tendon is enveloped with paratenon. All sutures were begun ½ in. (1.25 cm.) from the divided ends. A tourniquet was not used, for hæmorrhage was rarely a problem. Immobilization was achieved by the use of below-the-knee plaster casts with the ankle and toes in full plantar flexion. In this position no tension is exerted on the sutured tendon. All animals were immobilized for 21 days, as this has been well established as the optimum length of time during which tendon healing passes through the proliferative phase. Unrestricted active movement was then allowed through the maturation phase when the tendon regains its maximum strength.

Some difficulty was encountered early on in maintaining adequate immobilization, as a few of the animals chewed away most of their plaster. This was overcome by incorporating wire in the plaster and, later, by the use of a superior grade of plaster of paris bandage. Twenty tendon sutures of each type were performed; all were properly immobilized and free of infection. An additional 16 tendons were sutured to replace those discarded because of infection or inadequate immobilization.

The animals were sacrificed at intervals from two to 90 days; particular notes were made of the reaction, the distance between the markers and the tensile strength in grams per square millimetre. The degree of reaction was graded from zero to four plus. If the paratenon remained shiny and translucent and there were no adhesions, the tissue was considered to be free of

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*From the Department of Surgery, Faculty of Medicine, University of British Columbia, Vancouver, B.C.

TABLE I.—Tendon Suturing Techniques—Modified Bunnell No. 2 with 4-0 Silk

No.	Days	Reaction	Separation Markers	Separation Stumps	Gross pull Grams	Tensile strength G./sq.mm.	Average
OH105R	2	+	7/8″	7/16″	No healing; suture broken; inadequately immobilized.		
M2R	3	+	1/4	0	1270.0	75.6	
M2L	3	0	1/8	0	861.8	51.3	63.5
OH102R	4	+	1	5/8	No healing; suture broken; inadequately immobilized.		
M5R	5	0	1/8	0	1292.7	76.9	
M5L	5	0	3/16	0	1814.4	108.0	92.5
OH102R	7	+	1	5/8	No healing; suture broken; inadequately immobilized.		
OH101R	8	+	1-3/16	1/2	No healing; suture broken; inadequately immobilized.		
OH56R	8	++	1/8	0	1415.2	186.2	186.2
OH88R	9	+	1/4	1/8	1474.2	193.9	193.9
OH100R	10	+	1/8	0	1678.3	220.8	220.8
OH104R	11	+	1-1/16	5/8	No healing; suture broken; inadequately immobilized.		
M810R	13	++	3/8	1/16	1451.5	337.6	337.6
M820R	16	+	3/8	0	1133.9	298.4	
M820L	16	++	5/8	1/4	816.5	214.9	256.7
M804R	21	++	7/16	1/16	1315.4	346.2	
M804L	21	+	5/16	0	2041.2	537.2	441.7
OH60R	22	++	1/4	1/8	3175.1	417.8	417.8
M792R	28	++	7/16	1/8	1179.3	310.3	
M792L	28	+++	3/4	5/16	544.3	143.2	226.8
M824R	29	+++	3/8	3/16	2072.9	482.1	
M824L	29	+++	1/2	1/16	2267.9	527.4	504.8
41R	42	++	1/2	3/16	3084.4	717.3	
41L	42	++	3/16	0	3628.7	843.9	780.6
OH106R	90	+	1/2	0	7801.7	1026.5	1026.5

reaction. A hyperæmic paratenon with no thickening and with minimal œdema of the tendon ends was rated as plus one. Fine adhesions between tendon and surrounding tissue constituted a plus two. Plus three signifies more marked reaction in the form of denser adhesions, and a tendon completely enveloped in fibrous tissue with no gliding action was designated as plus four.

By measuring the separation of the markers, we could determine to what extent the accordion effect had been lost, as well as the separation of the tendon stumps. Tensile strength was determined by dividing the gross pull, in grams, necessary to separate the tendon ends by the cross-sectional area of the tendon in square millimetres. The cross-sectional area of each tendon was determined by measuring the large and small diameters and using the formula for the surface area of an ellipse. The gross pull necessary to break the tendon was obtained by the use of a tensiometer (Dillon tester), using a straight, gradual pull. Only in the case of the Yaeger wire-and-barb suture was the suture removed before testing.

Results

The disastrous results that follow inadequate immobilization or infection are again emphasized by this work. In 13 instances where plaster was removed from the foot alone, allowing free movement of the toes, the sutures were broken and the tendon ends widely separated. At 11

days, there was no evidence of any healing process between the stumps. In two cases at 19 days the reaction was so marked that the tendons could not be separated from their beds. In fact they could hardly be identified. In three infected legs the suture line was completely disrupted.

All techniques used are simple enough to be done atraumatically. The Yaeger wire-and-barb suture, while appearing the simplest, was actually the most difficult to carry out atraumatically. This suture as prepared by its manufacturer has much too large a needle on its distal end, and it could not be passed through the distal tendon segment with any degree of ease. The tendon would accordion away from the needle as it was inserted, and then invert markedly around the wire as it was passed, i.e., it would not glide over the wire readily.

It will be noted from Tables I, II, III, V and VI that the degree of reaction with these five techniques shows no significant variation. In the case of the Yaeger suture represented in Table IV there is a very significant increase in reaction.

The amount of separation of the tendon stumps is shown in Tables I to VI, and is summarized in Table VII. It is noteworthy that in every case, with the exception of six Mason and Allen sutures, the accordion effect was totally lost. This was found in the animals sacrificed as early as two, three, four and five days, as well as those sacrificed later. As might be expected the two interlacing methods were the only ones where no separation of the tendon stumps occurred. The Mason and Allen sutures showed somewhat less separation than the other three end-to-end methods, and the Yaeger sutures showed considerably more.

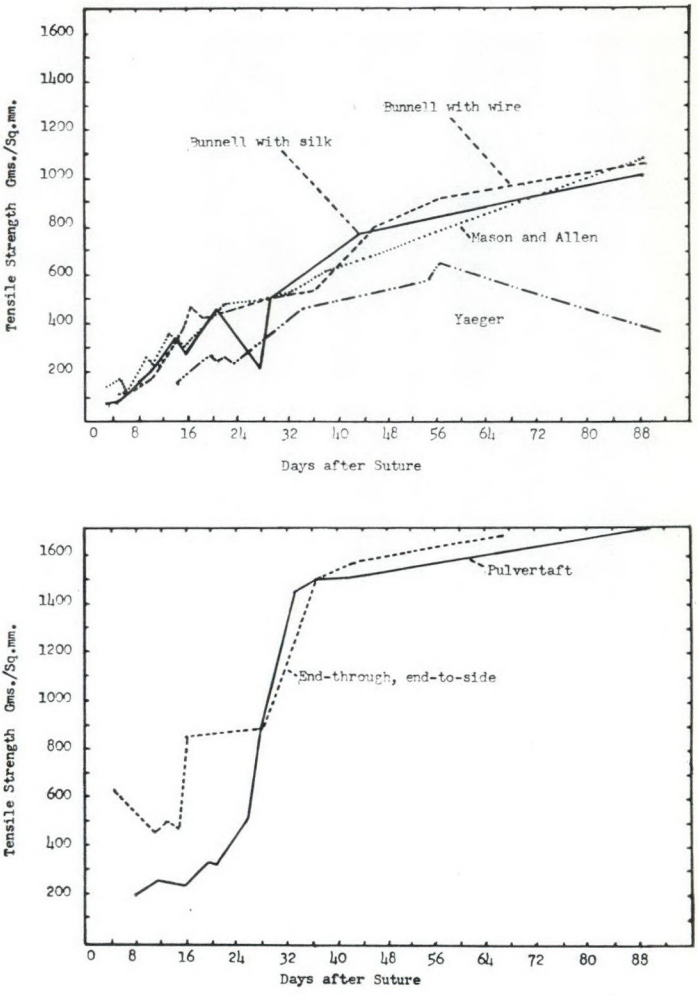

Fig. 1.

The tensile strengths as studied chronologically are seen most clearly on the two graphs (Fig. 1). It is interesting to note the similarity between the two Bunnell methods and the Mason and Allen method as plotted on the graph. The tensile strength of the tendons repaired by the Yaeger method was consistently less than of those previously mentioned, even after a period of 90 days. The interlacing methods showed little variation in tensile strengths after a period of four weeks. Prior to this the end-through end-to-side suture gave significantly higher values. The latter when tested on the tensiometer always separated initially at the junction of the small and the large tendon, i.e. the site of the end-to-side. In the Pulvertaft method it ruptured at the

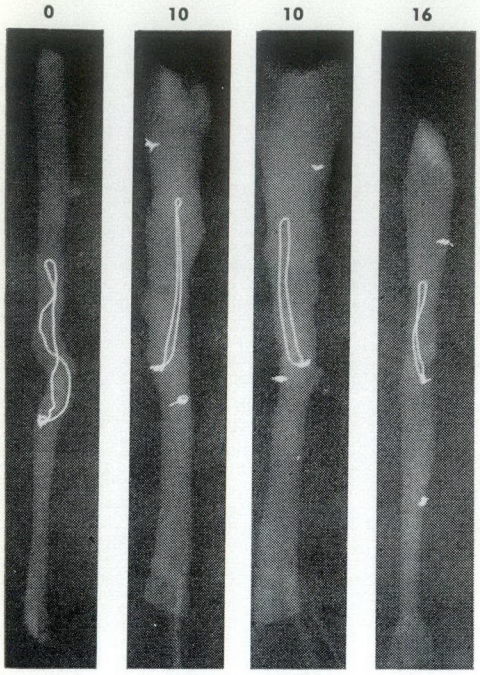

Fig. 2.

location of the first mattress suture. This was not true before 11 days. Up to this time the separation was by a gradual undoing of the interlacing. The tensile strengths of these two methods are considerably greater than of any of the other four methods tested, for a period of at least 90 days. The reaction of the interlacing methods compared favourably with the end-to-end methods, but the cross-sectional area at the suture site was usually greater.

Discussion

It is universally agreed that an atraumatic technique is necessary for successful tendon surgery. We think that the amount of trauma inflicted during this work did not vary significantly from one method to another. The Yaeger wire-and-barb suture, however, consistently showed more reaction than any of the others. Further, this is the only method which in every instance allowed at least ⅛ inch separation of the tendon stumps. This would certainly suggest that the reaction will be greater if the suture allows the tendon stumps to separate to the degree noted here. This statement is supported by the fact that with the other sutures the reaction was also greatest where separation was greatest.

The reason for this separation with the Yaeger suture appears obvious to us. The

TABLE II.—Tendon Suturing Techniques—Modified Bunnell No. 2 with 34 Gauge Stainless Steel Wire

No.	*Days*	*Reaction*	*Separation*		*Gross pull*	*Tensile strength*	
			Markers	*Stumps*	*Grams*	*G./sq.mm.*	*Average*
M4R	5	0	5/16″	0	1882.4	108.6	
M4L	5	+	3/8	1/16	1769.0	105.3	107.0
M132R	10	++	5/16	1/16	2766.9	164.6	
M132L	10	++	3/16	0	3628.7	215.9	190.3
42R	14	0	3/16	0	2812.3	370.0	
42L	14	+	1/4	0	2494.7	328.3	349.0
M802L	16	++	1/16	0	1814.4	477.5	477.5
38R	18	++	3/8	1/16	1542.2	405.8	405.8
M662R	21	+	7/16	0	3175.1	423.3	
M662L	21	+	7/16	1/8	3129.8	417.3	420.3
36R	27	+++	13/16	9/16	3900.9	513.3	
36L	27	++	5/8	3/8	3628.7	477.5	495.4
M226R	36	+	3/16	0	3991.6	525.3	
M266L	36	+	1/8	0	4195.7	552.1	538.7
264R	46	+	5/16	0	5955.6	794.1	
264L	46	+	5/16	0	5896.7	786.2	790.2
6R	54	+	1/4	0	7076.0	931.1	
6L	54	+	3/8	1/8	6894.6	907.2	919.2
M265R	90	++	5/8	1/8	7665.7	1008.6	
M265L	90	++	9/16	0	8255.3	1086.2	1047.4

TABLE III.—TENDON SUTURING TECHNIQUES—MASON AND ALLEN WITH 4-0 SILK

No.	Days	Reaction	Separation Markers	Separation Stumps	Gross pull Grams	Tensile strength G./sq.mm.	Tensile strength Average
OH105L	2	+	9/16″	3/8″	No healing; suture broken; inadequately immobilized.		
OH56L	3	+	0	0	1020.6	136.9	136.9
OH103L	4	+	9/16	3/8	No healing; suture broken; inadequately immobilized.		
M3R	5	+	1/8	0	1043.3	137.3	
M3L	5	+	1/16	0	1406.1	185.0	161.2
386R	6	0	1/16	0	594.6	79.3	
386L	6	0	0	0	907.8	121.0	100.2
OH102L	7	+	1/2	3/8	No healing; suture broken; inadequately immobilized.		
OH101L	8	+++	3/8	1/4	Marked infection; suture broken; inadequately immobilized.		
376R	9	0	3/16	0	1868.8	249.2	
376L	9	0	1/8	0	1927.7	257.0	253.1
OH88L	9	+++	1/4	1/4	816.5 Suture broken; inadequately immobilized.	108.9	
OH100L	10	+	0	0	1614.8	215.3	215.3
OH104L	11	++	13/16	1/2	No healing; suture broken; inadequately immobilized.		
M810L	13	++	3/8	0	1451.5	337.6	337.6
M852R	15	+	1/15	0	793.8	208.9	
M265L	15	+	1/16	0	1315.4	346.2	277.6
M778R	19	+	1/8	0	3016.4	402.2	
M778L	19	+	1/8	0	2902.9	387.1	394.7
OH60L	22	+	5/16	1/16	3229.6	430.6	430.6
29R	31	+	0	0	3628.7	483.8	
29L	31	++	0	0	3742.1	498.9	491.4
375R	34	++	5/16	1/8	3855.5	514.1	514.1
375L	34	++++	Marked infection; suture line completely disrupted.				
366R	38	+	0	0	4535.9	596.8	
366L	38	+	1/8	0	4649.3	611.8	604.3
OH106L	90	+	3/16	0	7937.8	1058.4	1058.4

holding power of the tendon for the suture was not sufficient to prevent the barb from travelling distally in the tendon. It is possible that this could be prevented to some extent by using the suture in the manner that Bunnell suggested for his Gig pull-out suture, that is, by making two oblique passes across its diameter before threading the suture straight down the centre of a tendon.[8]

The accordion effect produced by the Bunnell sutures, as mentioned previously, shortened the distance between the markers by ¼ to ⅝ inch, and this effect was completely lost by the second day. This was true whether wire or silk were used. The explanation for this occurrence may be found in the x-ray pictures of Fig. 2. Here are four tendons sutured by the modified Bunnell method; one is shown immediately, two at 10 days, and one at 16 days after suture. It will be noted in the three tendons to the right that the wire which was placed in the prescribed criss-cross fashion has now assumed the shortest distance between two points. Because silk was used with the Mason and Allen suture, it was not possible to determine radiologically whether this suture method, which involves two right angles for each tension suture,

TABLE IV.—Tendon Suturing Techniques—Yaeger Wire-and-Barb Pull-Out with 2-0 Tantalum

No.	Days	Reaction	Separation Markers	Separation Stumps	Gross pull Grams	Tensile strength G./sq.mm.	Tensile strength Average
M508R	15	++	3/8″	1/8″	1133.9	151.2	
M508L	15	+	1/4	1/8	1088.6	145.1	148.2
384R	20	+	3/8	1/4	1927.8	257.0	257.0
M840R	21	+++	5/16	1/8	1043.2	242.6	242.6
35R	22	+++	7/16	1/4	2267.9	302.4	
35L	22	+	1/4	1/8	1769.0	235.9	
OH24R	22	++	3/8	1/8	1632.9	217.7	
OH24L	22	+	5/16	3/16	1814.4	241.9	249.5
M826R	25	++++	7/16	1/4	907.2	238.7	
M826L	25	++++	7/16	1/4	816.5	214.9	226.8
M825R	35	+++	5/16	1/4	1632.9	429.7	
M825L	35	+++	7/16	1/4	1542.2	405.8	417.8
43R	42	++	5/16	1/8	3628.7	483.8	
43L	42	+++	3/8	1/4	3447.3	459.6	471.7
23R	55	+++	3/8	3/16	4626.2	608.8	
23L	55	++	3/8	3/16	4445.2	584.9	596.9
22R	56	++++	7/16	1/4	4490.5	590.8	
22L	56	++++	5/16	1/8	5488.4	722.2	656.6
M16R	90	++++	1-1/8	3/4	1133.9	298.4	
M16L	90	++++	1-1/8	3/4	1587.6	417.8	358.1

TABLE V.—Tendon Suturing Techniques—Pulvertaft with 40 Gauge Stainless Steel Wire

No.	Days	Reaction	Separation Markers	Separation Stumps	Gross pull Grams	Tensile strength G./sq.mm.	Tensile strength Average
M727R	8	+	3/8″	0	839.1	195.1	
M727L	8	+	1/4	0	861.8	200.4	197.8
M787R	11	+	1/8	0	635.0	147.7	
M787L	11	+	1/16	0	1700.9	395.6	271.7
45R	16	+	0	0	907.2	210.9	
45L	16	++	1/8	0	1133.9	263.7	237.3
OH107R	18	Marked infection; suture completely disrupted.					
OH107L	18	Marked infection; suture completely disrupted.					
OH240R	19	++++	7/8	1/4	Tendon could not be separated from its bed; inadequately immobilized.		
OH240L		++++	1	3/8	Tendon could not be separated from its bed; inadequately immobilized.		
384L	20	+	0	0	1451.5	337.6	337.6
M840L	21	++	0	0	1415.2	329.1	329.1
34R	26	++	0	0	2041.2	474.7	
34L	26	++	1/8	0	1982.2	460.9	467.9
33R	27	+	0	0	2267.9	527.4	
33L	27	++	0	0	2177.2	506.3	516.9
M657R	30	++	1/2	0	5624.2	1308.0	
M657L	30	++	1/4	0	5556.5	1292.2	1300.1
M654R	32	+	1/4	0	6350.3	1476.8	
M654L	32	+	1/4	0	6168.8	1434.6	1455.7
32R	42	+	0	0	6622.4	1540.1	
32L	42	+	1/8	0	6304.9	1466.3	1503.2
31R	90	0	0	0	5669.9	1667.6	
31L	90	+	0	0	5896.7	1734.3	1700.9

TABLE VI.—TENDON SUTURING TECHNIQUES—END-THROUGH, END-TO-SIDE WITH 4-0 SILK

No.	Days	Reaction	Separation Markers	Separation Stumps	Gross pull Grams	Tensile strength G./sq.mm.	Tensile strength Average
M856R	4	+	1/16"	0	2472.1	574.9	
M856L	4	+	1/16	0	2948.3	685.7	630.3
M821R	6	0	1/16	0	2449.4	560.3	
M821L	6	+	1/16	0	2608.1	606.5	583.4
241R	11	+	0	0	2494.7	580.2	
241L	11	+	0	0	1360.8	316.5	448.4
40R	13	+	0	0	2131.9	495.8	
40L	13	+	0	0	2109.2	490.5	493.2
M802R	16	+	1/8	0	1814.4	477.5	477.5
38L	18	++	0	0	3628.7	843.9	843.9
M834R	24	++	3/16	0	3129.7	833.4	
M834L	24	++	1/4	0	3288.5	865.4	849.4
M827R	29	+	0	0	3447.3	907.2	
M827L	29	+	0	0	3129.7	833.4	870.3
857R	34	++	0	0	6304.9	1466.3	
857L	34	+	0	0	6713.1	1561.2	1513.8
M855R	42	+	0	0	6803.8	1582.3	
N885L	42	+	0	0	6758.5	1571.7	1577.0
39R	67	+	0	0	7076.0	1645.6	
39L	67	0	0	0	7438.9	1730.0	1687.8

tends to form a straight line or not. Presumably, it is less likely to or the separation would be greater.

On the other hand any accordion effect produced by the Mason and Allen suture was always less than ¼ inch. From Table VII, it can be seen that this method allowed the least amount of separation of the four end-to-end sutures. It would appear that the holding power of the tendon is greater for the Mason and Allen than for the modified Bunnell suture.

As might be expected, the two interlacing methods allowed no separation of the tendon ends. Perhaps this would account for their greater tensile strength both immediately and after the healing process is well advanced. It is our belief that not only does separation of the tendon ends increase reaction, but it also decreases the tensile strength. The suture that permitted the greatest separation (Yaeger wire-and-barb) also had the lowest tensile strength values. Indeed, the Yaeger suture removed at 90 days had a separation of ¾ inch and a tensile strength less than the one removed at 35 days.

TABLE VII.—SEPARATION OF TENDON STUMPS

	Minimum inches	Maximum inches	Average inches
Bunnell with silk	0	5/16	.075
Bunnell with wire	0	9/16	.075
Mason and Allen	0	1/8	.009
Yaeger wire-and-barb	1/8	3/4	.247
Pulvertaft	0	0	0
End-through, end-to-side	0	0	0

The tensile strength of the end-through end-to-side suture was significantly higher than the Pulvertaft during the first four weeks. This is probably due to the fact that the interlacing is more extensive in the former than in the latter, allowing less slipping as evidenced by the measurement of the markers.

We did not experience the initial rapid drop in tensile strength during the first five days recorded by Mason and Allen.[2] The tensile strength did not drop below that of the suture material used. We found that it began rising above that of the suture material by the eighth or ninth day; the rise was steepest after the 21st day when active movement was allowed.

The modified Bunnell suture with 34 gauge stainless steel wire held no advantages over the one done with silk. The silk was easier to handle and produced no more

reaction and the tensile strength values were not significantly altered.

The Mason and Allen method, a surface stitch, produced no more reaction than the modified Bunnell core sutures. It holds in the tendon better, and for this reason allows less separation of the tendon stumps. Furthermore, it has six knots—excluding the appositional suture—in contrast to the single knot of the Bunnell suture. If a single knot breaks, separation of the stumps is more likely to occur with the Bunnell method than with the Mason and Allen method—as has been noted in humans. For speed and simplicity, the Bunnell method is superior.

To make use of these findings in the human hand, it would seem that the Mason and Allen method is the one of choice for suturing round tendons, e.g. at the wrist, the proximal palm or the long flexor of the thumb. If speed is the essential, then the Bunnell No. 2 method should be used because of its simplicity. For tendon transplants or grafts such as the dorsum of the hand or palm, where the increased bulk does not matter, then the interlacing methods would be preferable. If movement is to start at three weeks, the end-through end-to-side method should be the choice.

Conclusions

These experiments have indicated that the Mason and Allen method of tendon suture is slightly superior to the modified Bunnell No. 2 method because less separation of the tendon stumps occurs. The degree of reaction and the return of tensile strength are comparable with both methods. With these two methods there is nothing to indicate superiority of either a surface or a core stitch.

The Yaeger suture had the greatest amount of separation and reaction; it also had the lowest recovery of tensile strength. For these reasons, its use would seem to be contraindicated.

The interlacing methods are superior to the end-to-end methods as regards the tensile strength developed. The degree of reaction is similar to that with the Bunnell and Mason and Allen methods. However, the cross-sectional diameter is usually larger and would contraindicate the use of interlacing methods in the digit. These methods would appear to be the ones of choice in the wrist, palm or dorsum of the hand—particularly with tendon grafts or transplants.

