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HISTORY OF CANADIAN SURGERY

B.-G. BOURGEOIS (1877-1943)

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Le docteur Bourgeois, chirurgien réputé de Montréal, a de tout temps été mieux connu sous ses initiales B.-G. que sous son prénom, Benjamin-Georges (Fig. 1).

Il est né en 1877 à Bécancourt, coquet village situé sur la rive droite du Saint-Laurent, face à la cité des Trois-Rivières.

Un biographie relate qu'il doit à une école anglaise les prémices de son instruction.[2] Le Collège Saint-Joseph des Trois-Rivières reçoit le jeune Bourgeois dès 1890. Il est un élève modèle, doué, discipliné, conscient du devoir à accomplir. Son talent lui vaut en rhétorique le prix du Prince de Galles; il termine ses études au premier rang de sa promotion et il obtient la médaille du lieutenant-gouverneur.

B.-G. Bourgeois opte alors pour la médecine; il quitte les Trois-Rivières où il a fait si belle figure pour la grande ville de Montréal. A l'Université, il se montre un étudiant attentif, soucieux d'apprendre, assidu aux cours, premier rendu aux cliniques. En 1902, il termine ses études de médecine; une fois encore il est en tête de liste et il récolte le prix Hingston.

Il sollicite alors et il obtient facilement un poste d'interne au vieil hôpital de l'avenue des Pins. "Durant deux ans, son urbanité, son esprit de charité, sa capacité de travail et l'amour de son métier, son dévouement, les soins dont il entourait ses malades, dont il poussait toujours les examens à fond, la recherche et la perfection avec lesquelles il accomplissait ses devoirs d'état lui conquirent d'emblée l'estime et la confiance de ses supérieurs." Ces marques d'appréciation sont du docteur Urgel Gariépy; elles sont consignées dans l'Union Médicale du Canada.[2]

La chirurgie l'intéresse déjà au plus haut point; il va sans dire qu'il est l'assistant le plus couru de la salle d'opération (Fig. 2). Il se fait une joie et un devoir d'offrir son concours intelligent et dévoué aux chirurgiens de l'époque: Sir William Hingston, Henri Merrill, Amédée Marien, Eugène Saint-Jacques et Donald Hingston.[1]

L'internat terminé, il fait, en 1904, le pélerinage traditionnel à Paris. Deux années durant, il suit les cours à la Faculté et à l'amphithéâtre d'Anatomie et les cliniques des maîtres en chirurgie, Tuffier, Hartmann et Terrier. La chirurgie ne l'éloigne pas des disciplines différentes; il se plaît à compléter ses notions de neurologie et de dermatologie. La technique de Calot l'intéresse; il assiste à toutes les cliniques, même quand, à la belle saison, elles se donnent à Berck-sur-Mer.

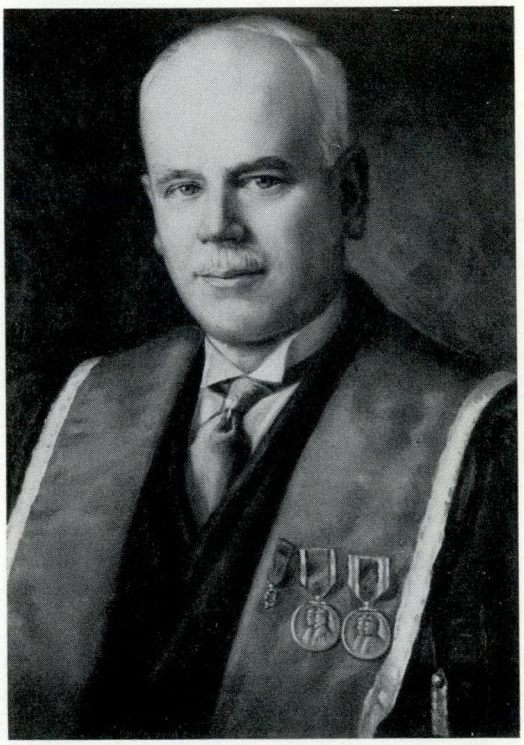

Fig. 1.—Le professeur Benjamin-Georges Bourgeois ancien vice-doyen et professeur de clinique chirurgicale à l'Université de Montréal. (Madeleine Delfosse, pinxit, 1939)