Stainless steel has no significant advantage over silk in tendon sutures, as regards reaction, tensile strength or separation of the stumps. It is more difficult to use.

The tissue reaction at the site of a tendon suture varies directly with the amount of separation of the stumps.

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Résumé

Les auteurs se proposent ici de comparer les mérites relatifs de six différentes techniques de sutures tendineuses, par l'expérimentation animale.

Ils utilisèrent des lapins de 5 à 10 livres. Sous anesthésie au pentobarbital avec asepsie complète, les essais de suture furent faits sur le tendon du long fléchisseur des doigts de la patte postérieure. Le tendon du muscle tibial postérieur, fut utilisé pour les méthodes d'interposition. Après l'intervention, le membre était immobilisé dans un plâtre, parfois renforcé de fil métallique, les animaux ayant une forte tendance à ronger leur plâtre. Les lapins furent sacrifiés à des intervalles variant entre 2 et 19 jours. L'intensité de la réaction (c'est-à-dire de la gaine) et la solidité de la suture furent notées.

De tous ces essais, il ressort que la méthode de Allen et Mason est légèrement supérieure à celle de Bunnell, car la coaptation des moignons tendineux y est mieux conservée. La méthode de Yaeger provoque une forte réaction et une séparation trop grande des extrémités: il semble que son usage soit contre-indiqué. Les méthodes d'interposition donnent des résultats supérieurs aux méthodes "bout à bout": elles semblent constituer les techniques de choix pour les sutures des tendons du poignet, de la paume et du dos de la main. L'emploi d'acier inoxydable en place de soie ne paraît pas avoir d'avantage particulier.

AN EVALUATION OF ASEPTIC AND ANTISEPTIC TECHNIQUES AS PRACTISED IN A MODERN HOSPITAL

II: Surgical Wards and Dressing Stations*

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

Introduction

Colbeck[2] recently stressed the importance of fomites in the spread of staphylococcal infections in hospitals. A comprehensive investigation by Nimeck *et al.*[4] of the aseptic and antiseptic techniques utilized in the operating theatres of a modern hospital has revealed many possible sources of surgical wound infection. After closure of the incision in the operating theatre the patient is transported to the postoperative surgical ward, and the purpose of the present investigation is to study aseptic and antiseptic techniques practised in these wards and in the adjacent dressing stations.

Experimental

Initially, two dressing stations and the surgical wards serviced by these dressing stations were chosen as study areas. The following possible sources of contamination were studied: (1) air; (2) depots (fomites); and (3) solutions which come in contact with the patient either directly or indirectly.

Although the initial study was carried out over a period of months when there was no evidence of an outbreak of infection, a unique opportunity for further study presented itself when a series of infections occurred in the pædiatric wards of the same hospital. Samples were obtained during this outbreak from the pædiatric wards and from an isolation unit composed of three wards and a small connecting hallway.

Since reference will be made in this paper to the results obtained from tests done following routine cleaning procedures it will be of interest to note the extent of these procedures. An all-purpose detergent is used for cleaning wards. Floors are mopped weekly with this agent and are then waxed with a water-base wax. Oil is used daily for dry-mopping. All wood furniture and fixtures are waxed with an oil-base wax. Furniture is damp-mopped daily with a solution of the all-purpose detergent. Washrooms are washed daily, with the use of 5% para-chlormetaxylenol if directed. Vacuum cleaners are used only on the furniture of solaria. Brooms are supposed to be used only on cement floors. Most of the bedding is changed daily but the extent of this change depends on supplies available at the time. Utensils used by all patients are washed in a detergent solution after use. Only private patients have individual utensils, including thermometers and bed pans.

In terminal cleaning (done on the discharge or death of a patient) all bedding is laundered except blankets which are merely aired. Drawsheets are usually washed with green soap containing 2.5% hexachlorophene (based on anhydrous soap). Mattresses are aired and sterilized with heat. Bedsprings are rubbed with turpentine. Bedside furniture is washed with a detergent solution or a disinfectant (such as quaternary ammonium compound 1/1000, para-chlormetaxylenol 5%, iodophor, green soap containing 2.5% hexachlorophene or a preparation of orthobenzyl-para-chlorophenol in a synthetic detergent).

Isolation units receive special terminal cleaning. Drapes and bedding are laundered. Mattresses and/or mattress covers are sterilized with heat. Furniture is washed with a disinfectant solution and placed on the beds. Walls and ceiling are then washed, using a detergent or one of the disinfecting solutions listed above. Floors are washed with a detergent or one of the disinfecting solutions and then waxed and polished. Furniture, bedding and drapes are then replaced and the ward is reopened for the admission of patients.

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

‡Director of Surgical Services, University of Alberta Hospital.

Materials and Methods

1. *Air sampling.*—Air samples were collected in the wards at hourly intervals from 7:00 a.m. until 1:00 p.m. Air samples in the dressing stations were also taken at hourly intervals, the first sample being collected between 5:00 and 6:00 a.m. and the remainder from 8:00 a.m. to 1:00 p.m. Air samples were taken with a General Electric Duplex Bacterial-Air Sampler using blood agar plates containing 5% sheep's red blood cells.[4] Tests for micro-organisms "settling-out" of the air were done by exposing open blood agar plates for definite intervals of time, incubating the cultures and subsequently counting the colonies. All plates were incubated for 48 hours at 37.5° C. before counting the colonies.

2. *Depots.*—Floors and other flat surfaces were sampled using sterile aluminum templates to limit and standardize the area sampled.[4] Before sampling, sterile swabs were moistened in serum broth to neutralize the hexachlorophene[3] used in the cleaning solution employed for such surfaces. If a quaternary ammonium compound had been used in the cleaning solution, the swab was moistened in Letheen broth prior to swabbing;[5] if an iodine solution had been used for cleaning, the swab was moistened in broth containing 0.5% sodium thiosulfate. If no disinfectant had been employed on the surface, swabs were simply moistened with sterile nutrient broth prior to sampling. Immediately after sampling the swab was placed in the appropriate neutralizing medium and subjected to mechanical shaking for 15 minutes. Following this, 0.1 ml. aliquots of the fluid were plated to blood agar medium.

Blankets and bedspreads were sampled by the sweep plate technique of Blowers and Wallace,[1] using blood agar plates which were rubbed over an area of one square foot.

Mops, brooms and brushes used in the test areas were sampled by culturing 1 inch strands of string or tufts of fibre. String or fibre was removed with the aid of sterile scissors and forceps. Samples were placed in serum broth, mechanically shaken for 15 minutes and 0.1 ml. aliquots of the suspension were then plated on blood agar.

3. *Solutions.*—Serial dilutions of samples aseptically obtained from various solutions used in the wards and dressing rooms were prepared in the laboratory. Dilutions were prepared in fluid medium containing the appropriate neutralizing agent for the particular disinfectant concerned or in sterile distilled water if no disinfectant was involved. Pour plates were made of the serial dilutions in nutrient agar. Duplicate surface streak plates were prepared on blood agar, using 0.1 ml. aliquots to aid in identification of the micro-organisms.

All cultures were incubated aerobically for 48 hours at 37.5° C. before colonies were counted. Micro-organisms were grouped according to colonial and microscopic morphology. Cultures of *Micrococcus pyogenes var. aureus** were checked for mannite fermentation, coagulase reaction, antibiotic sensitivity and bacteriophage type.

Results

1. *Air samples.*—Air samples showed that the greatest degree of contamination in surgical wards occurred during the period from 8:00 to 10:00 a.m., which corresponded to the time of the greatest activity in the wards, including bedmaking and cleaning. However, the amount of contamination was relatively high throughout the entire test period, averaging 17 organisms per cubic foot of air. The air in the dressing rooms was not as highly contaminated as that in the wards, averaging eight micro-organisms per cubic foot for the entire test period. Air samples taken in a student bacteriology laboratory of approximately 35,000 cubic foot volume showed 1.5 micro-organisms per cubic foot when no students were present and 9.7 micro-organisms per cubic foot of air when 60-70 students were present.

M. pyogenes var. aureus was present in 70 of the 98 samples of air obtained from dressing stations and postoperative surgical wards. Of the 90 strains of *M. pyogenes var. aureus*, obtained from the air samples, 78 were coagulase positive. A variety of bacteriophage types and patterns were isolated. Air from wards containing infected patients

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*Bergey's Manual of Determinative Bacteriology, The Williams and Wilkins Company, Baltimore; 6th edition, 1948.

TABLE I.—Bacterial Contamination and Incidence of *Micrococcus pyogenes var. aureus* on Depot Surfaces in Dressing Stations

Depot surface tested	*Number of samples taken*	*Average number of micro-organisms per square inch*	*No. of samples containing* M. pyogenes var. aureus
Cupboard	17	7.3×10^2	1
Doorknobs	5	3.2×10	0
Floors	7	2.4×10^3	1
Garbage receptacles	6	3.3×10^3	1
Laundry bags	2	2.9×10^2	1
Nailbrushes	3	2.6×10^3	2
Soap (bar)	1	1.9×10^3	0
Soap (dish)	1	1.3×10^3	1
Sink	7	6.3×10	0
Stethoscope	2	9.0×10^3	1
Stool	2	6.4×10^2	0
Supplies and containers:			
"Sterile" supply container.	11	9.3×10	2
"Non-sterile" supply container.	11	1.0×10^2	5
Dressing trays	13	5.2×10^2	0
Dressing wagons	9	5.9×10^2	0
Wash equipment	7	1.3×10^3	1
Windowsills	2	3.4×10^3	0

had an average of 23 *M. pyogenes var. aureus* per 200 cubic foot. Wards containing only non-infected patients had an average of 9 *M. pyogenes var. aureus* per 200 cubic foot while dressing stations had an average of 4.5 *M. pyogenes var. aureus* per 200 cubic foot of air.

2. *Depots.* — Cultures from depots in dressing stations and surgical wards showed a wide distribution of pathogenic micro-organisms. Results for dressing stations are given in Table I. Tables II and III show the degree of contamination of depots in surgical wards and the distribution of *M. pyogenes var. aureus.* The degree of bacterial contamination of various depots within a single surgical ward when infected patients were present, after cleaning by the routine methods previously described and after cleaning when non-infected patients were present, is shown in Table IV.

TABLE II.—Bacterial Contamination and Incidence of *Micrococcus pyogenes var. aureus* of Bedding in Surgical Wards

Type of bedding sampled	*Number of samples taken*	*Average number of micro-organisms per square inch*	*No. of samples containing* M. pyogenes var. aureus
Bedspreads*	27	7.8×10^2	3
Bedspreads†	29	2.5×10^2	17
Blankets*	4	3.8×10	1
Blankets†	38	2.2×10^2	19
Drawsheets (cotton)*	35	9.8×10^2	6
Drawsheets (rubber or plastic)	16	7.8×10^2	0
Sheets*	40	7.9×10^2	5
Pillows*	44	3.0×10^2	4
Mattresses*	49	1.1×10^3	5

*Sampled by swab technique.
†Sampled by "sweep-plate" technique.

3. *Solutions.* — Solutions found in the dressing stations include alcohol used for dressings, tincture of benzalkonium chloride 1/1000, aqueous benzalkonium chloride 1/1000 for instruments (sharps, forceps, etc.) and a return syringe bath containing aqueous benzalkonium chloride 1/1000 solution. All the solutions were tested and with the exception of the return syringe bath showed no contamination. The solution in the return syringe bath, which is supposedly bactericidal, had an average of 5.0 x 10^8 organisms per ml. Bacterial counts of ice used in the dressing stations were surprisingly low, considering the fact that little care is taken to avoid contamination.

Solutions found in the wards include drinking water, back rub lotion, thermometer solution (aqueous benzalkonium chloride 1/1000), patients' wash water, mouthwash and scrub water used for furniture and floors. All the solutions were tested. Drinking water, back rub lotion and the thermometer solution showed no contamina-

TABLE III.—Bacterial Contamination of Depots in Surgical Wards and Incidence of *Micrococcus pyogenes var. aureus*

Location of sample	*Number of samples taken*	*Average number of micro-organisms per square inch*	*No. of samples containing* M. pyogenes var. aureus
Bathtubs	1	5.7×10^2	0
Bedframes	108	1.4×10^3	21
Bedsprings	25	1.8×10^3	3
Bedcastors	10	1.4×10^3	4
Bedtrays	3	3.1×10^3	0
Bedpans	1	9.0×10^3	0
Blinds	27	4.1×10^2	1
Blindpulls	12	7.3×10	1
Brooms	1	3.0×10^3	1
Cuffs of manometers	4	1.1×10^3	2
Chairs	52	4.3×10^2	6
Coat hooks	2	3.0×10	0
Dishes	3	1.3×10	0
Doors	8	4.8×10^2	2
Doorknobs	41	6.4×10^2	6
Drapes	21	2.5×10^2	3
Floors	79	3.2×10^2	17
Footstools	16	7.6×10^3	6
Garbage receptacles	21	1.8×10^3	5
Lamps	25	1.1×10^2	0
Lightcords and switches	21	1.9×10^2	1
Laundry bins	6	3.7×10^2	0
Mask bins	3	2.0×10	0
Mops (for washing)	2	8.3×10^4	0
Mops (for waxing)	1	1.8×10^3	0
Oxygen equipment	15	1.6×10^3	0
Radiators	31	1.9×10^3	5
Shoes (patients' and nurses')	13	6.1×10^3	11
Sinks	25	1.0×10^3	3
Taps	12	3.9×10^2	0
Tables and cabinets	97	5.3×10^2	10
Cabinet castors	4	1.5×10^3	0
Thermometer bins	1	6.0×10	1
Toilets	11	2.0×10^3	5
Walls (ledges and mouldings)	95	8.6×10^2	17
Wash supplies	26	6.1×10^3	9
Waterjugs	19	1.0×10^3	2
Wastebaskets	19	7.8×10^2	3
Windowsills	33	7.4×10^2	5
Wheelchairs (arms)	4	2.2×10^2	0
Wheelchairs (treads)	4	6.9×10^3	4

TABLE IV.—Comparison of Degree of Bacterial Contamination of Depots in a Ward Containing Infected Patients, Same Ward after Routine Cleaning and Cleaned Ward Containing Non-Infected Patients

	Ward containing infected patients			*Same ward after routine cleaning*			*Cleaned ward containing non-infected patients*		
Location	*No. of samples*	*Average No. micro-orgs. per sq. in.*	*No. containing* M. aureus*	*No. of samples*	*Average No. micro-orgs. per sq. in.*	*No. containing* M. aureus	*No. of samples*	*Average No. micro-orgs. per sq. in.*	*No. containing* M. aureus
Bedding:									
Bedframes	8	4.6×10^2	0	8	2.0×10^3	4	6	9.8×10^2	2
Drawsheets (cotton)	3	3.9×10^2	1	2	6.0×10	0	2	0	0
Drawsheets (rubber)	1	3.0×10	0	1	9.0×10	0	2	9.0×10	0
Mattresses	4	1.8×10^3	2	5	2.8×10^2	1	4	3.9×10^2	2
Pillows	4	6.8×10^2	0	4	2.4×10	0	4	6.0×10	0
Sheets	4	1.3×10^3	1	5	3.0×10	0	3	1.0×10	0
Miscellaneous:									
Blinds	2	4.5×10^3	1	4	8.0×1	1			
Chairs	2	2.6×10^3	1	8	6.0×10	0	3	3.0×10	1
Drapes	3	7.3×10	0	3	1.0×10	0	1	1.8×10^2	0
Doorknobs	3	4.2×10^3	0	3	1.1×10^2	0			
Floors	5	7.9×10^3	1	11	2.0×10^2	2	6	1.7×10^3	1
Lamps	1	3.0×10	0	2	3.0×10	0	2	0	0
Light switches	2	9.2×10^2	0	2	7.5×10	0	1	0	0
Oxygen equipment	1	3.1×10^3	0	2	6.0×10	0			
Radiators	2	7.2×10^3	1	2	1.7×10^3	2	2	7.5×10	0
Sinks	2	5.4×10^3	1	2	1.4×10^3	0	2	0	0
Taps	1	4.0×10^2	0	1	1.2×10^2	0	1	2.4×10^2	0
Tables and cabinets	9	1.7×10^3	2	9	4.0×10	0	7	1.3×10	1
Toilets	1	1.6×10^3	0	1	4.0×10	0	1	3.0×10	1
Walls	4	7.0×10^3	2	7	9.2×10^2	3	5	2.6×10^2	0
Wastebaskets	1	9.0×10^3	1	2	6.9×10^2	0	2	4.5×10	1
Windowsills	2	4.9×10^3	1	2	3.4×10^2	0	2	6.0×10	0
Wash supplies	3	6.5×10^3	2	1	3.0×10	0			
Waterjugs	2	1.5×10	0	3	2.8×10^2	1			

* *M. pyogenes var. aureus*

tion. Patients' wash water was not contaminated before use but contained on the average 8.0×10^5 organisms per ml. after use. Many of the samples of used wash water contained organisms of the same phage type and antibiotic sensitivity as the organisms from infected lesions of the patient using the water. Mouthwash was only slightly contaminated before use, but, as expected heavily contaminated after use. Scrub water used for furniture and floors was contaminated.

Discussion

The air and a great variety of depot surfaces in the dressing stations were contaminated with *M. pyogenes var. aureus.* A need for more rigid cleaning techniques is indicated. The return syringe bath was heavily contaminated. It is possible that large numbers of micro-organisms together with organic matter were added to the solution over a prolonged period of time, since there seemed to be no control over the length of time each solution was used before changing. Obviously the return syringe bath should be replaced frequently and at regular intervals with fresh solution.

Wards containing infected patients provided some interesting information. For example, one ward contained a patient infected with *M. pyogenes var. aureus* phage pattern Q 6/47/53/54/81 and a strain of *Pseudomonas pyocyanea.* Eight of nine strains of *M. pyogenes var. aureus* isolated from the air in this ward were of the same phage pattern as the strain infecting the patient. Organisms of the same phage pattern were recovered from the blankets, bedsprings, mattresses, kidney basins, floors and garbage receptacle in the same ward. *Ps. pyocyanea* was recovered from the air, kidney basins, doorknobs, footstool and walls in this ward. Another example is that five different phage patterns of *M. pyogenes var. aureus* and one strain of *Streptococcus*

pyogenes were isolated from the lesions of four patients contained in a single ward. Six strains of *M. pyogenes var. aureus* recovered from air samples in this ward were of the same phage patterns as those infecting the patients. One or more of the same phage types of *M. pyogenes var. aureus* were isolated from mattresses, sheets, kidney basins, blinds, chairs, tables, soap dishes, shelves in a water closet, wastebaskets, blankets and bedspreads in this ward. In an isolation unit, four of six patients were infected with *M. pyogenes var. aureus* Q 81. *M. pyogenes var. aureus* Q 81 was recovered from the air, brooms, doorknobs, toilet, blankets, bedspreads, windowsills, floors, beds, chairs, light switches and sheets in two of the three wards composing this isolation unit. The same organism was isolated from the air in the hallway connecting the wards and the outer surface of the door connecting the isolation unit to the main corridor of the hospital.

In wards where special attention was given to cleaning, the degree of contamination was usually reduced significantly. For example, in one ward before cleaning, cultures taken from pillows, tables, chairs, sheets, windowsills, walls, mattresses and radiators respectively gave bacterial counts of 424, 530, 730, 1104, 1376, 1951, 2141 and 3309 micro-organisms per square inch; after cleaning, the respective bacterial counts were 60, 15, 30, 10, 0, 355, 353 and 100. However, despite increased attention to cleaning in this ward, *M. pyogenes var. aureus* was still recovered from several depots including a bed, chair, blanket, mattress and footstool.

Solutions in the wards were relatively clean. However, the presence of pathogenic micro-organisms in wash water used by infected patients indicates a need for increased care in decontaminating the wash basins, since these basins may be subsequently used by previously uninfected patients. At present these basins are washed in detergent solution in the utility room. Cultures of scrub water used for furniture and floors showed contamination, indicating a need for more frequent changing of these solutions and better methods for decontamination of mops and scrub buckets.

Summary

Possible sources of contamination in surgical wards and dressing stations which have been bacteriologically investigated are: air, a great variety of depot surfaces and the various solutions used in these hospital areas.

Investigations were carried out in the absence of any indication of an outbreak of infection and also during a time when a series of infections existed.

A high degree of contamination was found in the air of the wards, especially during periods of greatest activity, e.g. bedmaking. Air in the dressing stations was not as highly contaminated. *M. pyogenes var. aureus* was present in 70% of the air samples taken from surgical wards and dressing stations.

M. pyogenes var. aureus was isolated from a great variety of depot surfaces in dressing stations, wards containing non-infected patients, wards containing infected patients and wards which had just been cleaned by the routine cleaning procedure. Wards containing infected cases were more highly contaminated and the incidence of *M. pyogenes var. aureus* was greater than in the other areas mentioned.

Solutions found in the wards and in dressing stations were relatively clean, with the exception of the bath for return syringes, which was heavily contaminated.

Micro-organisms of the same phage type and antibiotic sensitivity as organisms isolated from patients' lesions were found to be widely distributed in the air and on depot surfaces in wards containing infected patients.

Since the completion of this project, steps have been taken by the hospital administration to remove the sources of contamination disclosed by this investigation.

Acknowledgments

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RÉSUMÉ

Les auteurs se sont appliqués à découvrir les sources de contage dans les salles de chirurgie et les chambres de pansements en analysant l'air, plusieurs surfaces où peuvent se poser des microbes et les différentes eaux et solutions employées dans ces départements d'hôpital. Ces recherches ont été poursuivies tant dans les périodes bactériologiquement calmes que dans celles ou l'on rapportait des infections multiples. L'air des salles montra un degré de pollution remarquable pendant les heures de grande activité et surtout au moment ou le personnel fait les lits. L'air des chambres de pansements n'atteignit pas le même degré de contamination. On trouva du staphylocoque doré dans 70% des prélèvements d'air de ces deux endroits. On en retrouva aussi sur un grand nombre de surfaces des chambres de pansements et des salles contenant des malades infectés ou non et aussi dans des salles qui venaient d'être nettoyées par les méthodes en usage courant. Le contage le plus élevé se trouva comme on peut s'y attendre dans les salles de malades infectés et la concentration de staphylocoque doré fut plus grande dans ces endroits que partout ailleurs. Les différentes eaux et solutions employées dans les salles furent trouvées relativement propres à l'exception des bassins de rinçage pour seringues ayant servi. L'antibiogramme et le typage au bactériophage montrèrent que les micro-organismes prélevés ici et là dans les salles correspondaient à ceux que l'on retrouva dans les lésions des malades sous traitement. Depuis la compilation de ces travaux l'hôpital où ils furent menés a pris des mesures pour enrayer ces sources d'infection.

CONTROL OF INFECTIONS WITHIN HOSPITALS

Because it was realized in a New England hospital that nearly every patient who had entered hospital "clean" and contracted an infection while there was the victim of an error in technique, a searching investigation of the causes of hospital infection was launched and a control program mapped out. This program, recently reported by Adams and his colleagues (*J. A. M. A.*, 169: 1557, 1959), has proved medically effective, practical and economically feasible.

It has been shown that frequently repeated and intelligently applied methods of mechanical cleansing in combination with a good disinfectant-detergent will clean any area, and that an efficient air-conditioning system can readily be made to deliver dust-free, nearly sterile air under positive pressure. This air will remain sterile indefinitely in a clean room unless infection is introduced by exhalations of personnel entering the room or by fomites from clothing, shoes or objects brought in. Infection due to human exhalations is at least 98% preventable by efficient filter masking of all people entering the room.

Whenever a person re-enters a clean area he must put on a freshly sterilized gown (double-breasted, wrap-around, lace-tying, covering the subject from neck to toes) and sterilized temporary shoe coverings.

The authors believe that nine-tenths of the wound infections in hospital arise in the operating pavilion area, and the latter is separated from the hospital by an interchange area inside which all aseptic criteria are fulfilled (shoes wrapped in sterile booties, filter mask worn) and which contains the scrub-up area. Outside the interchange area, there is a zone of complete cleanliness as regards room, floor and furniture.

The authors remark that "a room which looks clean, smells clean and feels clean is most likely to be clean and to be proven clean when subjected to critical testing for bacterial and particulate matter content. For nine-tenths of the hospital, a good housekeeper with an eye for dirt and an ability to get it removed is a better investment than complicated bacteriological testing equipment and routines. For the other one-tenth of the hospital area, meaning essentially the operating pavilion and the nursery, standards, controls, and fulfillment must be of the ultimate type from which no appeal for leniency is tolerable."

RECENT EXPERIENCE WITH ABSCESS OF THE PROSTATE

PHILIP G. KLOTZ, M.D., F.R.C.S.[C], F.A.C.S.,* *Toronto*

ABSCESS OF THE PROSTATE has become uncommon since antibiotics were made available. Persky[1] in 1955, could report only 12 cases seen at Western Reserve University in the previous 11 years. Chitty[2] in 1957 reported 27 cases from Bristol, the total for a six year period.

Gonorrhœal urethritis, which was the commonest cause of abscess because of its tendency to invade the prostate directly, has become rare. It is of interest that in 1931, 75% of abscesses of the prostate reported were due to gonorrhœa.[3] In Chitty's series of 27 cases, none were due to gonorrhœa. Nowadays, bacteria usually reach the prostate through the blood stream, and abscess has been reported following staphylococcal infection of the skin, pneumonia, appendicitis, and tonsillitis. Direct infection of the prostate may follow urethral instrumentation or non-specific urethritis.

The patient is likely to complain of vague general symptoms, such as malaise, fever, and poorly localized discomfort in the abdomen and the back. Local manifestations are dysuria, pain in the perineum or rectum, and in most cases retention of urine. Rectal examination reveals increased local heat, and a tender, œdematous, or fluctuant prostate, which may be mistaken easily for a prostate affected by benign hypertrophy.

Conservative therapy has been advocated, in which case the abscess is allowed to resolve spontaneously, usually into the urethra. The results of this treatment have been poor because persistent chronic prostatitis and metastatic blood stream infection have often resulted. Modern therapy demands surgical drainage as soon as the diagnosis of abscess of the prostate is determined.

Drainage has been provided by all the avenues available to the prostate. These are the transvesical, retropubic, perineal, and transurethral approaches. Each method has its advocates, and there is general agreement that if the diagnosis can be made before operation, transurethral resection with intra-urethral discharge of the abscess is most satisfactory. The resection must be complete so that all pockets of pus are drained.

Three cases of prostatic abscess have been treated recently. This is a sufficiently unusual occurrence to justify reporting.

CASE REPORTS

CASE 1.—W.W., age 50, was well until three weeks before admission to hospital, when he developed low grade fever, productive cough, low back pain, and mild dysuria. He was treated for "flu" by his family physician who administered broad-spectrum antibiotics. Three days before admission to hospital, dysuria became severe, and he developed complete urinary retention.

When admitted to hospital on November 7, 1957, his oral temperature was 100° F. The prostate was enlarged and soft, but not fluctuant or particularly tender. An intravenous pyelogram showed the upper urinary tract to be normal. Cystoscopic examination demonstrated enlargement of both lateral lobes of the prostate gland, more so than would be expected in a man of this age. The urine produced *Staphylococcus aureus hæmolyticus* on culture.

His condition improved on sulfonamide therapy, and after four days retropubic exploration of the prostate was carried out. When the capsule of the prostate was incised, thick pus was encountered and the true diagnosis established. The pus later proved to be sterile. The prostatic capsule was loosely sutured, and the bladder drained transurethrally for two weeks. He received chloramphenicol (Chloromycetin) 250 mg. 6 hourly, orally for five days, followed by sulfonamides for a month. The wound healed without infection, and no serious problems arose when the catheter was removed. He has remained well since.

CASE 2.—J.C., age 53, developed urinary retention after bilateral inguinal herniorrhaphy and required intermittent catheterization. He was having some difficulty with micturition when discharged from hospital seven days after operation. Three days later, acute retention developed and he was readmitted to hospital on April 21, 1958.

His temperature was normal. Physical examination demonstrated recent bilateral inguinal incisions, the distended bladder, and a slightly

*Attending Urologist, New Mount Sinai Hospital, Toronto, and Scarborough General Hospital, Scarborough, Ont.

enlarged tense, fluctuant, tender prostate gland. Further investigation revealed *Staphylococcus aureus hæmolyticus* growing in the urine. The upper urinary tract was normal, as shown by intravenous pyelography. Cystoscopy confirmed the diagnosis of prostatic abscess, when bulging lateral prostatic lobes were seen.

The patient was maintained on catheter drainage and antibiotics for four days, when transurethral prostatectomy was carried out. As each lateral lobe was incised, a gush of thick pus was obtained. The prostatic urethra was thoroughly resected, and the bladder drained by catheter for four days. Antibiotics and sulfonamides were administered for one month. The postoperative course was smooth, and no difficulties developed after removal of the catheter. He has remained quite well since.

CASE 3.—P.S., age 74, was initially admitted to hospital on April 28, 1958, with purulent infection in a large hydrocele sac. This failed to respond to conservative therapy, and surgical drainage and orchidectomy was performed on May 9, 1958. The pus was sterile. At this time he was found to have a benign enlargement of the prostate gland, but because of the lack of urinary symptoms, this was not investigated further. Eight days after operation, he was sent home with the incision healed.

Two weeks later he developed acute urinary retention. He had noted dysuria for one week previously. He was afebrile on admission to hospital. The striking sign was inability to palpate the enlarged prostate on rectal examination, when it had previously been found to be grossly hypertrophied. The anterior rectal wall felt quite flat and fluctuant, and the diagnosis of posterior prostatic abscess was made. Urine culture proved to be sterile. Cystoscopic examination revealed the prostate to be tremendously hypertrophied.

At operation, the prostate was approached transvesically. When the finger was introduced behind the middle lobe of prostate, a large abscess cavity was encountered and thick purulent material welled up. The cavity extended two-thirds of the way around the adenomyomatous portion of the prostate and had almost auto-enucleated the gland. Accordingly, the enucleation was completed. The specimen weighed 134 grams.

On culture, the pus grew *Pseudomonas pyocyanea*, sensitive to streptomycin and tetracycline. The bladder was drained by suprapubic tube and transurethral catheter as well. During the next ten days the tubes were removed. He received antibiotics for one week followed by sulfonamides for a further three weeks. The patient's postoperative period was uneventful, apart from some delay in wound healing for three weeks.

TABLE I.—THREE CASES OF PROSTATIC ABSCESS

	J.C.	*W.W.*	*P.S.*
Age	53	50	74
Previous surgery	Hernia repair 2 weeks previously	None	Orchidectomy 3 weeks previously (sterile pus)
Retention	Yes	Yes	Yes
Urine culture	*Staph. aureus hæm.*	*Staph. aureus hæm.*	Sterile
Culture of pus	Not done	*Staph. aureus hæm.*	*Ps. pyocyanea*
Approach	Transurethral	Retropubic	Transvesical

SUMMARY

Although abscess of the prostate has become a rare disease, its recognition and treatment are important. It is suggested that the results of conservative treatment are unsatisfactory, and that treatment should always be surgical. Three cases in which the abscesses were drained, though by different routes, have been reported. All made a complete recovery.

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158 St. George Street, Toronto, Ontario.

RÉSUMÉ

Depuis l'apparition des antibiotiques, les abcès prostatiques sont devenus très rares; de plus, si jadis le gonocoque était le germe causal le plus fréquent, il est maintenant exceptionnel, et la grande majorité des prostatites suppurées relèvent d'une infection hématogène.

L'auteur rapporte trois cas personnels: le premier malade, âgé de 50 ans, souffrait depuis trois semaines de douleurs vagues dans les reins, de dysurie légère et d'un état fiévreux discret. Il fut hospitalisé pour une rétention urinaire soudaine. A ce moment la température s'éleva à 100° F. A l'examen rectal, la prostate était légèrement hypertrophiée et de consistance normale; ces constatations furent confirmées par la cystoscopie. Le pyélogramme intraveineux était normal, mais par contre, les cultures d'urine firent apparaître du staphylocoque doré hémolytique. Après une préparation de quelques jours par une thérapeutique aux sulfamides, on procéda à une exploration rétropubienne de la prostate: c'est alors que l'on trouva un abcès

rempli de pus épais, qui fut drainé; le patient reçut des antibiotiques et guérit simplement.

Le deuxième malade, âgé de 53 ans, souffrait de rétention urinaire intermittente depuis un certain temps; il fut finalement hospitalisé pour une crise plus grave. Un abcès de la prostate fut découvert à la cystoscopie, et l'on pratiqua l'incision des deux lobes transurétralement. Sous le couvert d'antibiotiques et de sulfamides, l'évolution postopératoire fut satisfaisante.

Le troisième cas était celui d'un homme de 74 ans hospitalisé pour une infection purulente d'un sac d'hydrocèle. Cet état ne répondait pas à la thérapeutique conservatrice; on fit un drainage et une orchidectomie. Le malade semblait guéri et fut renvoyé à domicile huit jours plus tard. Deux semaines après sa sortie il fit une crise de rétention urinaire aiguë: là encore, le diagnostic d'abcès prostatique se fit par la cystoscopie. Le drainage fut fait par voie transvésicale et les suites opératoires furent sans histoires.

En conclusion, l'abcès de la prostate est une maladie qui devient rare, mais comme elle peut encore se présenter, il convient d'y penser.

ACUTE AND CHRONIC BREAST ABSCESS*

T. S. WILSON, M.D., F.R.C.S.[C], *Edmonton, Alta.*

SINCE THE ADVENT of antibiotics, the natural history of suppuration in various parts of the body has changed.[1] It seemed worth while to review the cases of breast abscess seen in the University of Alberta Hospital in the five-year period 1953 to 1957, during which time all of the common antibiotics were in widespread use. Penicillin, streptomycin, the tetracyclines, chloramphenicol, and erythromycin were used throughout the five years, and novobiocin during the last two years.

There were 30 patients with acute breast abscess and 14 with chronic breast abscess. Thus chronic breast abscess was almost half as common as acute breast abscess, which is a rather startling finding in view of the fact that chronic breast abscess was almost a medical curiosity 20 years ago.

Our definition of chronic breast abscess was one that had been present longer than six months, or, in cases in which the patient did not know the length of time present, one with a pathological diagnosis of "chronic breast abscess". The wall of these chronic abscesses is thick and fibrous and on microscopic examination is seen to contain chronic inflammatory cells in the wall. In most cases, chronic breast abscess had no relationship to pregnancy and lactation. It was invariably sterile except for the one tuberculous breast abscess,[2] and in most cases the preoperative diagnosis was carcinoma.

It is realized that some of the small superficial acute breast abscesses are never treated in hospital and are incised in the doctor's office or emergency department. Many may be aborted by antibiotics in their early stages before actual pus forms, although once suppuration develops these drugs rarely cause resolution. These factors may weight the scales so that chronic breast abscess appears relatively more common than it actually is.

ACUTE BREAST ABSCESS

By far the commonest variety was the puerperal breast abscess seen in the lactating breast (Table I).

TABLE I.—ACUTE BREAST ABSCESS, U.A.H., 1953-57

Puerperal (lactating breast)	23
Older women (no relation to pregnancy)	4
Patient five months pregnant	1
Female baby, age 14 months	1
Male baby, age four weeks	1
Total	30

The average age of these patients, omitting the two babies was 29 years. More than half the abscesses occurring in the lactating breast developed after the first pregnancy (Table II).

*From the Departments of Surgery and Bacteriology, University of Alberta, and from the University of Alberta Hospital.
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TABLE II.—Acute Breast Abscess Developing in Lactating Breast, U.A.H., 1953-57

After first baby	14
After second baby	7
After third baby	1
After fifth baby	1
Total	23

Thus it would seem that the young mother with her first child is more prone to develop breast abscess. Some of the mothers had subsequent babies in the hospital, and none developed a second acute breast abscess.

Of particular interest is the fact that 19 of the 23 puerperal breast abscesses occurred in 1954, most frequently in the fall, when there was a sudden epidemic of staphylococcal infections on the obstetrical service.

The infection gains access to the breast by the nipple, but quite frequently no crack nor fissure was demonstrated. Retention of milk, due to inspissated material in larger ducts, may predispose the breast to infection, and it is possible that staphylococci from the baby, or environment, or the patient herself, may invade a relatively healthy nipple and its ducts.

Cultures were reported in 27 out of 30 patients with acute breast abscess. There was no report on two patients, and the abscess was sterile (no growth) in one. The infecting organism in every case was *Staphylococcus aureus. In vitro* sensitivity tests were carried out on all cultures by the disc method. For the first three years, sensitivity tests were carried out with penicillin, erythromycin, chloramphenicol, the tetracyclines, streptomycin, and as well, in the last two years with novobiocin (Table III).

TABLE III.—Sensitivity Tests on Cultures from Acute Breast Abscess, U.A.H., 1953-57

Sensitive to all six antibiotics	4
Sensitive to erythromycin, chloramphenicol, streptomycin	16
Sensitive to erythromycin, chloramphenicol	3
Sensitive to all but penicillin	4
Total	27

Thus the table shows that in 23 out of 27 patients with acute breast abscess the infecting organism, in every case *Staphylococcus aureus*, was resistant to penicillin; in other words, in only *15%* of patients with breast abscess is the *organism sensitive to penicillin.* It is realized that these are *in vitro* sensitivities to ordinary concentrations of the antibiotic, but we have found them very useful in planning treatment.

In reviewing the 23 acute breast abscesses occurring in the lactating breast, it was found that nine of the 23 had required a secondary drainage procedure, i.e., 40% required two or more operations. This point will be stressed again in considering treatment.

Chronic Breast Abscess

Chronic breast abscess developed in 14 patients. The average age of these patients was 42 years, hence chronic abscesses tend to occur in middle-aged women. In only four cases was there any relation to pregnancy or lactation.

There were two varieties of chronic breast abscess, and in our cases they were about equally common. The first type presented as a mass in the periphery of the breast, diagnosed most frequently preoperatively as carcinoma. The mass was generally not tender, or very slightly tender, indurated, blending with breast, and the patient had no systemic reaction and no leukocytosis. The surgeon was surprised to encounter pus in the biopsy specimen.

The second type presented as a mass beneath the nipple or in the subareolar region, with a history of some discharge from the nipple, and invariably with some retraction of the nipple. Again there was very little tenderness, no systemic reaction, and no leukocytosis.

The etiology of both types of chronic abscess is obscure. In four cases, there was some relation to lactation, and these cases had been treated with antibiotics. Antibiotics may have "masked" an acute abscess, locking it in the breast, the organism being attenuated but not killed. Trauma to the breast, with hæmatoma formation and later abscess in the hæmatoma, may account for a few cases. There was no history of trauma in any of our patients.

Whether these chronic abscesses are "infective" or not is uncertain. Cultures

were reported in about half of the cases and no growth was obtained. Those seen in the periphery of the breast may represent a residuum of an acute breast abscess which has not quite resolved. The type seen beneath the nipple or areola probably represents a chemical inflammation, resulting from changes in the major ducts of the breast, or duct ectasia. Here there is dilatation of the ducts, stasis and some blockage of secretions, with inspissation of toothpaste-like material in the ducts, and perhaps finally a chemical inflammation set up as a reaction to this material, if it escapes outside a duct. Adair[3] called this lesion "plasma cell mastitis", and Bloodgood[4] "varicocele tumour of the breast".

Pathologically, the peripheral chronic breast abscess is usually small and thick-walled, and microscopic examination reveals chronic inflammatory cells in the wall. Those beneath the nipple are again small, the actual abscess cavity measuring only 1 to 3 cm. in diameter, and there are dilated ducts with inspissated debris. Large numbers of chronic inflammatory cells, lymphocytes and plasma cells are seen.

Tuberculosis of the breast is a rare disease—only one of the 14 cases was definitely proven to be tuberculous. This patient had a long-standing tuberculosis of the hip joint, and in this case guinea pig inoculation was positive. Recently, and not included in this series, we have had another tuberculous breast abscess, in an Indian woman with minimal pulmonary disease. This abscess was proven tuberculous by finding acid-fast bacilli on direct smear and tubercles in the wall of the abscess on microscopic examination, and by positive guinea pig tests.

Treatment

Acute Breast Abscess

Since most of our acute abscesses in the past five years occurred when staphylococcal infections were endemic on the obstetrical wards, we feel that the most important prophylactic measure is the control of staphylococcal infections in general in the hospital.

Many cases of acute mastitis in the lactating breast never go on to suppuration. If a portion of a breast is indurated and tender, manual or pump expression may help, and if the infection is more severe, feeding from the affected breast should be discontinued. The value of antibiotics at this stage is debatable. Some breast abscesses are undoubtedly aborted by antibiotics, but there is the danger of development of resistant organisms, of the reactions to the antibiotics themselves, and of the occasional development of chronic breast abscess. The figures above show that penicillin, the most widely used antibiotic, has only a 15% chance of doing any good anyway. On the whole, we are against giving prophylactic antibiotics in these cases, as well as in most other situations. Very occasionally acute streptococcal infections of the breast may occur, and take the form of a massively spreading cellulitis or abscess, and in these cases penicillin is, of course, invaluable. We have not seen this type of infection in the past five years.

Once suppuration has occurred and an abscess has formed, the only reliable treatment is by surgical incision and drainage. Antibiotics alone will not cure these patients. Since nine of our 23 patients with acute puerperal breast abscesses (or 40%), required two or more drainages, we believe that operation for any abscess in the substance of the breast should be fairly radical and should consist of through and through drainage. For very superficial breast abscesses, a radial incision directly over the most tender and indurated portion may suffice, but deeper abscesses require, besides radial incision and breaking down of loculations, a counter incision in the most dependent portion of the breast. The incisions may be packed loosely with petrolatum gauze. Antibiotics should be given on the basis of clinical judgment until culture and sensitivity reports are obtained. In our cases, novobiocin, chloramphenicol and erythromycin were most likely to be of value.

Chronic Breast Abscess

The treatment in these cases is surgical excision rather than incision. There were three recurrences among the 14 cases of chronic breast abscess treated surgically, and in each case a surgical incision alone had been done. Since these chronic ab-

scesses are generally sterile on culture, there would seem to be little danger in excision. There is often considerable fibrosis and thickening of breast tissue surrounding the abscess, which can be ablated by surgical excision similar to the wedge excision of the breast carried out for bleeding from the nipple when a duct papilloma cannot be localized. The breast may be reconstituted with fine catgut sutures, perhaps leaving in a small Penrose drain for one or two days. In most of the cases of chronic abscess in the periphery of the breast, this method of surgical excision was carried out, complications were non-existent, and the cosmetic result was excellent.

The treatment of chronically recurring subareolar abscess is much the same. An attempt at local surgical incision or excision with sparing of the nipple may be made, but if there is much disease in the major ducts and retraction of the nipple, the only sure treatment is elliptical excision of the nipple, areolar and subareolar tissues. If the elliptical excision is transverse, there is not much deformity, and most of the breast tissue is left. These patients have no further complications and are happy to be rid of a chronic inflammatory process that has often been going on for months or years. We have not encountered a patient who had become pregnant again after elliptical excision of the nipple, but doubt if it would cause any trouble.

In some cases, antibiotics were given after surgical excision of a chronic breast abscess; in other cases no antibiotics were used. We doubt whether they are necessary, for these abscesses are usually sterile.

Summary

Forty-four cases of breast abscess seen in a large hospital over the past five years have been reviewed. Chronic abscess was half as common as acute breast abscess.

Acute breast abscess was seen most commonly in the lactating breast of a mother with her first child. Invariably the infecting organism was *Staphylococcus aureus*, insensitive to penicillin in 85% of cases.

Chronic breast abscess was most often diagnosed as carcinoma preoperatively. There was little or no sign of inflammation and the surgeon was surprised to encounter pus. These chronic abscesses may be more common because of the widespread use of antibiotics. They presented as peripheral or subareolar lesions.

The treatment of acute breast abscess is surgical, with radial incision and, if the abscess is large or deep in the breast, with an additional counter-incision in the most dependent portion of the breast. Novobiocin or erythromycin or chloramphenicol should be given at the same time.

The treatment of chronic breast abscess is by surgical excision; since these abscesses are generally sterile, the breast is reconstituted with or without drainage. If the abscess is subareolar in position and if there is much retraction of the nipple, excision of the nipple, areola and subareolar tissue should be carried out. Antibiotics are probably of little value.

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Résumé

Cet article porte sur les cas d'abcès du sein vus à l'hôpital de l'université d'Alberta dans la période de cinq ans se terminant en 1957. La série comprend 30 malades avec des abcès aigus et 14 avec des abcès chroniques. La plupart des abcès chroniques n'offraient aucun rapport à la grossesse ou à la lactation. Ils étaient tous stériles (sauf pour un cas d'abcès tuberculeux) et la plupart du temps un diagnostic pré-opératoire de carcinome avait été porté.

Les abcès aigus se voient le plus fréquemment pendant la période de lactation et la majorité se produit après la première grossesse; l'infection pénètre dans le sein par le mamelon mais il est souvent très difficile sinon impossible de trouver une crevasse ou une fissure comme porte d'entrée. L'agent étiologique le plus fréquemment identifié fut le staphylocoque doré. L'âge moyen des 14 malades présentant un abcès chronique du sein était de 42 ans. Cette lésion peut se présenter comme une masse presque indolore, indurée, située à la périphérie du sein ou sous le mamelon ou la région de l'aréole. Elle n'a aucune répercussion sur l'état général. L'étiologie de ces lésions est obscure.

Le traitement de ces affections commence par des mesures prophylactiques comme l'éradication

du staphylocoque doré dans les hôpitaux et surtout dans les pouponnières. L'auteur recommande également l'emploi d'une pompe ou l'expression manuelle des sécrétions dans la mastite aiguë de la lactation. Le choix des antibiotiques doit être dirigé par les résultats de l'antibiogramme qui dans cette série a montré que seulement 15% de ces infections répondaient à la pénicilline. Lorsqu'il y a suppuration déclarée le traitement chirurgical par incision et drainage s'impose. Dans les cas d'abcès chronique, l'auteur préconise l'excision entière de la lésion au lieu de la simple incision.