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Fig. 2.—Une salle d'opération de l'Hôtel-Dieu de Montréal en 1903, représentant le docteur Henri Merrill, chirurgien, ses assistants, des spectateurs et le docteur Benjamin-Georges Bourgeois alors interne, donnant l'anesthésie.

B.-G. Bourgeois s'imprègne à cette époque de la philosophie contemporaine qui accorde la préséance au pluralisme médical. Durant toute sa carrière, il demeure fidèle à ce concept et il se refuse toujours à croire qu'un chirurgien ne doive pas être simultanément urologue, orthopédiste, angiologue et réparateur. La spécialisation lui semble une diminution de l'acquit scientifique. Pour lui, tout chirurgien doit être un omnipraticien du bistouri susceptible de posséder la gamme entière du clavier technique. Cette thèse ne lui est pas particulière, car elle est soutenue dans maints grands centres européens et américains; elle marque toutefois l'orientation de la politique propre à B.-G. Bourgeois.

La vie parisienne fourmille au début du siècle d'occasions magnifiques de se cultiver et le choix est aisé entre mille facilités: arts, sciences, lettres, musique. B.-G. Bourgeois le comprend tout de suite; positif et ordonné, il assimile avec logique la somme des valeurs culturelles qui lui sont offertes.

De retour à Montréal, en 1906, il entre à l'Hôtel-Dieu où il est nommé assistant-chirurgien, quelques mois avant Pierre Zéphir Rhéaume et François L. de Martigny. Ironie du sort; aucun de ces trois chirurgiens ne fait carrière entière à l'Hôtel-Dieu. François de Martigny s'oriente vers l'Hôpital Français, le futur Ste-Jeanne d'Arc et Pierre Z. Rhéaume répond à l'appel de l'Hôpital Saint-Luc.

Le chirurgien en chef de Notre-Dame, le professeur Oscar Félix Mercier, à court d'hommes de première valeur, réussit en 1909, à convaincre B.-G. Bourgeois qu'un bel avenir l'attend au sein de son service. Et depuis la vie professionnelle de B.-G. Bourgeois se déroule sans interruption de 1909 à 1943 au service de cet hôpital. Il y occupe tour à tour toutes les fonctions, hospitalières, universitaires et administratives. Il gravit avec régularité tous les échelons qui conduisent au sommet de la hiérarchie. Sans les avoir recherchés, les honneurs viennent à lui les uns à la suite des autres.

Tous savent reconnaître ses qualités exceptionnelles de meneur d'hommes, sa sagacité, sa prudence, son jugement sain, son sens inné de l'administration compétente et l'extrême générosité qui fait qu'il se donne sans restriction à l'œuvre entreprise et qu'il ne refuse jamais sa collaboration à qui la demande. B.-G. Bourgeois est débordé de travail, mais il ne se dérobe jamais aux exigences des responsabilités qu'il a assummées de son plein gré.[2, 3, 6, 7]

Il devient chef du service de chirurgie à la mort du professeur Oscar Félix Mercier survenue à l'été 1929. Les charges qu'il y recueille absorbent toutes ses énergies; il les accepte, car le devoir lui en apparaît impérieux. La gamme d'obligations rigoureuses qui s'annoncent ne le fait pas reculer.

Lors de l'avènement en 1938 d'Albert LeSage à la tête de la Faculté de Médecine, on confie à B.-G. Bourgeois le poste de vice-doyen. Il connaît les rouages, car il est déjà professeur de chirurgie depuis vingt ans, mais cela ne minimise pas les efforts supplémentaires que la nouvelle tâche lui apporte.