REPLACEMENT OF A SEGMENT OF THE COMMON BILE DUCT BY AN IVALON PROSTHESIS IN DOGS*

C. C. KRAUSE, B.A., M.D., C.M. and
A. B. McCARTEN, M.D., F.R.C.S.(Eng.), F.R.C.S.[C], *Edmonton, Alta.*

INJURY TO THE extrahepatic biliary tree, with its sequelæ, continues to be an important surgical entity.[1] By far the commonest cause is trauma to the common bile duct during the performance of cholecystectomy. The usual sequence of events is stricture formation at the site of injury, with obstruction of the proximal duct leading to obstructive jaundice with recurring attacks of cholangitis and eventually biliary cirrhosis with portal hypertension, splenomegaly and formation of œsophageal varices. This condition, the result of surgery in a young patient, is a particularly distressing lesion.

We lack a satisfactory solution to the problem, although many methods of overcoming the stricture have been suggested. All methods have in common a tendency towards re-formation of strictures. Prostheses used to bridge gaps or to maintain the patency of the new anastomoses invariably become occluded with bile salts and pigments. These facts have placed emphasis on prevention rather than cure, leaving the problem of methods of repair open for further investigation.

Since the site and extent of damage to the ducts varies, different methods of repair have been devised. These fall into three groups:

1. Anastomosis of proximal duct to distal duct or some part of the gastrointestinal tract.[1-3]

2. Grafting of the defect between proximal and distal duct.[4-7]

3. Bridging of the defect with prosthetic conduits.[8, 9]

At the present time the method of choice is the anastomosis of duct to duct, and if this is impossible, anastomosis of the duct to the gastrointestinal tract. However, cases occur which are unsuitable for this method and it is in these that a graft or prosthesis must be used.

Early workers on bile duct repair employed tubes of rubber, silicon and vitallium. Murray and Janes[9] in 1955, reported survivals for eight years in dogs, using vitallium tubes. Clinical use in human subjects of all prostheses has been less successful, indicating that success in experimental animals does not ensure success in persons.

Tolerance to infection of the biliary passages is higher in dogs than in man. There are differences in the chemical composition of the bile which may influence its precipitability. All substances thus far used in the formation of conduits have become encrusted and occluded with bile salts and pigments with resultant obstruction. In addition the use of rigid tubes gives rise to other complications such as ulceration, hæmorrhage, fistula formation, and eventual extrusion.

The subsequent report is concerned with the experimental use in dogs of a biliary

*From the McEachern Cancer Research Laboratory and Department of Surgery, University of Alberta, Edmonton, Alberta.
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conduit formed from a synthetic substance —ivalon.

Material and Methods

Ivalon[10] is a polyvinyl alcohol which is foamed and hardened with formaldehyde. It is white, with a bread-like texture, hard and rigid when dry but soft and resilient when wet. It is elastic and tough with a great tensile strength, and very light. When formed and heated to 95° C. and cooled, it maintains the shape given it by the form. It is inert except to concentrated acid and aqueous solutions of 30%-70% alcohol. When it is implanted into the body there is little inflammatory reaction and fibrous tissue grows into the interstices of the material.

The tubes for the bile ducts were constructed by driving small glass rods through rectangular pieces of ivalon. The ivalon was then compressed around the tubes by wrapping them with gauze. This was boiled for thirty minutes and allowed to cool. The ivalon took the shape of the tube; the outer wall remained rough, but the inner surface was smooth and shiny. The possibility existed that fibrous tissue would grow into the interstices to the inner surface and allow epithelialization, thereby preventing encrustation.

Ten adult mongrel dogs of both sexes were selected. Under pentobarbital (Nembutal) anæsthesia, a midline incision was made and the common bile duct exposed. The duct was divided and a tube of ivalon of the proper size was sewn end to end to the severed ends of the common duct, using 4-0 silk as interrupted sutures. The tubes were 2-3 cm. in length and 2-4 mm. in diameter. No form of drainage was provided.

Serum bilirubin was estimated at intervals from four to 10 weeks. A rise in serum bilirubin level was quickly manifest by an icteric tinge to the sclera. The dogs were weighed at intervals. As the animals died or were sacrificed, post-mortem studies were carried out with particular attention to the biliary systems.

Results

It may be seen from Table I that of the ten dogs, two died of infection—one of peritonitis on the fourth postoperative day, the other of pneumonia on the twenty-seventh day. In the latter dog, the serum bilirubin level the day before death was normal, and at post-mortem the prosthesis was patent and enclosed in a fibrous capsule. Two animals died of complications resulting from technical errors. One of these survived for 27 days and at post-mortem examination the cause of death was found to be bile peritonitis, due to disruption of the proximal suture line, caused by angulation of the duct by a prosthesis of excessive length. The other animal became jaundiced on the seventh postoperative day and survived 57 days. Post-mortem examination showed a perforated duodenal ulcer. The proximal suture line of the bile duct was stenosed.

The fifth animal, a poor operative candidate, had a precarious postoperative course. On the seventh postoperative day it developed a wound dehiscence which was resutured. It did well for three months, when it became jaundiced and died at six months. At post-mortem the prosthesis could not be found but the site of the prosthesis was stenosed. One animal survived thirteen months. It had been well and died suddenly. The cause of death was not discovered since at autopsy the liver appeared normal. The prosthesis was enclosed in a fibrous capsule, the lumen was patent and there was no sediment in the bile ducts.

The next animal died at fourteen months. The cause of death was not obvious. It developed melæna and died. At autopsy the small bowel contained a considerable amount of blood and the mucosal surface showed numerous ulcerated areas. The gallbladder was thickened and contained muddy bile. The ducts were not dilated. The prosthesis was loose in a fibrous capsule and was encrusted with bile salts.

The three remaining animals were sacrificed at 28 months. One had produced three litters of pups in the interim, the father being one of the experimental group with a common bile duct prosthesis. The animals appeared to be well, with normal serum bilirubin values, but at post-mortem the proximal ducts were found to be dilated. The bile contained a considerable amount of sludge and sediment. The prostheses

TABLE I.—Summary of Follow-Up Studies in Dogs with an Ivalon Biliary Prosthesis

Dog No.	*Previous surgery*	*Survival*	*Cause of death*	*Post mortem findings*
1.	Nil	14½ months	Hæmorrhage ??	Liver firmer than usual; gall-bladder thickened, contained turbid bile; prosthesis encrusted but patent.
2.	Common duct cut with external drainage-24 hours	6 months	Obstructive jaundice	Cachexia; proximal ducts dilated; gall-bladder thickened, contained yellowish-green turbid bile. Prosthesis not found but at site marked stenosis.
3.	Cholecystectomy	13 months	Unknown; sudden death	Liver appeared normal. Ducts not dilated. No sediment or encrustation.
4.	Nil	28 months	Sacrificed	Serum bilirubin normal. Proximal ducts slightly dilated, contained turbid bile and sludge. Prosthesis encrusted. Gall-bladder thickened.
5.	Cholecystectomy	4 days	Bile peritonitis	Proximal suture line given way with bile leak.
6.	Cholecystectomy	28 months	Sacrificed	Serum bilirubin normal. Liver grossly firm. Proximal ducts dilated, contained turbid bile. Prosthesis encrusted.
7.	Cholecystectomy	27 days	Pneumonia	Liver normal, no dilatation and no evidence of sludge.
8.	Cholecystectomy	27 days	Bile peritonitis	Prosthesis kinked with bile leakage and scar. Animal jaundiced.
9.	Nil	28 months	Sacrificed	Gall-bladder thickened. Proximal ducts slightly encrusted with bile salts. Serum bilirubin normal.
10.	Nil	57 days	Perforated ulcer	Cachexia, jaundice. Proximal end of duct stenosed.

were encrusted with bile. The livers were smooth but firmer than normal. It is probable that the common ducts in these animals would have eventually become obstructed.

Discussion

From the foregoing results, several factors became apparent. Any prosthesis must be accurately placed without tension and without kinking to avoid breakdown of suture lines and biliary obstruction due to angulation. The evaluation of a prosthesis in the biliary tree requires at least a two-year follow-up, and success depends on avoidance of encrustation, a process which may be slow in developing. The lives of several of these animals would undoubtedly have been prolonged had they been re-operated upon and a new prosthesis inserted when the jaundice became apparent. Ivalon as used in this experiment does not resist the deposition of bile salts with eventual obstruction, a fate common to all substances thus far used as biliary duct prostheses.

Conclusion

An ivalon prosthesis in the common bile duct of a dog will act only as a temporary conduit for the transmission of bile. Prolonged follow-up proved that the prosthesis will become encrusted and the proximal duct dilated, indicating partial obstruction of the extrahepatic biliary tree.

Acknowledgment

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RÉSUMÉ

Les blessures de l'arbre hépatique constituent une inquiétude chirurgicale constante. La cause la plus fréquente est évidemment, de loin, la blessure du cholédoque au cours de la cholécystectomie: les conséquences en sont la formation d'une sténose cicatricielle avec obstruction rétrograde et ictère. Les méthodes qui visent à réparer ces lésions peuvent être classifiées comme suit: (1) anastomoses du canal proximal au canal distal ou au tractus gastro-intestinal; (2) greffe entre les canaux proximaux et distaux; (3) réparation par prothèse.

Il est certain qu'à l'heure actuelle la méthode de choix est l'anastomose des segments bout à bout, ou leur insertion dans le tractus intestinal. Toutefois il est des cas où elle est impraticable, et l'on doit recourir à la prothèse. De nombreuses expériences ont été faites à ce sujet chez le chien, qui ont donné de beaux résultats; malheureusement il n'en est pas de même chez l'homme.

Les auteurs décrivent leurs propres expériences, faites chez le chien, avec une matière plastique: l'ivalon. Cette substance donne peu de réactions inflammatoires et est facile à modeler. Elle fut essayée comme prothèse chez dix chiens, après section du cholédoque; les résultats fournis sous forme de tableaux montrent que, à la longue comme les autres substances déjà testées, l'ivalon se recouvre d'une croûte de sels biliaires qui entraîne l'obstruction biliaire.

CORRIGENDUM

In the article by Dr. R. C. Harrison on "The Present Status of the Gastric Antrum" (*Canadian Journal of Surgery*, Vol. 2, No. 3, April 1959, pages 295-300), a typographical error occurred on page 298 whereby the word "secretion" was substituted for "secretin". The paragraph in which the error occurred should read as follows:

"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 *secretin* 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."

GASTRIC POLYPS*

G. R. DAVIES, M.D. and B. A. JACKSON, F.R.C.S., *Saskatoon, Sask.*

THE WORD "POLYP" without further qualification has become synonymous with polypoid adenoma and statistically this is essentially correct. However, there are other tumours of the stomach, such as leiomyoma, lipoma, adenomyoma, neuroma and angioma, which can assume a polypoid configuration. It is also noteworthy that the x-ray appearance of a polyp may be simulated by a foreign body or a bezoar.

Of all the synonyms for gastric polyposis, polyadenoma gastrica is probably most apt since it describes the adenomatous nature of the lesion. Those terms which infer an inflammatory origin should be avoided, for whatever may be the cause of these neoplasms, it is quite well established that they are not inflammatory.

Polypoid gastritis (hyperplastic polyposis, gastritis polyposa) is an advanced variety of hypertrophic gastritis and is not neoplastic. Many cases are associated with hyperchlorhydria and peptic ulcer, while others appear to have no relation to peptic ulceration and are associated with hypoacidity or anacidity. Differentiation can usually, though not always, be made from true polyps by careful roentgenological examination and by gastroscopy. Operation is indicated only for the treatment of an associated ulcer, though it is sometimes performed because these mucosal folds cannot be distinguished radiologically from polypi.

It is necessary to distinguish between multiple polyps and polyposis. The distinction may be significant, since it is said that polyposis has a greater malignant potential. One outstanding paper (Brunn and Pearl, 1926) included in the polyposis group those cases with more than three polyps. Spriggs and Marxer defined polyposis as that situation in which diffusely scattered polypi are countable only with difficulty. In this series there are two cases which are equivocal because the polypi were confined to a small area and were not distributed throughout the stomach. This suggests that these two cases are not true examples of polyposis, but they have been included in that group because of the number of polypi present.

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

PATHOLOGY

Gastric polyps may be single or multiple. They may vary in diameter from a few millimetres to several centimetres and their bases may be broad or narrow. Their colour varies from pale pink to red to plum colour. The surface may be smooth and regular, lobular or tufted. Consistency ranges from soft and boggy to firm, but is never stony hard or craggy. In large series they are multiple in about 35% of cases and the vast majority occur in the lower half of the stomach. The surrounding mucosa is usually atrophic but may be normal.

Microscopically, adenomatous polyps exhibit proliferation of both epithelial elements and supporting connective tissue. The connective tissue varies considerably in its cellularity, and sometimes œdema and mucoid degeneration are seen. There is little or no evidence of inflammation. The muscularis mucosæ may be fragmented and enter into the stalk of the growth, a feature not present in hypertrophic gastritis (Fig. 1).

The epithelium is mucus-secreting and undergoes irregular hyperplasia, with the result that the gastric glands become irregular, tortuous and cystic; many of the cysts show papillary changes. The process is clearly demarcated from the surrounding mucosa which may be atrophic, normal, or on rare occasion hypertrophic.

MALIGNANCY POTENTIAL

The incidence of benign epithelial tumours in relation to malignant tumours of the stomach has been reckoned as 5% to 10% (Stout; Spriggs; Rigler and Erickson, the latter as quoted by Spriggs). If it were 10% and if 40% of these became malignant, then approximately 4% of carcinomata of the stomach would arise from polypi. Any precancerous lesion deserves

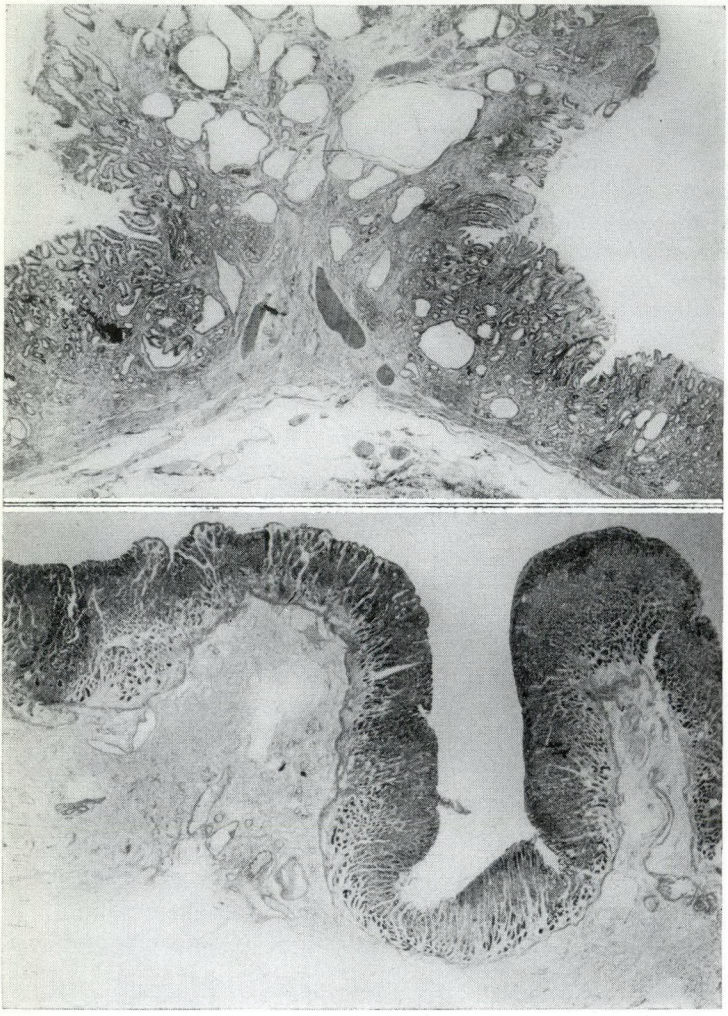

Fig. 1.—Microscopic appearance of gastric adenomatous polyp and of hypertrophic gastritis (polypoid gastritis) for comparison. (Magnification x 25.) **Fig. 1a.**—*Gastric adenoma.* — Observe the cystic hyperplasia of gastric glands as well as the fragmentation of muscularis mucosæ which enters into the stalk of the polyp.

Fig. 1b.—*Hypertrophic gastritis.*—Notice that the glandular hyperplasia is regular and that the muscularis mucosæ does not enter the stalk.

attention, for surgery is most rewarding at this stage.

There are several pathological situations which suggest that benign gastric polyps do become malignant. It may be difficult to determine whether a polypoid adenocarcinoma did or did not arise in a benign polyp. It is more convincing if multiple discrete polypoid malignant tumours occur together, or if one is found in the presence of benign polypi. It is most apparent when only part of a polyp is anaplastic.

An ulcerative carcinoma may have occurred coincidentally with benign polyps, or the malignant tumour may have been polypoid at an earlier stage in its development.

Another possibility is that the same mucosa is a potential source of both benign and malignant neoplasms and that the latter arise *de novo* instead of from a benign polyp. If this is true, all potentially malignant mucosa should be resected as a prophylactic measure.

A number of observers have reported the incidence of malignancy in their series of gastric polyps. A sampling of these reports is found in Table I. Stout presents some rather shocking figures. Of 32 cases of gastric polyps, 15 were associated with carcinoma, and of eight cases with multiple polyps, seven were associated with carcinoma. Hay concludes from his observations that the chances of malignancy occurring in small polypi are much less than the older literature would suggest. In a series of 81 lesions less than 2 cm. in diameter, only one

TABLE I.—Reported Incidence of Malignancy

Observer	Year reported	Incidence
*Meulengracht	1913	17%
**Mills	1922	4 out of 20
Brunn & Pearl	1926	12% (84 cases)
Miller, Eliason & Wright	1930	8 out of 23
***Lawrence	1936	6% with evidence of a malignant degeneration 12—22% associated with a cancer
Spriggs & Marxer	1943	9 out of 48 cases with evidence of malignant degeneration 3 out of 48 cases associated with a cancer
Hay	1956	1 out of 81 lesions less than 2 cm. in diameter was malignant 6 out of 14 cases greater than 2 cm. in diameter were malignant.

* As related by Brunn & Pearl[4]
** As related by Spriggs & Marxer[1]
***As related by Miller, Eliason & Wright[5]

was malignant. In contrast six out of 14 lesions greater than 2 cm. in diameter proved to be malignant.

Present Series

This is primarily a study of the clinical and radiological features of benign adenomatous gastric polyps. The cases described have been culled from the files of the Saskatoon Cancer Clinic, and only those treated surgically have been included so that the nature of the lesion could be verified microscopically.

The Cancer Clinic has followed up, for from two to three years, four patients who have not been operated upon but who satisfy all of the clinical criteria for benign gastric polyp. During the period of observation, there has been no change in the x-ray appearance of the lesions, nor has there been any deterioration in the patients' condition. These cases have not been included.

No attempt has been made to study the polypoid adenocarcinomata, so that it is not possible to arrive at any statistical conclusions as to the incidence of that type of lesion. Nor can it be suggested what percentage of benign polyps become malignant. For these figures it is necessary to depend upon the literature, some of which is reviewed earlier in this paper.

While the paper was in preparation, two patients with polypoid gastric lesions were operated upon at the University Hospital. Each was considered to have a malignant tumour preoperatively. Their radiographs were almost identical (Figs. 2a and 2b). Macroscopically, the tumours were practically identical but microscopically one was composed of adenocarcinoma throughout and the other was an adenoma. Both cases have been included to point out their similarities and to emphasize that it may be impossible to distinguish between the two by all available means short of microscopy. There are, therefore, 13 cases of benign polyps and one of polypoid adenocarcinoma reported here.

Symptoms and Signs

The average age in this group of 14 patients was 58.9 years at the time the diagnosis was made. None occurred in the first or second decades as others have reported, but ages ranged from 48 to 76 years. There are seven women and seven men in the series.

Many polypi must be asymptomatic, for they are found incidentally at fluoroscopy and autopsy. Symptoms include pain, indigestion, vomiting, hæmatemesis, malæna, anæmia and weight loss. The only remarkable pain is that which occurs as a result of obstruction, when a polyp prolapses through the pylorus, and it is accompanied by vomiting. This occurred only once in this series, but when the patient was seen for the first time, the acute episode had subsided. Pain is rarely described as severe, but this depends on the patient's pain threshold and reliability. It was described as occurring in the upper abdomen (epigastrium or right upper quadrant) by eight of these patients. In three there was simply discomfort or indigestion and two others complained of pain below the umbilicus. The history was of less than six months' duration in six cases, six months to a year

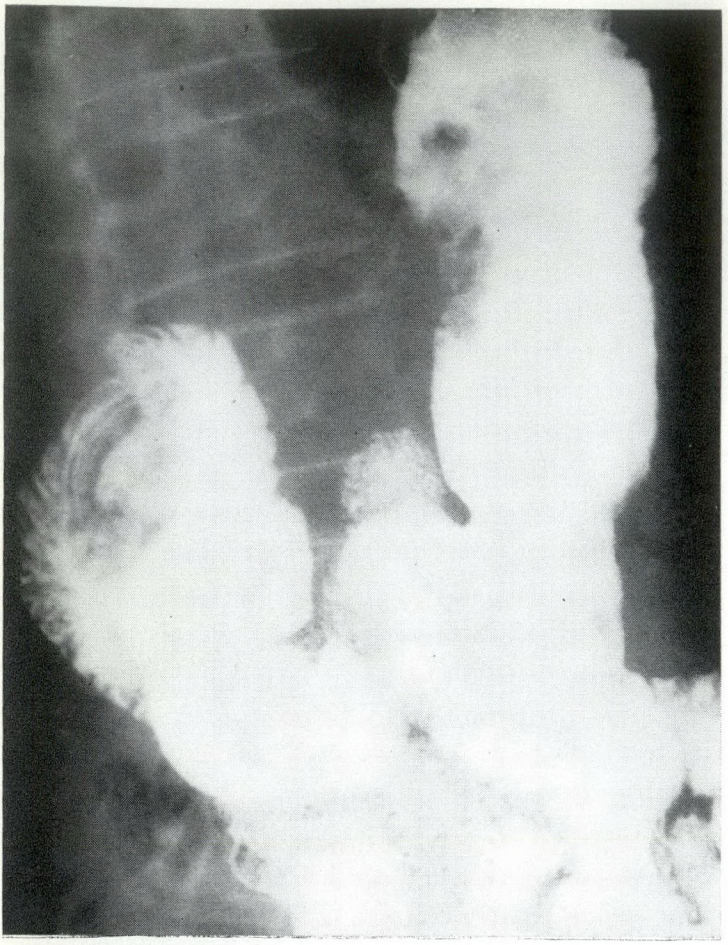

Fig. 2a.—*Benign adenomatous polyp.*—76 year old woman presented with massive hæmatemesis. No weight loss. Friable tufted tumour measured 5 x 3 cm. and was attached by a pedicle 3 cm. below the cardia. Observe similarity to polypoid adenocarcinoma in Fig. 2b.

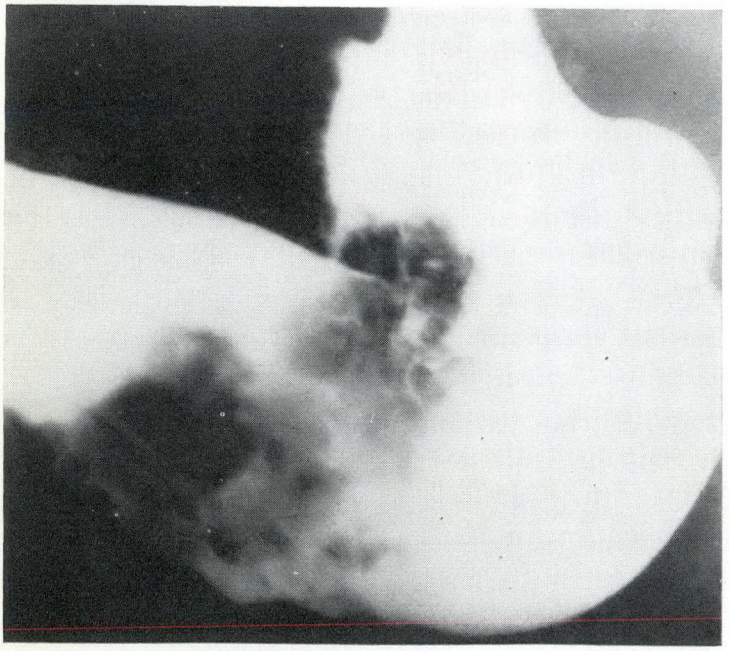

Fig. 2b.—*Polypoid adenocarcinoma.*—69 year old man presented with weight loss over a 10 month period and microcytic hypochromic anæmia. Friable tufted tumour measured 9 x 5 x 3 cm. and was attached by a pedicle 2 x 1 cm. Both cases (Figs. 2a and 2b) were diagnosed radiologically as malignant.

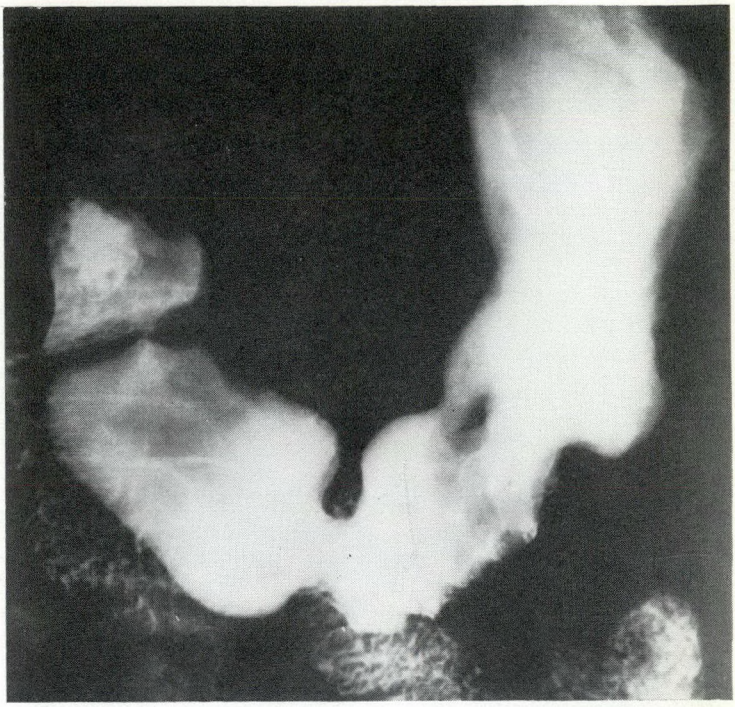

Fig. 2c. – *Multiple benign polypi.*—measuring up to 1 cm. in diameter. Confined to an area 6 x 8 cm. on the greater curvature.

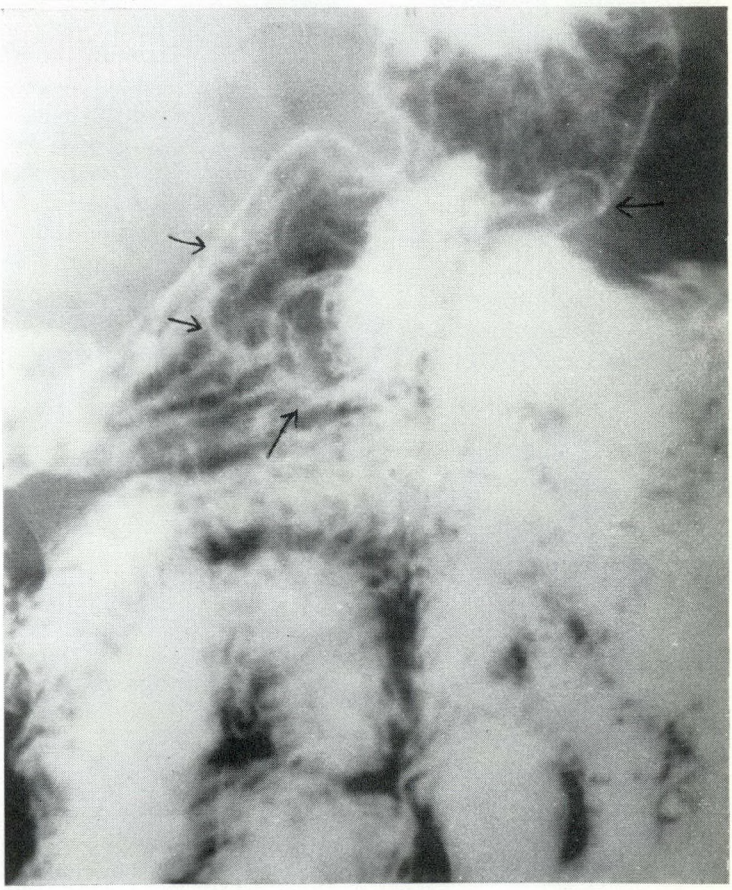

Fig. 2d.—*Multiple benign polypi.* – associated with hypertrophic gastric mucosa.

TABLE II.—Duration of History

Less than 6 months	6 cases
6 months to 1 year	2 cases
1 to 5 years	4 cases
5 to 10 years	1 case
Longer than 10 years	1 case
Total	14 cases

in two, one to five years in four, five to ten years in one, and longer than ten years in one (Table II).

Seven patients suffered weight loss of from eight to 17 pounds. In one case, this was attributable to a reducing diet. Two had gained weight. In one of these cases the lesion was diagnosed radiologically and gastroscopically as carcinoma but its benignity was proven histologically. In the remaining cases there was no remarkable change in weight.

Chronic anæmia was not a prominent finding. "Pernicious anæmia" occurred once, having been diagnosed four years before the polyp was discovered (see footnote, Table V). Hypochromic anæmia caused the presenting symptoms in the only case of malignant polyp in this study. Severe hæmorrhage occurred twice. In one instance, the patient suffered massive melæna one month after the polyp was found. In the second case, the first evidence of the polyp was hæmatemesis eight hours before admission to hospital. In the remaining ten patients the hæmoglobin level exceeded 12.8 grams per 100 ml. The stool was examined for occult blood in only a few instances, but in all of these the result was negative.

Eight patients had complete achlorhydria and one patient hypoacidity; gastric acidity was not stated in the other five. This agrees with the findings of other observers. Perhaps in many cases it is the achlorhydria and not the presence of the polyps which is responsible for digestive complaints.

Physical signs are usually absent, and none were elicited in this series. A mass may be palpable if the bulk of the stomach is increased by a greatly thickened adenomatous mucosa, or if the polypi are large. Signs of pyloric obstruction may be demonstrated during an acute episode, but those of chronic obstruction rarely occur since the obstruction is usually intermittent and transient.

Associated Diseases

One patient in the group suffered from "pernicious anæmia" and this same man had had poliomyelitis in early childhood. Three patients had a history of previous biliary tract disease and two of these had had cholecystectomy seven and eight years before the diagnosis of gastric polyp was made. One male had a gastro-enterostomy 37 years previously for peptic ulcer, and had had sporadic episodes of indigestion throughout the intervening years. None of the patients were reported to have oral or ano-rectal pigmentation, nor were any of them known to have polypi elsewhere in the gastrointestinal tract. In only the occasional case was the colon thoroughly investigated, however.

Diagnosis

"Benign polyp" was diagnosed roentgenologically in eight patients in this series. In a few, however, carcinoma could not definitely be ruled out. Malignancy was definitely diagnosed by the radiologists three times, and two of these tumours proved to be benign microscopically. Gastroscopy was employed in only four cases and was confirmatory or otherwise helpful in two of these.

Satisfactory x-ray investigation demands adequate preparation to ensure that the stomach is empty at the time of examination. The barium suspension must be of a rather watery consistency and must be administered slowly. Fluoroscopy is performed carefully and films should be obtained in several projections. If a filling defect is found, the criteria of benignity include a smooth contour, unimpaired peristalsis and distensibility of the stomach. Sometimes a pedicle can be demonstrated. Malignancy is suggested by a rough outline, broad base, fixity and lack of distensibility in the region of the tumour. The rugæ close to the lesion are distorted and impairment of peristalsis is an early feature. Representative x-ray pictures are shown in Fig. 2.

TABLE III.—Type of Operation Performed
14 operations — 9 surgeons

5 cases of polyposis	3 subtotal gastrectomies 2 gastrotomies (local extirpation)
3 cases of single polyp	1 subtotal gastrectomy 2 gastrotomies (local extirpation)
6 cases with two polyps	3 subtotal gastrectomies 2 partial gastrectomies 1 sleeve resection

Indications for Surgery in the Present Series

These patients were all referred to the Saskatoon Cancer Clinic on the suspicion that they harboured a malignant lesion. Suspicion runs high in patients of this age group with digestive complaints, achlorhydria and perhaps weight loss, in whom a filling defect is demonstrated in the stomach. This was the foremost indication for operation in 12 of these cases.

The other indications for surgery are pyloric obstruction, bleeding and pain. Only one of the histories suggested a recent bout of obstruction and in this case the chief indication was fear of malignancy. One patient experienced massive melæna, and operation was performed on this account. At operation this particular patient was found also to have a jejunal diverticulum in the wall of which was an adenomyoma. It was not possible to determine the source of the bleeding. The diverticulum was resected and the polypi were removed through a gastrotomy incision.

The oldest patient in the group was asymptomatic until serious hæmatemesis occurred on the day of admission. Her blood was restored to normal by transfusion and in a few days her condition had stabilized sufficiently for x-ray investigation and gastroscopy to be undertaken. The lesion was considered to be malignant. Resection was subsequently carried out and the polyp was shown microscopically to be benign.

As stated previously, pain was rarely severe, even though present to some degree in most instances. It is difficult to determine just what part it played in the decision to operate, for it was overshadowed in every instance by the suspicion of malignancy.

TABLE IV.—Number of Polyps

A single polyp	3 cases
Two polyps	6 cases
Polyposis	5 cases

Types of Operation Performed

(See Table III)

Nine surgeons participated in the management of these 14 cases. Three patients had a single polyp and of these, two were removed through gastrotomy incisions and one was treated by subtotal resection.

There were six cases with two polyps, and each of these patients was treated by some form of resection procedure. The tendency in most cases was towards conservatism and none of the operations was radical with regard to node dissection. Three were standard subtotal gastrectomies. Twice, only the distal one-third was resected and on one occasion a modified sleeve resection was performed.

The same conservative attitude prevailed in the treatment of the five examples of polyposis. Three were treated by subtotal gastrectomy. In one patient there were two large polyps and eight small ones, the latter localized to a small area of mucosa. It was possible to remove all of these through a gastrotomy. The final case of polyposis was the one described previously, in which laparotomy followed a serious bout of melæna. In addition to a circumscribed area of adenomatous gastric mucosa, a jejunal diverticulum was found, in the wall of which was an adenomyoma. The diverticulum was resected and the plaque of gastric adenomata was removed through a gastrotomy incision.

Pathology in this Series

Three stomachs contained a single polyp; six others contained two polyps and five had polyposis. In seven cases the polyps exceeded 2 cm. in diameter and three of these cases were in the polyposis group. The adenomata were diffusely scattered throughout the entire stomach in one case only. The lower half of the stomach was

TABLE V.—Follow Up

Type of polyp	*Number of cases*	*Duration of follow up*	*Results of operation*	*Recurrence of polyp or carcinoma*
Malignant	1	5 months	Good	No
Benign	1	Postoperative death		
	2	6 months or less	Good	No
	2	6—12 months	Good	No
	2	1—2 years	1 good 1 dumping syndrome	No
	2	2—3 years	Good	No
	2	3—4 years	Good	No
	1	5—6 years	Fair Loss of weight, no other complaints	No
	1	7—8 years	*Good	No
TOTAL	14			

*This male patient was found four years before operation to have "pernicious anæmia" which responded poorly to specific therapy. Following subtotal gastrectomy the anæmia improved and for several years he has maintained a satisfactory hæmoglobin level without the use of any drugs.

affected in 11, and the upper half in two patients.

The gross appearance of the 13 benign lesions varied but remained within the limits of the description given earlier in this paper. Microscopically there were differences not only between cases but between polypi found in the same stomach. The histological criteria of benign polyp were fulfilled in each instance.

There was only one case of malignancy and this stomach contained two polypi. The larger measured 9 x 5 x 3 cm. and was attached to the anterior wall by a short thick pedicle. The smaller measured 2 x 1 cm. and hung from the posterior wall near the greater curvature. Each of these polyps was an adenocarcinoma and each exhibited invasion at its base.

Results and Follow-up

There was one postoperative death in this series of 14 cases. This patient had diffuse gastric polyposis and was treated by a subtotal gastrectomy. Wound disruption occurred in the third postoperative week and was followed by peritonitis and pneumonia.

The solitary patient with a polypoid adenocarcinoma had a subtotal gastrectomy six months before completion of this article. When examined five months postoperatively, he was symptom-free and had gained several pounds.

A serious dumping syndrome occurred in one patient after a high subtotal gastrectomy. She was seen at intervals for 21 months but then was lost to follow-up. When last examined she had lost weight and was experiencing diarrhœa and postprandial distress. A barium series examination at that time revealed a functioning stoma and no evidence of tumour.

The remaining 11 patients have been followed up for periods varying from less than six months to more than seven years (Table V). All of them have had satisfactory results and none shows evidence of recurrence or of malignancy. Considering the protracted course of the disease, the duration of the follow-up is admittedly inadequate.

Discussion

An adenoma probably grows slowly over a period of years. It is not known how rapidly, or in fact how frequently, malignant change occurs; many polypoid adenocarcinomata may develop *de novo* and not in adenomata. It seems that multiplicity enhances the probability of malignancy, and Hay presents evidence that polyps larger than 2 cm. in diameter are more likely to be malignant.

Hay (1956) recommends operation: (a) for polyps larger than 2 cm. in diameter, (b) for any polyps suspected of being malignant by the radiologist or gastroscopist, (c) if symptoms are considerable and (d) if for any reason a program of proper observation cannot be followed. It is his policy to perform a 75% to 85% resection

even for single lesions, if associated with an atrophic mucosa and achlorhydria. Single polyps near the cardia are removed with a margin of mucosa through a gastrotomy. If it should prove to be malignant when frozen sections are examined, a total gastrectomy with removal of the greater and lesser omenta and the spleen is carried out. If multiple benign lesions are present in the cardia, he believes that total gastrectomy is indicated.

Miller *et al.*, in 1930, impressed with the high incidence of malignancy, proposed subtotal gastric resection with removal of lymph nodes, rather than local excision or sleeve resection. Mordvinkina reported 25 cases, in two-thirds of which malignant changes were found. He emphasizes early diagnosis and radical surgery, that is to say, resection rather than gastrotomy and local extirpation, and total gastrectomy in diffuse polyposis.

The mucosa in the distal half of the stomach would appear to have a greater propensity toward adenomatous growth and presumably therefore toward carcinoma. On this basis, a partial or subtotal gastrectomy is a rational procedure for polyps situated in this portion of the stomach.

Total gastrectomy is known to be more hazardous and is followed by considerably greater morbidity and mortality. It is never undertaken lightly and cannot be considered as a practical prophylactic operation. A conservative attitude is recommended, therefore, in regard to benign polyps in the upper third and near the cardia.

The interior of each stomach should be scrutinized through a gastrotomy incision. A polyp near the cardia should be removed with a full thickness wedge of stomach wall or with a rim of mucosa, whichever is feasible, for frozen section examination. It is unlikely that this cautious approach to a malignant polyp will have any adverse effect on the ultimate outcome. If frozen sections are reported to show malignancy, a radical total gastrectomy may be the procedure of choice.

If such a lesion from the cardia is pronounced benign on the basis of frozen sections, no further resection is required on its account. Should the paraffin sections subsequently reveal cancer, further resection may be performed as early as two weeks after the first procedure. In this way, the patients with benign lesions not destined to become malignant are spared the risks and unpleasant consequences of more extensive surgery. A few of the patients so treated may later succumb to gastric carcinoma, but this disadvantage will be offset by the smaller operative mortality.

Summary

Benign gastric polyps have been discussed generally with regard to their pathology and potential for malignant change. Fourteen cases treated surgically in this centre have been described. In 13 of these, the lesion was benign and in one malignant. The latter is included primarily to point out its clinical and pathological similarities to an adenomatous polyp. The series of cases is discussed from the point of view of diagnosis, indications for surgery and surgical management.

Benign polyps frequently simulate carcinoma clinically. In many, if not most, of these cases, it is impossible to determine their benignity by x-ray methods. Gastroscopy was employed so infrequently in this series of patients that it is not possible to assess its value. Any patient in whom the nature of a filling defect in the stomach is doubtful should be afforded the benefits of a laparotomy.

The considerable morbidity and mortality associated with total gastrectomy militates against its use as a prophylactic procedure. Polyps situated near the cardia should be dealt with conservatively until their nature is determined. Subtotal or partial gastrectomy, on the other hand, is a rational and practical procedure for benign polyps in the distal half of the stomach.

Acknowledgments

The authors are grateful to Dr. T. A. Watson, Director of the Saskatoon Cancer Clinic, for making its records available. The opinions expressed in this paper are those of the authors and not necessarily those of the Cancer Clinic. Gratitude is due also to the following for their assistance in the preparation of this article: Dr. E. M. Nanson,

Dr. D. F. Moore and Dr. E. Andres, all of the University Hospital. They also thank the surgeons concerned for permission to report their cases.

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Résumé

Le terme polype sans autre qualificatif évoque un adénome polypoïde et du point de vue des statistiques cet usage est justifiable. Il existe cependant d'autres tumeurs de l'estomac comme les léiomyomes, les lipomes, les adénomyomes, les neuromes et les angiomes qui peuvent aussi assumer une forme polypoïde. Ces tumeurs ne sont pas d'origine inflammatoire et les excroissances polypoïdes de la muqueuse que l'on voit dans les gastrites ne sont pas d'origine néoplastique. Il convient d'établir une différence entre polypes multiples et polypose puisque celle-ci est susceptible de dégénérescence maligne.

Les polypes gastriques varient en dimension de quelques millimètres à plusieurs centimètres et leur pédicule peut être mince ou large. Ils sont multiples dans environ 35% des cas et la grande majorité est située dans la moitié inférieure de l'estomac. Leur potentiel néoplastique doit être évalué car c'est au stage de la lésion pré-cancéreuse que la chirurgie remporte ses plus grands succès. La preuve la plus évidente de cette possibilité consiste à découvrir une région d'anaplasie dans un polype par ailleurs bien différencié. Les chiffres de fréquence de ce phénomène varient avec les auteurs (*vide* Tableau I). D'autres se basent sur la grosseur de la tumeur, la malignité commençant à se manifester au-delà de 2 cm. de diamètre.

La présente série est extraite des archives de la clinique du cancer de Saskatoon et comprend 13 cas de polype bénin et un adéno-carcinome polypoïde. La lésion est habituellement silencieuse et latente. Les symptômes, lorsqu'il y en a, sont la douleur, l'indigestion, les vomissements, l'hématémèse, le melæna, l'anémie et l'amaigrissement. L'examen physique peut révéler une masse gastrique si la tumeur est volumineuse; la plupart du temps il ne montre rien. S'il y a invagination de la tumeur à travers l'anneau pylorique on pourra observer des signes transitoires d'occlusion. La principale indication opératoire repose sur la probabilité de cancer chez des sujets de cet âge qui présentent de l'achlorhydrie, une perte pondérale et une image lacunaire à la radiologie. La gastroscopie et l'examen aux rayons X favorisent la découverte de ces tumeurs mais ils ne peuvent en déterminer la nature histologique.

La gastrotomie ou la résection gastrique partielle sans évidement ganglionnaire sont considérées comme des interventions suffisantes dans les cas de tumeurs bénignes. La gastrectomie totale en raison de sa mortalité et de sa morbidité ne peut être envisagée comme une mesure prophylactique. Le seul malade de cette série porteur d'adénocarcinome possédait deux polypes dont chacun montrait un envahissement du pied. Il y eut une mortalité post-opératoire et un cas grave de dumping. Les autres malades observés pendant une période variant de six mois à sept ans se portent bien. Les opinions de Hay et de Miller sont données dans le texte. Selon les auteurs de cet article une attitude conservatrice peut être tolérée dans les cas de lésion bénigne du tiers supérieur de l'estomac près du cardia.

DIAGNOSIS OF GASTRIC POLYPS

In the hospitals of Saratov, Russia, a series of 280 cases of gastric polyps or polyposis was reviewed by Protopopov (*Klinicheskaya Meditsina* No. 4: 73, 1959) with special reference to diagnosis. He states that the radiologist has no difficulty in diagnosing polyps of a diameter of 0.3-0.5 cm. or more, but may not discover smaller or relatively flat lesions on barium examination.

In 75% of the present series the polyps were small (0.3-1 cm. in diameter) or else there was an early stage of gastric polyposis. In many cases with a doubtful radiological diagnosis, the surgeon cannot palpate any abnormality through the stomach wall, and the author advocates an exploratory gastrotomy in such instances, although even this is insufficient at times. In five out of 20 cases in this series, polyps overlooked at exploratory gastrotomy were subsequently seen on x-ray examination. Where the surgeon cannot confirm at operation the presence of polyps discovered by the radiologist, he should have the latter present with him in the operating room to help locate them.

In view of these diagnostic difficulties the author is against the use of localized measures (excision, electro-coagulation, wedge resection) to deal with polyps, and feels that gastrectomy is the operation of choice, particularly in view of their tendency to become malignant.

SMALL BOWEL OBSTRUCTION: A REVIEW OF OPERATIVE CASES FROM THE ROYAL ALEXANDRA HOSPITAL, EDMONTON, 1953-1957

JOHN HNATUIK, M.D., and H. L. RICHARD, M.D., F.R.C.S.[C], *Edmonton, Alta.*

THIS PAPER IS A REVIEW of 128 cases of small bowel obstruction treated by surgical intervention at the Royal Alexandra Hospital, Edmonton, between 1953 and 1957. During this period 340 cases were admitted with a diagnosis of small bowel obstruction, but the following categories were excluded from the present study: (1) Cases of pyloric obstruction due to duodenal ulceration. (2) Cases in which there was a preoperative diagnosis of small bowel obstruction but in which the obstructing lesion was found to be in large bowel. (3) Cases of incomplete small bowel obstruction in which conservative treatment was elected. (4) A single case of internal herniation and strangulation in which no operation was undertaken and in which diagnosis was made only at post-mortem.

ETIOLOGY

The underlying lesions responsible for the 128 small bowel obstructions which came to operation are tabulated in Table I.

Adhesions accounted for the obstruction in 60.1% of cases. Of these 77 patients, 74 had undergone a previous laparotomy, and in 34 of these the previous operation was an appendectomy.

Only 20 patients, or 15.6%, had small bowel obstruction due to hernia. That this is only slightly more than one-quarter the number obstructed by adhesions is attributed to the increasing number of abdominal operations and the increasing number of early herniorrhaphies carried out at the present time.

There was one case of prolapse of bowel through the vagina. This was in a 50 year old woman who had had radiation treatment for cancer of the uterus. During intercourse one night she experienced severe pain and noticed blood on the perineum, and a loop of bowel protruding through the vagina. She was in the operating room within one hour of the incident. The surgeon replaced the bowel through the hole in the posterior vaginal wall, and then proceeded with a pelvic laparotomy. There was a 12 inch (30 cm.) loop of bluish-purple bowel present, which after a few minutes' observation was considered viable, and no resection was carried out. The postoperative course was uneventful.

One patient, a 22 year old woman, presented with a superior mesenteric artery syndrome. She had experienced repeated bouts of vomiting since childhood. The diagnosis was made preoperatively by the radiologist. At laparotomy a duodenojejunostomy was performed.

The patient, who was ultimately found to have an adenoma of jejunum, had been admitted two years before operation with a history of severe gastrointestinal hæmorrhage, and was discharged without a diagnosis. Her last admission was for pain and vomiting for two days and for tarry stools. The adenoma had produced a jejuno-jejunal intussusception. A resection of the involved bowel was carried out.

AGE DISTRIBUTION

Table II shows the age distribution by decades. Adhesions produced obstructions from infancy through the ninth decade, the highest incidence being found between the ages of 20 and 60. Intussusception was confined to the ages six days to four years, except for the one case in a 29 year old woman with adenoma of the jejunum.

Hernias produced small bowel obstruction in every age group, except for the decade between 10 and 20 years. The highest incidence occurred between the ages of 50 to 70 years.

CLINICAL PICTURE

Although the "typical" clinical picture of small bowel obstruction consists of colicky pain, vomiting, constipation, and abdominal distension, one or more of these features may be absent, as shown in Table III. It is of interest that in 28 cases the absence of vomiting is specifically recorded in the history. An illustrative case is that

TABLE I.—Causes

	No. of cases	% of total	Male	Female	Resections	Deaths
1. Adhesions	77	60.1	34	43	11	4
2. Hernias	20	15.6	6	14	8	1
Inguinal	12					
Femoral	4					
Umbilical	3					
Internal	1					
3. Intussusception	15	11.7	13	2	1	2
4. Volvulus due to bands	6	4.6	6	0	3	0
5. Carcinomatosis	3	2.4	2	1	3	1
6. Malrotation*	1	0.8	1	0	1	0
7. Prolapse of bowel through vagina.	1	0.8	0	1	1	0
8. Sup. mes. artery syndrome	1	0.8	0	1	1	0
9. Argentaffinoma of small bowel	2	1.6	1	1	2	0
10. Adenoma of jejunum	1	0.8	0	1	1	0
11. Trichobezoar	1	0.8	1	0	0	0
Totals	128	100%	64	64	32	8

*Both incomplete duodenal obstruction and non-strangulating midgut volvulus present.

of a 30 year old woman who had been ill for three days and was admitted to hospital in a moribund state. She was markedly dehydrated. Her abdomen was flat, soft and silent. There was no history of vomiting. After vigorous preoperative therapy she was taken to the operating room, where a gangrenous loop of bowel caused by pelvic adhesions was found. Her postoperative course was uneventful except for a transient psychosis.

Of the 28 cases with no vomiting:

a. Eight had small bowel obstruction due to adhesive bands with only slightly dilated loops.
b. Seven at surgery had definitely dilated loops of bowel with impaired blood supply, but which did not require resection.
c. Four had strangulated hernias.
d. Two had a volvulus requiring resection.
e. Three were found to be obstructed by adhesive bands and to require bowel resection.
f. One had an intussusception.
g. One had an intraluminal foreign body.
h. Two had carcinomatosis peritonei with obstruction.

Thirty-seven patients had no preoperative x-ray examination (Table IV). These were mainly cases of strangulated hernias, or instances in which the clinical picture was clearly one of a small bowel obstruction and radiological confirmation was not deemed necessary.

In only eight cases were the x-ray findings reported as normal in the presence of intra-abdominal lesions. These were:

TABLE II.—Age Distribution

	0–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80	80–90
1. Adhesions	8	4	12	10	12	14	7	8	1
2. Hernias	2	0	3	1	2	5	5	1	1
3. Intussusception	15	0	1	0	0	0	0	0	0
4. Volvulus due to bands	1	0	1	0	1	1	2	0	0
5. Carcinomatosis	—	0	—	—	—	—	1	2	—
6. Malrotation*	0	0	1	0	0	0	0	0	0
7. Prolapse of bowel	0	0	0	0	0	1	0	0	0
8. Sup. mes. artery syndrome	0	0	1	0	0	0	0	0	0
9. Argentaffinoma of small bowel	0	1	0	0	0	0	0	0	1
10. Adenoma of jejunum	0	0	1	0	0	0	0	0	0
11. Trichobezoar	0	1	0	0	0	0	0	0	0
	26	6	20	11	15	21	15	11	3

*Both incomplete duodenal obstruction and non-strangulating midgut volvulus present.

TABLE III.—Preoperative Findings

No vomiting present	28 cases
No abdominal distension	42 "
Bowel movements stated to be normal	29 "
Diarrhœa	6 "

1. Volvulus of small bowel due to non-fixation of mesentery.

2. Three cases of volvulus around adhesions with strangulation of bowel.

3. Bowel obstruction due to adhesions with markedly dilated loops.

4. Internal hernia through mesentery of small bowel.

5. Intussusception in a child in which only plain films of the abdomen were taken.

TABLE IV.—Relation of X-ray Findings to Lesion Seen at Operation

1. No x-ray examination preoperatively	37 cases
2. Neg. x-ray report but definite lesion	8 "
3. X-ray findings correlated with findings at operation	83 "

Preoperative Preparation

Although long tubes are used in many centres, and are said to have certain advantages in mechanical small bowel obstruction, they were employed in only 18 of our 128 cases. In only nine had the tube passed beyond the stomach and in only one beyond the duodenum. Intravenous fluids were administered preoperatively to all patients in this series.

Table V shows the use of antibiotics in the cases reviewed.

TABLE V.—Antibiotics

One antibiotic used	63 cases
More than one antibiotic	33 "
No antibiotic	32 "

Accuracy of Preoperative Diagnoses

The numbers of correct, incorrect, and incomplete preoperative diagnoses are indicated in Table VI. The preoperative diagnosis was that reported on the operative sheet.

Those three patients without a preoperative diagnosis, and who were booked for "exploratory laparotomy", were found to have:

1. Argentaffinoma of small bowel.

TABLE VI.—Relation of Preoperative Diagnosis to Lesion Found at Operation

	No. of cases	%
1. Correct diagnosis of "small bowel obstruction" and etiology	53	41.4
2. Called "small bowel obstruction" but etiology not specified	57	44.4
3. Exploratory laparotomy without diagnosis	3	2.3
4. Preop. diagnosis incorrect	14	11.1

2. Volvulus of small bowel due to non-fixation of mesentery.

3. Adhesions.

Those cases of small bowel obstruction, which were incorrectly diagnosed preoperatively, were most often mistaken for acute appendicitis or perforated duodenal ulcer.

Delay in Surgical Intervention

As indicated in Table VII, the average time from onset of symptoms until operation was 71 hours. The delay on the part of the patient in seeking medical attention was not as great as the delay which occurred in hospital.

TABLE VII.—Preoperative Delay

Average time from onset to admission to hospital	30.5 hours
Average time from admission until operation	40.5 "
Average time from onset of symptoms to operation	71.0 "

The interval from the time of admission to operation in the cases requiring resection is shown in Table VIII. Of the 32 patients requiring resection 26 had gangrenous bowel. Only 14 of these 26 patients were operated on within 24 hours of admission, and 12 were in hospital between 24-72 hours before operation.

Delay, whether or not it was necessary for preoperative preparation, probably in-

TABLE VIII.—Delay before Resection

Resections required—32 (23.4%)

Time in hospital before resection	*No. of cases*	%
Less than 24 hours	14	43.7
24 - 32 hours	5	15.6
48 - 72 hours	7	21.8
72 - 96 hours	5	15.6
11 days	1	3.3

TABLE IX.—Indications for Resection

	No. of cases	%
Bowel gangrenous	26	81.6
Viable bowel but resected for tumour, stenosis, etc.	6	18.4

creased the number of cases requiring resection.

TABLE X.—Details of Resections: Types of Anastomoses and Stomata

	No. of cases	%
1. End to end	26	81.6
2. Side to side	3	9.4
3. Ileo-transverse colostomy	1	3.0
4. Ileostomy	1	3.0
5. Jejunostomy	1	3.0

Operation

The appropriate surgical procedure was carried out by a variety of surgeons and members of the resident staff. One patient out of four required resection of bowel, and the indications for these bowel resections are given in Table IX. The types of anastomoses and stomata which were made are indicated in Table X.

Complications

The complications which occurred at operation or postoperatively are shown in Table XI. If the patient was discharged and returned a few months later with another obstruction, this was considered as another case of small bowel obstruction, rather than a complication, for purposes

TABLE XI.—Postoperative Course

1. Average stay in hospital 15.7 days		
2. Complications	44 cases	34.3%
Atelectasis and pneumonia	7	15.9
Pulmonary embolism	1	2.2
Broncho-pleural fistula	1	2.2
Coronary occlusion	3	6.8
Congestive heart failure	3	6.8
Cardiac arrest	1	2.2
Cerebro-vascular accident	1	2.2
Brain abscess	1	2.2
Acute psychosis	1	2.2
Wound infection	8	18.8
Wound hæmatoma	4	9.1
Severe postoperative ileus	4	9.1
Peritonitis	4	9.1
Pelvic abscess	1	2.2
Electrolyte imbalance	3	6.8
Chickenpox	1	2.2
3. Mortality	8	6.2%

TABLE XII.—Intussusception in Children: 15 Cases (11.7%)

Average age	12.3 months	
Youngest	9 days	
Oldest	4 years	
Males	13	
Females	2 Ratio 6.5:1	
Average time from admission to O.R.		4.7 hours
X-ray findings:		
No x-ray exam.	4	16.6%
Flat plate with diagnosis small bowel obstr.	5	33.3
Ba enema with diag. intussusception	5	33.3
Flat plate only—called normal	1	6.6
Deaths	2	13.3
Resections	1	6.6

of this review. Pulmonary complications and wound infections predominated (Table XI). The majority of the complications occurred in the older age group. There were eight deaths—a mortality rate of 6.2%.

Intussusception in Children

It was thought that a separate review of intussusception in children would be of interest (Table XII). More than 15 cases of intussusception in children were seen in the five year period under review, but this study does not include those cases in which a complete reduction was accomplished by barium enema. There were two deaths—a mortality rate of 13.3%.

Summary

A series of 128 cases of mechanical small bowel obstruction, operated upon, is reviewed. Symptoms, physical findings, laboratory data, and x-ray findings, as well as the preoperative diagnoses, are correlated with the lesions found at operation. Delay from the time of admission to the time of operation may have increased the number of cases requiring resection. The long intestinal tube was used preoperatively only 18 times. There was a mortality rate of 6.2%, which is acceptable, considering that 51 of these 128 patients were over 50 years of age. Finally, 15 cases of surgically treated intussusception in children are reviewed separately.

Résumé

Les auteurs analysent les faits cliniques de 128 patients atteints d'occlusion intestinale du grêle et opérés à l'hôpital Royal Alexandra d'Edmonton durant la période de 1953 à 1957 inclusivement. Les adhérences intestinales furent la cause dans 60% des cas, la hernie dans 15.6% et l'invagination intestinale dans 11.7%. Si l'on considère la fréquence en rapport avec l'âge, les adhérences se sont manifestées le plus souvent dans le groupe de 20 à 60 ans, l'invagination chez les sujets de six jours à quatre ans et la hernie dans tous les groupes d'âge (sauf de 10 à 24 ans) avec un maximum de fréquence entre 50 et 70 ans. Bien que le tableau clinique de l'obstruction du grêle consiste en crampes abdominales, vomissements, constipation et météorisme, il est intéressant de noter que 28 des 128 cas, soit 22% ne présentaient pas de vomissements, 42 ne montraient pas de distension abdominale, et 6 avaient la diarrhée. Chez 37 patients on ne fit pas de radiographie parce que le diagnostic semblait évident; chez huit des 91 autres cas la radiographie se montra inefficace et chez la moitié d'entre eux (quatre cas) la lésion était un volvulus. Dans 86% des cas le diagnostic pré-opératoire fut confirmé à l'opération.

La préparation pré-opératoire consista en cathétérisme gastro-intestinal dans 18 cas sur 128; tous les patients reçurent des solutés intraveineux en abondance suivant leur besoin. On administra un ou plusieurs antibiotiques à 96 malades. La recoupe des statistiques révèle qu'il s'écoula un temps moyen de 30.5 heures entre le début des symptômes et l'admission à l'hôpital, et de 40.5 heures entre l'admission et l'intervention. Bien que les auteurs n'expliquent pas si le délai à l'hôpital releva du retard dans la décision opératoire ou de la préparation adéquate du malade, ils opinent qu'un certain nombre de résections auraient pu être évitées si on était intervenu plus tôt. La résection s'imposa dans 25% des patients; on procéda à un abouchement du grêle bout à bout dans 81.6% des cas.

Des complications post-opératoires se manifestèrent dans 34% des cas et consistèrent surtout en complications pulmonaires (20%) et infections de la plaie (18.8%). Il y eut 8 décès, soit un taux de 6.2%.

Les auteurs concluent cette étude avec un rapport sur 15 cas d'invagination intestinale survenue chez des enfants. Le plus jeune était âgé de neuf jours et le plus vieux de quatre ans: le groupe était composé de 13 garçons et de deux filles. L'intervalle moyen entre l'admission et l'intervention fut de 4.7 heures. On pratiqua une résection chez un patient. Deux malades moururent dans cette série, soit 13.3%.

En somme les auteurs nous rapportent leurs observations sur une série intéressante de cas d'occlusion du grêle; on en retient que la période entre le diagnostic et l'opération devrait être abrégée et qu'une intervention plus précoce permettrait d'obtenir des résultats encore plus satisfaisants.

TEXTBOOK OF SURGERY. Edited by Guy Blackburn and Rex Lawrie. 1122 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1958. $20.00.

This book has been written by Mr. Guy Blackburn and Mr. Rex Laurie with the assistance of 14 of their colleagues, all members of the staff of Guy's Hospital, London. While they have called on others to write upon special subjects, the book is strengthened by the fact that the major portion has been written by the authors themselves. It contains more than 1000 pages, and yet one gains an impression of commendable brevity; fewer words could not well have been used. Because of this, it is frankly a textbook for the undergraduate. It is liberally and clearly illustrated. There is a well balanced presentation of the various fields of surgery, and over-emphasis on a field or fields in which the author has a particular interest has been avoided. It has been written over a short period and can claim to be really up-to-date as it leaves the press. Paper and type are excellent.

This is a modern British textbook of surgery which teachers may well recommend to students.

BONE TUMOURS. Louis Lichtenstein. Veterans Administration Center, Los Angeles. 402 pp. Illust. 2nd ed. The C. V. Mosby Company, St. Louis, 1959. $12.00.

This is the second edition of this book. The general arrangement is identical to that of the first edition, with but minor additions and alterations to the text. Several new illustrations have been added; as in the first edition the quality of reproduction is excellent.

New chapters have been added on clinical management of bone lesions that may be tumours, benign osteoblastoma, tumours of periosteal origin, and tumours of joints, bursæ, and tendon sheaths. The appearance of these chapters does not reflect new knowledge as much as the author's changing views on the classification of bone tumours.

Apart from the author's occasional literary lapses (e.g. "this tumour predilects the bones of the lower extremity") the book is of high standard and continues Dr. Lichtenstein's reputation as a world authority in the bone tumour field.

NEW INSTRUMENTS FOR SYMPATHECTOMIES

A. CLIFFORD ABBOTT, F.R.C.S.(Edin.), F.R.C.S.[C], F.A.C.S., *Winnipeg, Man.*

FOR THE PAST 25 years, I have been interested in developing a set of dissectors and hooks suitable for all types of sympathectomies, especially those in the lumbar and thoracic regions. It has been my experience that the vast majority of instruments manufactured heretofore have many defects. Many are too short, others are too light to give the proper delicate sense of touch, especially those with hollow handles. Some are too cumbersome and incorrectly designed for bloodless dissection. A properly balanced dissector is hard to find.

In my opinion, proper weight with perfect balance is essential for easy manipulation. The handles must be finely tooled to prevent slipping, especially where a gentle rolling of the instrument in the fingertips is essential for meticulous and accurate dissection. Fingertip control is a necessity. The instrument must be of proper length to reach the area of dissection with ease. The shape, size, and angle of the tip of the instrument are very important. It must be fine enough to carry out a meticulous bloodless dissection. The various tips must be so constructed as to make it possible to approach an area of dissection from any angle.

The instruments to be described were originally designed for sympathetic surgery only. We now use them for dissection of the renal pedicle, pneumonectomy, vagotomy, dissection of the biliary tree, and vascular surgery.

The basic set consists of four dissectors and four hooks. Each instrument consists of two parts, a handle and a dissecting or hook shaft. The instruments are of solid chrome steel. The proximal portion, or handle, is 5 inches (12.5 cm.) long, ¼ inch (0.6 cm.) in diameter, and finely tooled. The distal portion of the handle is 1½ inches (3.75 cm.) long, and gracefully tapered down to the junction with the dissecting tip or hook. Fig. 1 illustrates the various dissecting tips and hooks. From top to bottom:

1 and 2—Sharp and ball-pointed small curved hooks.

3—Right-angled ball-pointed hook, measuring ⅜ of an inch (0.9) cm. in diameter.

4—Large curved hook.

5—Straight dissector—the actual dissecting tip is ⅝ of an inch (1.5 cm.) in diameter.

6—Right-angled dissector – identical in size but the dissecting tip has been turned to a right angle.

7—Dinner fork dissector. The dissecting tip is set at an angle of 20 degrees and is mildly convex. This instrument is very useful when dissecting in a shallow depression.

8—Goose-neck dissector. This instrument resembles a golf putter. The dissecting tip is ½ inch long, set at an angle of 120 degrees to the shaft and mildly rotated on itself. It is very useful when dissecting around a ganglion or larger vessel, especially the posterior part of the dissection in a ductus arteriosus operation.

For thoracic sympathectomies, I have used the bed of the eighth or ninth rib, or preferably an intercostal incision between the seventh and eighth, or eighth and ninth rib. A long instrument is required to reach the first and second lumbar ganglia and fourth dorsal ganglion. To facilitate this dissection, a second set of similar instruments has been made, 13 inches long. Each component part has been increased 1 inch in length; otherwise the instruments are identical.

ACKNOWLEDGMENTS

I would like to express my appreciation to Mr. Albert Stewart for making all my original models. Over the intervening years, these instruments have been modified and refined. His suggestions have been very helpful.

These instruments are now manufactured by Down Bros. and Mayer and Phelps, Limited, 70 Grenville Street, Toronto, Ontario.

Department of Surgery,
University of Manitoba and St. Boniface Hospital.
The Abbott Clinic, Winnipeg, Manitoba.

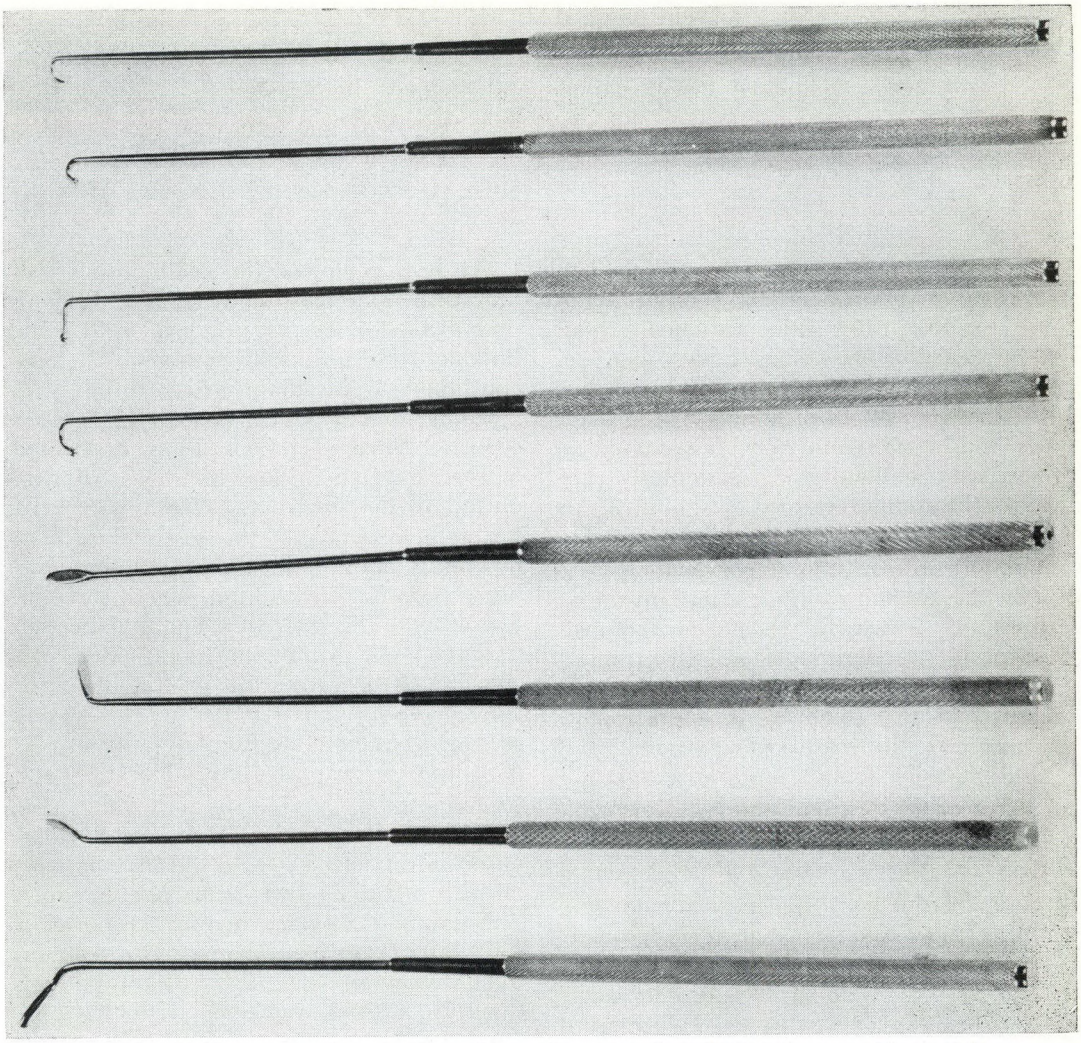

Fig. 1.—Illustrating four hooks and four dissectors described in the text. The weight in these instruments has been so delicately distributed that they are constantly balanced in the surgeon's hand.

Résumé

Présentation d'une série de nouveaux instruments développés par l'auteur pour les sympathectomies: crochets courbes pointus et mousses; crochet pointu à angle droit; grand crochet courbe; dissecteur coudé à angle droit; dissecteur type fourchette; dissecteur type "cou d'oie". Les usages de ces divers instruments sont expliqués; outre les sympathectomies, ils peuvent être utilisés avec commodité pour d'autres interventions (pédicule rénal, pneumonectomie, chirurgie sur l'arbre biliaire, chirurgie vasculaire).

INTRA-ATRIAL MYXOMA*

Review of Literature and Report of a Right Atrial Myxoma Diagnosed Preoperatively and Successfully Treated

R. K. PADHI, M.B., B.S., H. G. KELLY, F.R.C.P., and R. B. LYNN, F.R.C.S.,
Kingston, Ont.

IN SPITE OF GREAT ADVANCES in the field of heart surgery, intracardiac tumours continue to pose diagnostic problems. About 90 intracardiac myxomas have been reported in the Anglo-Saxon literature since 1951, but in only 16 of these was the diagnosis made preoperatively. Twenty-seven others were encountered accidentally during exploration for suspected mitral or tricuspid stenosis.

The purpose of this paper is to review briefly the subject of intracardiac myxomas and report a case in which the diagnosis was made preoperatively and the tumour successfully removed under hypothermia. Because of the difficulty in preoperative diagnosis of these tumours, we feel that individual case reports are warranted.

REVIEW OF THE LITERATURE

Yater in 1931[1] reviewed the subject of tumours of the heart and pericardium and found about 75 myxomas in the literature. He added two cases of his own. The next review in the English literature was by Prichard[2] almost 20 years later. He reported 146 metastatic and four primary tumours of the heart. Prichard found 126 intracardiac myxomas in the literature and added two of his own. However, Mahaim[3] in 1945 as quoted by Brewin[4] had estimated the total number of reported myxomas as near 200. Intracardiac myxoma is the commonest primary tumour and constitutes over 50% of all primary intracardiac tumours. According to Prichard 75% of them occur in the left atrium, the great majority of them being attached to the intra-atrial septum in the region of fossa ovalis. "Rarely the tumour may arise on a valve and only one has been reported in the left ventricle".[52]

We have reviewed the available English literature from the time of Prichard's comprehensive review reported in 1951. The bulk of the intracardiac myxomas have continued to be diagnosed on the post-mortem table.[7-30] Out of a total of 90 cases reported since 1951, only 13 were located in the right atrium, and none has been reported in the ventricles. One report[30] describes a myxoma in the left atrium of a newborn infant, and these tumours have been reported in children aged three to five years.[31] The sexes were equally affected.

Forty-seven of the 90 myxomas were discovered at necropsy. In 16 the diagnosis was made preoperatively and 27 others were encountered accidentally during exploration for suspected mitral or tricuspid stenosis.

The first intracardiac myxoma to be diagnosed during life was reported by Goldberg *et al.*[31] in 1952, but their patient died. Bahnson and Newman in 1952[32] reported a right atrial myxoma diagnosed preoperatively by angiocardiography. In this patient, the right atrium was opened during one-minute inflow occlusion but only a part of the tumour was removed. Twenty-four days later the patient died. Many unsuccessful attempts have been made since.[31-45]

The first intracardiac myxoma, diagnosed preoperatively and successfully and completely removed, was reported by Crafoord.[49] Crafoord's patient had a left atrial myxoma and the diagnosis was made by angiocardiography. The tumour was removed on July 16, 1954, using cardio-pulmonary bypass. Bigelow[50] is credited with the second successful removal of a left atrial myxoma under hypothermia. Up to the time of writing 18 intra-atrial myxomas, including the one in our own case, have been successfully removed and reported in the literature (Table I).

*Department of Surgery—Cardio-Thoracic Unit, Queen's University, Kingston, Canada.

TABLE I.—Successful Removals of Intra-Atrial Myxomas

Reference	*Age and sex*	*Location*	*Diagnosis*	*Surgical adjunct, etc.*
49	40-50 F	Left	Angiocardiography	Pump oxygenator, July 16, 1954.
50	56 F	Left	During exploration for mitral stenosis.	Hypothermia, April, 1955.
51	33 M	Left	During exploration for mitral stenosis.	Hypothermia.
52	57 F	Left	During exploration for mitral stenosis.	Pump oxygenator.
53	61 F	Right	Angiocardiography	Pump oxygenator.
54	25 M	Left	During exploration for mitral stenosis.	Hypothermia.
55	38 F	Left	During exploration for mitral stenosis.	Hypothermia.
56	45 F	Left	During exploration for mitral stenosis.	Pump oxygenator.
56	48 M	Right	During exploration for tricuspid stenosis.	Pump oxygenator.
57	52 F	Left	During exploration for mitral stenosis.	Hypothermia.
58	51 F	Left	Angiocardiography	Pump oxygenator.
59	14 F	Right	Fluoroscopy	Hypothermia.
59	51 F	Left	Angiocardiography	Hypothermia.
60	51 M	Right	Angiocardiography	Pump oxygenator.
61	50 F	Right	Angiocardiography	Pump oxygenator.
62	43 F	Left	During exploration for mitral stenosis.	Closed technique using double cardiotomy.
63	26 M	Right	Calcified tumour, preoperative diagnosis.	Hypothermia.
Present report	46 M	Right	Angiocardiography	Hypothermia.

All except one, reported by Fatti,[62] were removed through an open cardiotomy using either hypothermia or the extracorporeal circulation.

A review of the clinical history of the reported cases shows that definite diagnostic criteria were common to all. Briefly, an intracardiac tumour manifests itself by virtue of its space-occupying nature, by its faculty of obstructing or distorting a valve orifice, or by embolization. One author[17] reports three left atrial myxomas which were fairly large and pedunculated. These three patients, who died from other causes, had no signs or symptoms referable to the myxoma. This, however, is unusual. The authors had occasion to see the necropsy of a person who had died suddenly and inexplicably four days after a straightforward cholecystectomy. At necropsy, a large pedunculated myxoma had passed through the mitral valve and completely blocked it. This patient had given no history referable to the heart. Brewin[6] reports the case of a man of 42 who while engaged in heavy labour suddenly lost the power in both legs and collapsed. Before this, the man had no symptoms referable to the cardiovascular system. At post-mortem, a myxoma from the left atrium was found to have detached itself from its pedicle and obstructed the aortic bifurcation.

The above-mentioned cases, however, are exceptions. The majority of patients with intra-atrial myxomas present with intractable congestive heart failure. Left atrial myxomas mimic mitral stenosis so closely that at times it is virtually impossible to make a distinction. Absence of an opening snap with the clinical picture of mitral stenosis is said to be diagnostic where a myxoma is suspected, but Lefcoe[48] reports a case of left atrial myxoma in which an opening snap was recorded by phonocardiography. Atrial myxomas produce embolic phenomena quite frequently and this has been well documented. Low-grade intermittent fever occurs not infrequently and subacute bacterial endocarditis may be difficult to rule out.

In this clinical confusion there are, however, a few points which may raise the clinician's suspicion: (1) absence of a history of rheumatic disease; (2) history of exaggeration of symptoms—dizziness, feeling of suffocation, and occasionally syncope—on change of body posture; (3) alteration in the character of murmurs with changes

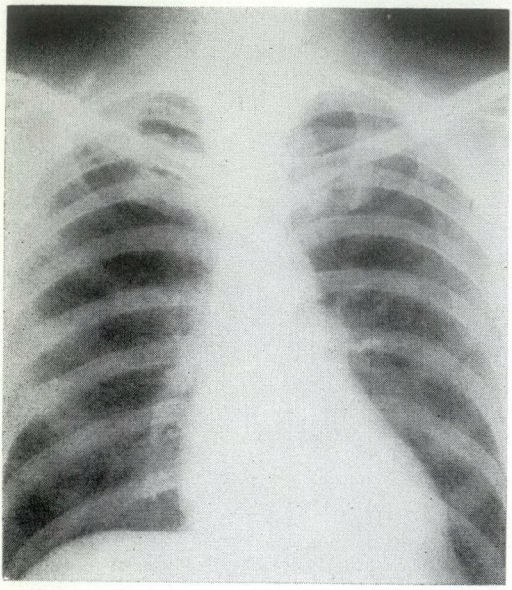

Fig. 1.—Postero-anterior radiograph of chest.

in posture; (4) progressive cardiac failure in spite of medical management; (5) disproportionate size of the heart compared with the severity of the symptoms; (6) low-grade and intermittent fever associated with embolic phenomena with persistently negative blood cultures.

Case Report

Mr. J.C.S., a farmer aged 46, had been unable to work for 18 months because of increasing shortness of breath and weakness. He had been admitted to hospital on three occasions during this period. At the time of his second admission he was in a marked state of depression and was unable to climb a flight of stairs. For the past six months he had noted numbness and crampy pain in the extremities on mild exertion or upon exposure to cold. A low-grade, intermittent fever was noted at the previous and present admissions. About six weeks before the present admission he developed a choking sensation and dizziness on lying on the right side.

He had enjoyed good health before the onset of the present illness and there was no history of rheumatic fever or heart disease.

Physical Examination

He was well built and well nourished with a sallow complexion. There was no dyspnœa at rest, or cyanosis or clubbing. The neck veins were full. Heart rate was 90 and blood pressure 120/95 mm. Hg. The heart was not clinically enlarged. A soft systolic murmur was heard in the tricuspid area; a changing diastolic murmur in the tricuspid area was also present. The liver was enlarged three fingers' breadths below the costal margin but not tender. The spleen was not palpable. All peripheral pulses were present. Physical examination was otherwise negative.

Investigations

Hæmoglobin value 14.2 g. per 100 ml., hæmatocrit 47%, total W.B.C. 19,300, E.S.R. 27 mm. in one hour; the differential count was normal. Repeated blood cultures were negative. All other laboratory investigations were negative.

The E.C.G. showed widespread repolarization and the impression was of myocardial ischæmia or myocarditis. X-ray examination of the chest and fluoroscopy showed clear lung fields and a normal sized heart (Fig. 1). At this stage of investigation, tricuspid stenosis or a constricting lesion involving the ventricles was considered.

Right heart catheterization showed the following pressures (in mm. Hg); P.C. (mean) 7; M.P.A. 24/12; R.V. 20/4; R.A. 18/8. Right heart catheterization was followed by angiocardiography which revealed a large filling defect occupying the right atrium (Fig. 2).

A diagnosis of right atrial myxoma was made preoperatively. On February 9, 1959, the patient was cooled to 29.8° C. by immersion in ice water. The chest was entered through a median sternotomy. The superior and inferior venæ cavæ were taped and the right atrium was then explored through the appendage. The presence of a large and firm tumour occupying almost the entire right atrium was confirmed. The incision in the appendage was closed. After occluding the

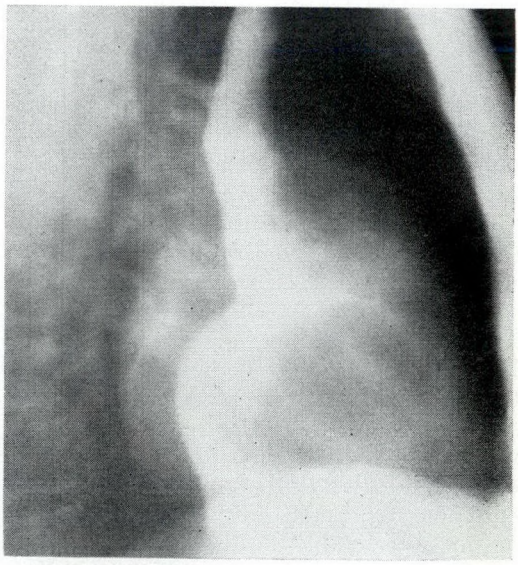

Fig. 2.—Lateral view of angiocardiogram.

venæ cavæ, the right atrium was widely opened. The tumour was attached to the auricular wall in between the openings of the inferior vena cava and the coronary sinus. The pedicle was short and about one cm. in diameter. The tumour was completely removed by cutting the pedicle flush with the atrial wall. The period of inflow occlusion was 4.8 minutes and during the entire operative period the heart showed no signs of strain or arrhythmia.

The postoperative course was uneventful and the patient was discharged on the 12th postoperative day relieved of all symptoms. He was seen one month after discharge and was back at work.

Pathology

The tumour weighed 100 grams, was egg-shaped and measured 9 cm. in length and 5 cm. in thickness. The surface was smooth, firm, and yellow at one end and pinkish at the other. On section, the tumour was composed of dark red and light pink material. The histological appearance was compatible with myxoma of the heart (Figs. 3 and 4).

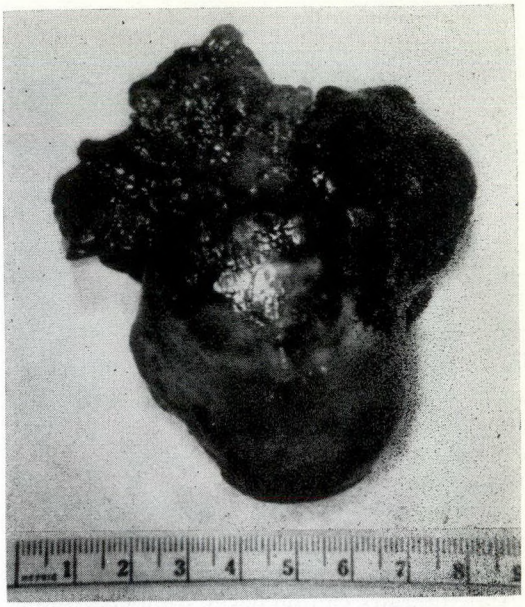

Fig. 3.—Gross appearance of myxoma removed.

Discussion

Right atrial myxomas are usually mistaken for tricuspid stenosis but the rarity of the latter lesion as an isolated finding should always arouse suspicion. In the present case the history of changing signs and symptoms led to cardiac catheterization and angiocardiography which established the diagnosis beyond any shadow of doubt.

The use of hypothermia and pump oxygenators in open-heart surgery has simplified the surgical treatment in intracardiac tumours. Removal of an intracavitary myxoma or any other pedunculated tumour presents little problem. However, their diagnosis still baffles the clinician. Gerbode wrote "the diagnosis of left atrial myxoma depends primarily on a high index of suspicion." All too often in the past the diagnosis of an intracardiac myxoma has been made on the autopsy table. With increasing awareness of these tumours and widespread application of valvular surgery, myxomas are being diagnosed preoperatively more frequently. The left atrial tumours mimic mitral stenosis so closely that a large proportion of these will continue to be diagnosed at the time of exploration for suspected mitral stenosis.

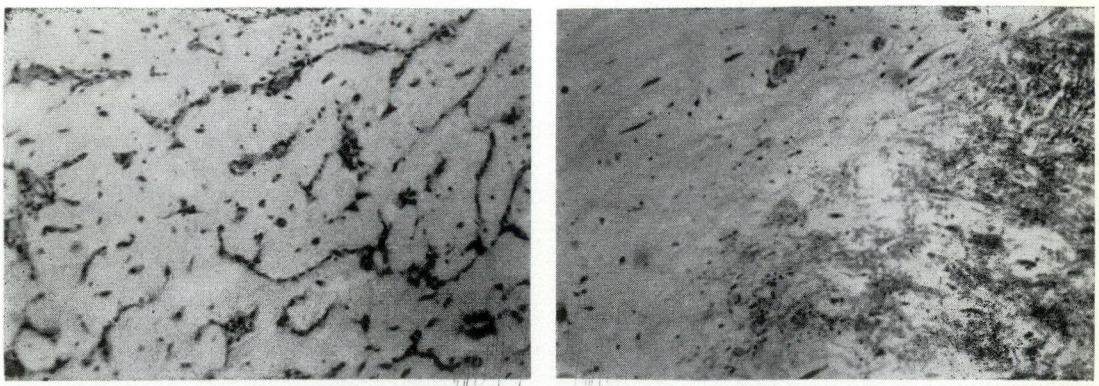

Fig. 4.—Microscopic appearance of tumour.

Since Goldberg's report in 1951, angiocardiography has become a standard diagnostic tool. Calcification of the intra-atrial myxoma is extremely rare although two calcified benign pedunculated intra-atrial tumours[42, 63] have been reported. Preoperative diagnosis in these cases becomes relatively simple.

Various ingenious methods have been employed in removing intra-atrial tumours. The closed techniques have rarely succeeded except in one case reported by Fatti.[62] Since the introduction of open-heart surgery there is no need for risky closed techniques. All intracardiac tumours should be removed under direct vision. The choice between hypothermia and cardiopulmonary bypass, as a surgical adjunct to open-heart surgery, is a matter of individual preference. It is our feeling that right atrial tumours can be easily removed under hypothermia during a brief inflow occlusion lasting four to six minutes. In dealing with a large right atrial tumour, the risk of breaking the tumour during cannulation of the inferior vena cava is eliminated by hypothermia. Simple exploration alone has resulted in massive fatal embolization.[40-42, 45] In dealing with left atrial myxomas it would be preferable to use the pump-oxygenator and elective cardiac arrest.

TABLE II.

Total number of cases reported since 1951		90
Post-mortem diagnosis	47	
Ante-mortem diagnosis	43	
Diagnosed at exploration	27	
Preoperative diagnosis	16	
No definitive treatment	4	
Unsuccessful removal	21	
Successful removal	18	7 from right atrium 11 from left atrium

Summary

A case of right atrial myxoma diagnosed preoperatively and removed successfully under hypothermia is reported.

The available English literature since Prichard's review in 1951 is reviewed (Table II).

Eighteen intra-atrial myxomas, including the present case, have been successfully removed. Only nine of these were diagnosed preoperatively.

Addendum

Since this paper was written Cooley, D. *et al.* (*A.M.A. Arch. Surg.*, 78: 410, 1959) have reported two further successful removals.

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Résumé

Environ 90 myxomes intra-cardiaques ont été rapportés par les auteurs de langue anglaise depuis 1951, mais on n'a posé un diagnostic pré-opératoire que dans 16 de ces cas. Vingt-sept autres furent trouvés accidentellement lors d'une exploration cardiaque. Au cours d'un rappel des principaux travaux sur ce sujet les auteurs de cet article font remarquer que des 90 cas cités plus haut seulement 13 de ces tumeurs étaient situées dans l'oreillette droite et aucune ne provenait des ventricules. C'est à Crafoord que revient l'honneur d'avoir fait l'exérèse complète d'un myxome intra-cardiaque avec diagnostic pré opératoire. Le diagnostic avait été posé grâce à l'angiocardiographie et l'intervention fut pratiquée à l'aide d'une dérivation cardio-pulmonaire (16 juillet 1954). Bigelow serait le second à avoir réussi cette opération et il eut recours à l'hypothermie.

Une tumeur intra-cardiaque peut manifester sa présence par sa masse même, par son effet sur l'orifice valvulaire ou par embole. La majorité des malades porteurs de myxomes dans une oreillette présentent des symptômes de défaillance cardiaque réfractaire à tout traitement. Lorsque la tumeur est située du côté gauche le tableau clinique ressemble à s'y méprendre à celui de la sténose mitrale.

Les auteurs rapportent le cas d'un fermier de 46 ans qui accusait de la dyspnée et de la faiblesse depuis 18 mois. En dépit d'un cœur de dimensions normales l'angiocardiographie montra une masse dans l'oreillette droite. On posa un diagnostic de myxome. Le 9 février 1959 grâce à l'hibernation le malade fut opéré et l'on retira une tumeur de 100 grammes mesurant 9 cm. sur 5 cm. Les auteurs recommandent l'intervention à cœur ouvert pour la résection de ces tumeurs. Les tumeurs de l'oreillette droite peuvent être enlevées facilement sous hypothermie permettant une interruption de la circulation pendant quatre à six minutes. Il serait préférable d'avoir recours à une pompe oxygénatrice dans les cas de tumeur de l'oreillette gauche.

LES STRUCTURES INFRAMICROSCOPIQUES NORMALES ET PATHOLOGIQUES DES CELLULES ET DES TISSUS. A. Policard et C. A. Baud. 476 pp. Illust. Masson & Cie, Paris, 1958. 5.200 fr.

Les histologistes du siècle dernier, faisant un travail remarquable avec ce que nous considérerions aujourd'hui comme des "moyens de fortune", décrivaient dans le protoplasme des structures *alvéolaires* ou *réticulaires* ou *fibrillaires*. Assez rapidement cependant, on dut reconnaître que tout cela n'était qu'artefacts de fixation et l'on considéra le cytoplasme comme "optiquement vide", c'est à dire comme formé d'une substance colloïdale sans structure. Puis, des considérations diverses, notamment cytochimiques, firent soupçonner dans ce gel une organisation, un compartimentement, un squelette. Effectivement, et c'est ce que la microscopie électronique a révélé, il existe, au delà du pouvoir de résolution du microscope optique "quelque chose". Ce quelque chose est fondamentalement un ensemble d'arrangements complexes de molécules de tailles extrêmement variables, donnant naissance à des structures diverses, mais définissables sinon toutes définies. C'est ce que l'on désigne par le terme de *structure ultramicroscopique* ou encore *infrastructure*.

Le but du livre des Professeurs Policard et Baud est de résumer et de condenser sous une forme commode l'ensemble des découvertes faites grâce à la microscopie électronique depuis les trente dernières années. Œuvre hardie à l'heure actuelle, car, sans aucun doute, beaucoup des résultats et des solutions proposés devront être révisés dans l'avenir. Œuvre énorme quant au travail de recherche personnelle et au travail de compilation que cette publication représente.

Cet ouvrage, dans l'intention des auteurs, s'adresse à des non spécialistes; dans ce cas il faut regretter que seulement dix pages aient été consacrées à l'exposé des méthodes utilisées: pour le profane, ce chapitre est à la fois trop long et trop court et à tout prendre, de compréhension difficile. Il est également regrettable que peu ou même rien ne soit dit des méthodes "de recoupement", telles la microscopie en contraste de phase, la microscopie interférentielle, la microspectrophotométrie et autres. Le lecteur peu informé risque d'être amené à conclure qu'une barrière infranchissable est baissée entre la microscopie optique et la microscopie électronique. Or, précisément, un avenir des plus prometteurs est celui des méthodes permettant de faire le pont entre les deux.

La deuxième partie, consacrée à la cellule, et la troisième, qui constitue un véritable traité d'histologie générale et spéciale à l'échelle électronique, sont dans l'ensemble bien conçues. Mais—est-ce par souci d'économie?—pourquoi les auteurs ont-ils préféré transcrire la grande majorité des microphotographies électroniques publiées ici en des schémas au trait, au lieu d'en donner des reproductions fidèles? Tout cela parle peu à l'imagination.

La bibliographie donnée en fin de chaque chapitre est volontairement sommaire. Il est regrettable que les références de littérature n'apparaissent pas dans le texte: ceci aurait grandement facilité la tâche du lecteur curieux d'en savoir plus sur tel ou tel point en particulier.

En conclusion, cet ouvrage constitue une excellente "mise à jour" de ces questions. C'est un résumé et un aide-mémoire commode pour le chercheur. Nous doutons cependant qu'il soit susceptible d'intéresser beaucoup le médecin non engagé dans la recherche.

DUPUYTREN'S CONTRACTURE: HEREDITY AS AN ETIOLOGICAL FACTOR

STUART D. GORDON, *Toronto*

ALTHOUGH IT IS now more than a century and a quarter since Dupuytren described the lesion which bears his name, we are still very much in the dark as to its etiology. One factor that has received fairly general support is heredity.[2, 5-8] The exact part played by heredity as an etiological factor is not yet fully known. The shifting population of a country like Canada makes the obtaining of accurate genealogical histories very difficult, and often impossible, as Corlette[3] has noted for Australia. Thus, when one comes across a well documented family history indicating the presence and absence of the disease through five generations, it would seem worth publishing.

While heredity does appear to play a role in the natural history of the disease, there is no agreement whether the factor concerned is a dominant or recessive one.

Conway has suggested that the disease may be sex-linked since, in his opinion, it occurs more frequently in men than in women. Since my observations[3] have led me to believe that the incidence of the disease is equal in the sexes it is, in my opinion, not sex-linked.

Fig. 1 illustrates the occurrence of Dupuytren's contracture through five generations. Two of the family on whom I have operated, a brother and sister, indicated by the encircled solid circles, have had the disease in both hands and both feet. Their maternal great grandfather had the disease in both hands, and for years before his death could not open his hands. Their two maternal uncles who had the disease died at 85. One maternal uncle is alive, aged 83, without evidence of the lesion. The two paternal uncles had the disease in both hands. They have two nephews and six nieces in their twenties who have not shown any evidence of Dupuytren's contracture.

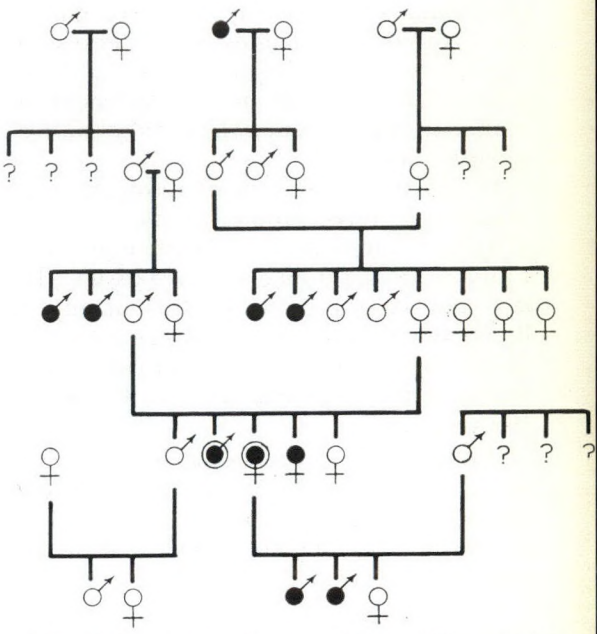

Fig. 1.—Five generations are shown. Open circles indicate individuals free of disease, solid circles those known to have had Dupuytren's contracture. Where nothing could be found out as to the presence or absence of the lesion, the individual is represented by a question mark.

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RÉSUMÉ

Le rôle des facteurs héréditaires dans l'étiologie de la contracture de Dupuytren a déjà été abordé par plusieurs. L'auteur présente ici, sous forme d'un tableau, l'histoire généalogique d'une famille suivie pendant cinq générations, où de nombreuses personnes ont été atteintes de l'affection.

RETROPERITONEAL LIPOMA CAUSING RENAL DISPLACEMENT*

R. G. WINRAM, M.D.† and J. N. WARD-McQUAID, M.S., F.R.C.S.,‡ *Mansfield, Eng.*

RETROPERITONEAL TUMOURS have intrigued physicians since the days of Morgagni[1] in the 17th century. This is partly because of the enormous size they may attain and also because of the extreme displacement of neighbouring viscera which they so frequently cause.

Herdman[2] classes primary retroperitoneal tumours in three groups—simple cysts, tumours of adrenogenito-urinary origin, and tumours arising from connective tissue. This last and largest group consists mainly of lipomata. One of these, an enormous tumour of 275 lb. at the time of the patient's death, was described by Delameter[3] of Cleveland 100 years ago.

We are now reporting the successful removal of a considerably smaller, but still large, retroperitoneal lipoma, which had displaced the left kidney so that it lay beside its fellow on the right.

CASE REPORT

G.W.T., a white man aged 60, was first seen at the Mansfield General Hospital in July 1956. He complained of increasing abdominal enlargement for the last 10 years, swelling of the legs and breathlessness for two years, and swelling of the scrotum for the past year. He had been treated for "heart trouble" with diuretics and digitalis during the previous few months.

On clinical examination there was no abnormality of the cardiovascular system except a raised blood pressure of 180/105 mm. Hg. A large mass filled most of the abdomen. This was mainly on the left side and was dull on percussion. There was no shifting dullness. An attempt at paracentesis was made and failed.

An intravenous urogram (Fig. 1) showed two normally functioning kidneys on the right side. The left kidney was rotated and lay in front of and slightly below the right kidney. A barium enema examination (Fig. 2) was reported on by Dr. E. J. S. Townsend as showing displacement of the descending and pelvic colon to the right.

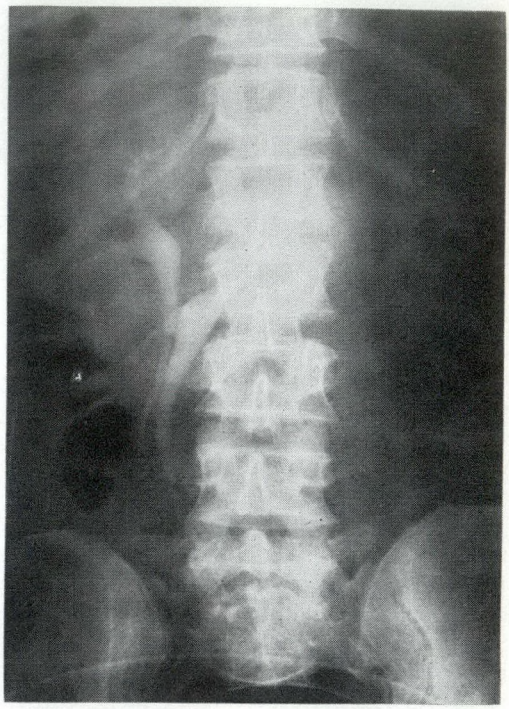

Fig. 1.—Intravenous urogram showing displacement of left kidney to the right.

A diagnosis of retroperitoneal tumour was made but the patient did not agree to operation until the following year.

Operation was finally carried out in June 1957, under general anæsthesia given by Dr. H. A. Buck. A left transverse subcostal muscle-cutting incision was made. A very large lobulated retroperitoneal lipoma was separated easily from the peritoneum, left kidney and ureter, and was found to extend into the left inguinal canal. The mass was excised and the kidney and ureter then fell back to their normal position. No special fixation was employed. The hernia was repaired and the hydrocele sac excised. The tumour weighed 21 lb. and Dr. A. B. Hill reported that it was a benign lipoma.

Convalescence was delayed by a microcytic anæmia and urinary retention with infection. Both responded to simple conservative measures. The patient was seen a year later when he was well and had returned to his employment. There was no evidence of recurrence. An intravenous urogram (Fig. 3) showed normally functioning kidneys in their usual position.

*From the Mansfield Group of Hospitals, Mansfield, Notts., England.
†Surgical Registrar, Mansfield Group of Hospitals.
‡Consultant General Surgeon, Mansfield Group of Hospitals.

Discussion

A retroperitoneal tumour causes no specific symptoms, and the clinical signs and symptoms depend on the abdominal enlargement and the characteristic renal and sometimes colonic displacement, which were all features in our case.

Schulte and Emmett[4] found associated extreme renal displacement in 72.5% of these tumours. Greene[5] described three patients, all with considerable renal displacement. Abdominal pain may occasionally occur and is due to either hæmorrhage into or degeneration of the tumour.

The abdominal enlargement and common œdema of the legs due to impaired venous return suggests cardiac failure with ascites. This may be excluded by negative paracentesis.

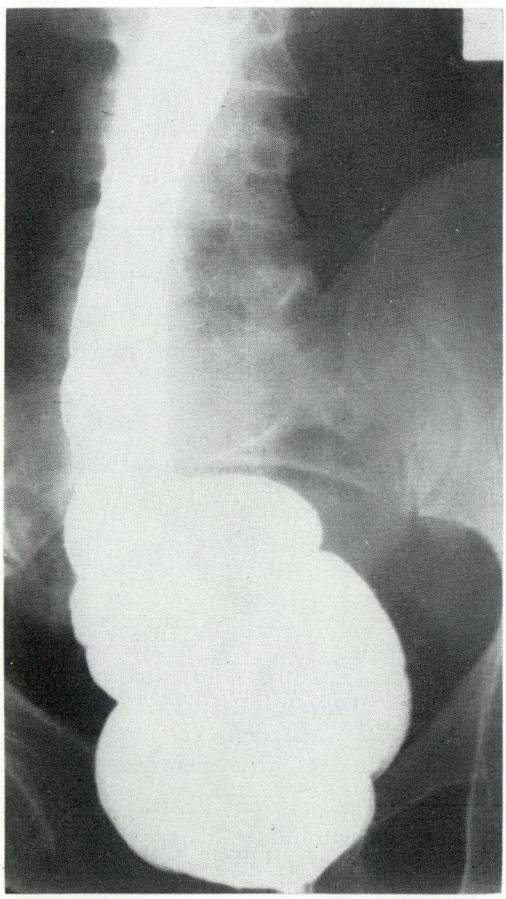

Fig. 2.—Barium enema examination showing displacement of descending and pelvic colon to the right.

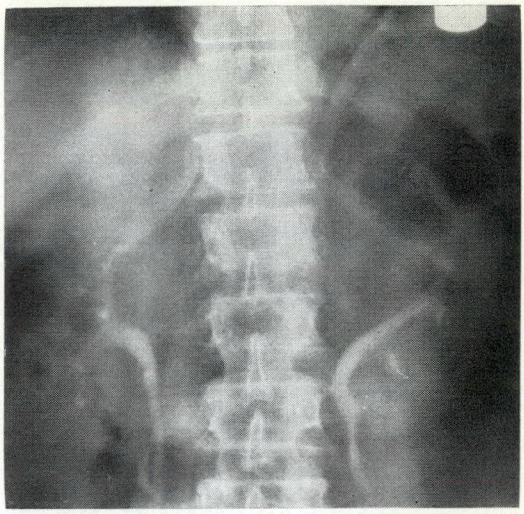

Fig. 3.—Normal postoperative urogram.

There is considerable variation in the incidence of malignancy reported in different series. Thus Herdman[2] found one malignant tumour (a liposarcoma) among 14 primary retroperitoneal tumours in Oxford. Von Wahlendorf[6] found that 14% of 168 cases reported in the literature were malignant. De Weerd and Dockerty[7] found that no less than 28 out of 43 cases were malignant; these patients were seen at the Mayo Clinic between 1910 and 1946.

Retroperitoneal lipomata are usually well encapsulated but often have long finger-like projections, as in our case, where part of the tumour passes through the inguinal canal into the scrotum. Complete removal may be difficult and recurrence is not uncommon. This may be due to incomplete removal or sometimes to liposarcoma degeneration in a remnant of the tumour. Certainly the lipomata are not as innocuous as those that occur in the subcutaneous tissues and the prognosis must be guarded.

After removal of the tumour the displaced kidney does not require special fixation and functions well.

Summary

The successful removal of a benign retroperitoneal lipoma weighing 21 lb. is described. The left kidney was completely displaced to the right side. After operation the kidney was normal in position and function.

ACKNOWLEDGMENT

We are indebted to Dr. E. J. S. Townsend for the radiographs and Dr. A. B. Hill for the pathology reports.

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RÉSUMÉ

Les auteurs rapportent ici le cas d'un homme de 60 ans qui présentait depuis deux ans une tuméfaction abdominale avec enflure des jambes et du scrotum. A l'examen du système cardio-vasculaire tout était normal sauf une T.A. à 180/105. Une masse, matte à la percussion, tendait à envahir tout le côté gauche de l'abdomen. Une urographie intraveineuse permit de voir que le rein gauche était tourné sur son axe, placé en avant et en dessous du rein droit; de plus le lavement baryté montra un déplacement du côlon descendant vers la droite. On posa le diagnostic de tumeur rétropéritonéale et une exploration fut ultérieurement pratiquée: on trouva un énorme lipome rétropéritonéal lobulé, facile à disséquer, qui fut enlevé en totalité. La tumeur pesait 21 lb. (9.5 kg.). A l'heure actuelle le patient est en excellente santé.

Après un bref aperçu de la littérature, les auteurs insistent sur les deux points suivants: ces tumeurs, qui sont fréquemment très volumineuses sont faciles à extirper, mais du fait de leurs lobulations et de leurs digitations, il est toujours possible d'en oublier un fragment qui sera à l'origine d'une récidive; d'autre part, les organes déplacés par la tumeur se remettent en position correcte d'eux mêmes et aucune fixation chirurgicale n'est nécessaire.

BOOK REVIEWS

(See also pages 411 and 420)

BRITISH SURGICAL PRACTICE: SURGICAL PROGRESS, 1958. Edited by Sir Ernest Rock Carling and Sir James Paterson Ross. 442 pp. Illust. Butterworth & Co. Ltd., London and Toronto, 1958. $10.50.

"This is the eighth of an annual series of supplements to British Surgical Practice. By this means the eight volumes of the main work are kept up to date in the ever increasing field of surgical knowledge, by original articles, surveys and abstracts".

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Some 25 additional new titles appearing in the 1958 volume are necessitated by recent surgical advances, so that it truly merits the title of a progress volume.

Subjects which have not hitherto appeared in preceding volumes are the following: collagen diseases; operative cholangiography; the carpal tunnel syndrome; the use of the ileum in urology; and pre-clinical carcinoma of the cervix.

Other items brought up to date are blood transfusions; fluid and electrolyte balance; Crohn's disease; ileostomy for ulcerative colitis; surgery of the œsophagus; septal defects in the heart; surgical induction of labour; management of acute head injuries; surgery of glaucoma; surgery of the spinal cord; cervical rib.

The remainder of the book contains excellent surveys of recent developments in surgery of the stomach and duodenum, in pulmonary surgery and in surgery of the central nervous system.

On the whole this 1958 Progress Volume can be considered a valuable edition to the forward looking surgeon's library.

VASCULAR SURGERY. Geza de Takats, Presbyterian-St. Luke's Medical Center and Research and Educational Hospitals, Chicago, Illinois. 726 pp. Illust. W. B. Saunders Company. Philadelphia and London, 1959. $17.50.

Geza de Takats has produced an excellent monograph dealing in detail with the problems of arteries and veins. He has wisely excluded the heart from his discussion and has made this book an authoritative modern reference book on vascular lesions.

It is quite apparent that a tremendous amount of reading and personal research have formed the foundation for this book. In a subject which is so young and to which new discoveries are coming almost weekly, he has imparted this information apparently up to date of publication.

His physiological and patho-physiological descriptions are vivid and very helpful. Some of his diagrams are brilliant. His methodical descriptions of the lesions are followed by brief yet complete reports on treatment.

(Continued on page 428)

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BOOK REVIEWS

(Continued from page 424)

The only disappointment is his small manual of operative descriptions with diagrams. Appearing at the end of the book, almost as though it were an afterthought, it is incomplete and cannot replace a proper atlas of surgical procedures on this subject in which techniques and materials are new. It might better have been omitted.

De Takats has been a giant among vascular surgeons. It is, therefore, only fitting that such a magnificent volume should come from him. This will be a standard book of reference on the subject if frequent new editions keep up with this fast-moving specialty.

A PRACTICE OF THORACIC SURGERY. A. L. d'Abreu. 619 pp. Illust. 2nd ed. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1958. $17.00.

It is five years since the first edition of this book and this second edition demonstrates the definite changes that have taken place in the realm of thoracic surgery. The author covers the fundamental parts of the book in his usual satisfactory manner and there is relatively little change in the first two parts.

In Part Three on pulmonary tuberculosis there is a little less to be said, as the treatment of pulmonary tuberculosis is being relatively well standardized and on the whole the disease is much nearer to being controlled.

The great change in the book, of course, is in the surgery of the heart and great vessels and the author covers very satisfactorily the field of cardiac surgery. He limits his discussion to the procedures that are moderately well established and recognized at the present time, and discusses quite fully the new methods of investigation. Certainly, with the rapid strides being made in cardiac surgery, a new edition was indicated and the author has maintained the high quality of the first edition.

MODERN TRENDS IN SURGICAL MATERIALS. Edited by Leon Gillis. 266 pp. Illust. Butterworth and Co. Ltd., London and Toronto, 1958. $14.50.

"As the physician must know the chemistry, the available forms, the reactions of and the reactions to a new drug before giving it to his patients, so must the surgeon know all about the materials he proposes to bury in his patients". This is the tenor of this remarkable book. It puts under one cover a discussion of all the materials used by surgeons. This includes metals, plastics, suture material, newer anæsthetic gases, plaster of paris, hæmostatic agents, glove powders and others. There are chapters separately dealing with the problems peculiar to certain surgical specialties, such as abdominal surgery, neurosurgery, dental surgery, tendon repairs, thoracic surgery, vascular surgery and head and neck surgery. There are also sections dealing with the preservation of various tissues used in homografting, newer devices in anæsthesia and materials used for making various facial and limb prostheses. There is a final chapter on sterilization methods.

This book contains a wealth of facts and is enthusiastically recommended to the surgical scientist and craftsman who appreciates the need for knowing the virtues and limitations of the materials with which he works daily. The idea of gathering all this information under one cover is one for which the eidtor and publishers deserve much thanks.

UROLOGY IN OUTLINE. T. L. Chapman, Victoria Infirmary, Glasgow, Scotland. 176 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1959. $4.70.

Medical literature has of late been enriched by a number of "basic urology" books, which endeavour to simplify the specialty of urology for the general medical practitioner and medical student.

Mr. Chapman's effort is unique, in that its presentation is mainly pictorial—in fact, almost cartoon-like, with a minimum of reading material. One has the impression that the entire subject is over-simplified. He is inconsistent, in that many technically difficult surgical procedures, e.g., the pull-through operation for urethral stricture, are presented in picture form without printed explanation. This renders the procedure difficult or impossible for the novice reader to understand, and really has no place in an outline.

The author approaches carcinoma of the prostate in a pessimistic manner. He refers to the difference in attitude in American and British clinics towards radical treatment. More emphasis is placed on the palliative treatment of advanced carcinoma than on early recognition and extirpation.

The usual subjects are covered as adequately as the method allows, considering that the book is an outline only. It can be recommended mainly for its ease of reading—little text, and many illustrations.

CLINICAL OBSTETRICS AND GYNECOLOGY. Vol. 1, No. 4. Symposium on Operative Obstetrics. Edited by J. Robert Willson. Symposium on Genital Cancer. Edited by Daniel G. Morton. Pages 857 to 1138. Illust. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1958. $18.00 per volume.

The fourth number issued in December 1958 completes Volume I of the quarterly publication, *Clinical Obstetrics and Gynecology*. The editors, contributors and publisher are to be congratulated upon the quality of this first year's compilation.

The December number contains comprehensive symposia on two important aspects of this

(Continued on page 430)

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(Continued on page 428)

field, namely, operative obstetrics and genital cancer. In keeping with most current thinking, the contributors to "operative obstetrics" have presented a conservative and rational philosophy. Despite the need for brevity, descriptions of techniques and methods of management contain sufficient detail to be valuable. In addition to discussion of the mandatory topics such as breech delivery, forceps, Cæsarean section, etc., two sections of particular interest cover the management of dystocia due to the large or abnormal fetus and also the operations to preserve pregnancy.

Genital cancer has become such an extensive subspecialty of gynæcology that it is manifestly impossible to include, in such a brief presentation, more than a superficial view of the overall problem. However, while a few of the essayists have dealt with specific techniques, the majority have succeeded in fulfilling adequately what is probably a more important role for this type of publication; namely, they have conveyed to those readers who are not managing cases of genital cancer every day a broad view of currently available and acceptable diagnostic and therapeutic approaches to these diseases.

ELECTROLYTE CHANGES IN SURGERY. Kathleen E. Roberts, Parker Vanamee and J. William Poppell. 113 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1958. $5.00.

Several books dealing with electrolyte changes in surgery have appeared recently, but this one adheres only to those problems occurring in the postoperative period and therefore is particularly attractive to the surgeon who is interested only in this practical aspect of the problem.

The authors assume that the reader has some basic knowledge of biochemistry, but this should not frighten away the surgeon who is shy of chemical knowledge. Indeed so clear and didactic is the presentation that a bare minimum of scientific information suffices.

The treatment suggested is of a practical nature so that this text can be used as a manual in the treatment of the various disturbances. The book carries the lesson that the diagnosis and treatment of an electrolyte abnormality is not an end unto itself but forms the foundation upon which the diagnosis of the etiological disorder is built, and offers support to the sick patient while definitive treatment is undertaken.

TUMORS OF THE LUNGS AND MEDIASTINUM. B. M. Fried, Montefiore Hospital, New York, N.Y. 467 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1958. $13.50.

This volume on tumours of the lungs and mediastinum is devoted primarily to bronchogenic carcinoma. The entire aspect of the disease is covered, with extensive bibliographies at the end of each chapter. Considerable emphasis is placed on the problem of metastases from a primary bronchogenic carcinoma, and also secondary metastases occurring in the lung.

Uncommon malignant tumours and benign tumours of the lungs and pleura are discussed in separate chapters, and a section on tumours and cysts of the mediastinum is included. This book is both comprehensive and authoritative. It is written with care and thoroughness, and cannot fail to be of interest to the physician and surgeon.

Books Received

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

The Hand: Its Anatomy and Diseases. John J. Byrne, Boston University School of Medicine. 384 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1959. $11.50.

Chirurgie der Hand. Atlas der Operationstechnik (Surgery of the Hand. Atlas of Operative Techniques). Marc Iselin, with the assistance of L. Gosse, S. Boussard and D. Benoist, Nanterre, 325 pp. Illust. Georg Thieme Verlag, Stuttgart, West Germany; Intercontinental Medical Book Corporation, New York, 1959. DM 69.—

Les Plaies de la Main (Hand Injuries). R. Souquet and A.-R. Chancholle, Toulouse. 295 pp. Illust. G. Doin et Cie, Paris, 1959. 3.600 fr.

Orthopædics: Principles and Their Application. Samuel L. Turek, Northwestern University Medical School, Evanston, Ill. 906 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1959. $22.50.

Clinical Orthopædics No. 13: The Hand, Part I. Editor-in-Chief, Anthony F. De Palma. 393 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1959.

VA Prospectus Research in Aging. Veterans Administration Advisory Committee for the Problems of Aging, and others. 125 pp. Illust. Veterans Administration, Washington, D.C., 1959. $1.50.

Nouvelle Pratique Chirurgicale Illustrée Fascicule XIII (New Surgical Practice Illustrated, Fascicle XIII). Edited by Jean Quénu. 276 pp. Illust. G. Dion et Cie, Paris, 1959. 3.350 fr.

Bleeding Esophageal Varices in: Portal Hypertension. Hirsch Robert Liebowitz; with a section on Surgical Treatment in collaboration with Louis M. Rousselot, New York University College of Medicine. 986 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1959. $27.00.