A la même époque ou peu s'en faut, il est élu vice-président du Collège Royal des Médecins et Chirurgiens du Canada. Il y joue un rôle de premier plan en véritable ambassadeur de la pensée canadienne-française. Il seconde à merveille les efforts entrepris par Wilder Penfield pour obtenir aux francophones une audience attentive de leurs confrères de langue anglaise. L'un et l'autre les incitent à repenser les problèmes propres à leurs collègues des facultés françaises du Québec.[5]

B.-G. Bourgeois s'allie à Wilder Penfield pour résoudre au Collège Royal un problème épineux qui risque de compromettre gravement l'harmonie entre les deux communautés.

La saine raison leur inspire les mots qui convainquent et une formule acceptable pour tous naît de leur froide logique. L'association Penfield-Bourgeois, cette union de deux esprits droits, donne le ton de la discussion qui s'ensuit. La juste mesure conduit à l'élaboration d'une politique qui sourit aux deux parties. Ainsi, justice est rendue.

Le problème est simple, vu avec le recul du temps. Les difficultés portent uniquement sur la terminologie différente des échelons professoraux.

Penfield saisit les nuances de la nomenclature française et il souligne que la divergence n'est qu'apparente et superficielle. C'est la fin de ce qui aurait pu être une querelle durable et stérile.[5]

B.-G. Bourgeois se mêle à tous les groupements professionnels, auxquels il apporte l'appui de sa parole et le génie de sa pensée. Son nationalisme n'est pas obtus; au contraire, il l'incite à rechercher les occasions de porter le fait français au sein des organismes qui l'ignorent. Il recherche le contact étroit avec les associations chirurgicales canadiennes et américaines; il leur apparaît comme un porte-parole éloquent et persuasif des chirurgiens d'expression française. Bien plus, il organise à l'Université de Montréal et à l'Hôpital Notre-Dame de nombreuses séances scientifiques; il veut, en effet, démontrer à tous les médecins hors-du-Québec qu'il se fait du beau et du bon travail au Canada français; il n'est pas de ceux qui cachent leurs trésors sous le boisseau. Gariépy a écrit qu' "il prêche et préconise par la parole et par les actes l'union avec nos confrères anglais".[2, 6] Il y gagne l'amitié durable d'Edward Gallie dont l'œuvre accomplie à Toronto conquiert son admiration. L'estime est d'ailleurs réciproque.

Les efforts entrepris par l'Ecole Gallie pour la revalorisation à long terme de la chirurgie par la formation scientifique des jeunes chirurgiens lui apparaissent s'inspirer d'un idéal élevé. Il croit ferme à la nécessité d'une discipline rigoureuse et au respect parfait des règles de l'éthique professionnelle. Le "Gallie course" marque une étape notable dans l'histoire de l'éducation chirurgicale au Canada et c'est la voie que le professeur Bourgeois recommande sans réserve à ses meilleurs élèves.

B.-G. Bourgeois reste toutefois loyal à son allégeance française; il est d'ailleurs membre honoraire étranger de l'Académie de chirurgie de Paris et il se fait un devoir de recevoir princièrement les délégués que la France dirige à Montréal.

Le docteur Bourgeois défend ses opinions avec conviction; s'il admet la controverse, il trouve facilement les arguments-

choc pour emporter le morceau et gagner son point. Il ne craint jamais d'affirmer sa personnalité canadienne et française; il parle avec autorité et il n'affirme rien qu'il ne puisse prouver.[3, 7]

Son successeur à la tête du service de chirurgie de l'Hôpital Notre-Dame, le docteur Urgel Gariépy a écrit qu' "il était très conservateur en chirurgie et qu'il n'adoptait les modes opératoires ou les théories nouvelles qu'après l'épreuve du temps, à l'étranger et chez nous; tout était analysé à la lumière de son expérience et de sa science. Il n'eut jamais permis l'expérimentation sur les patients."[2]

Chirurgien classique, il n'a rien de l'opérateur éclatant qui recherche les gestes spectaculaires, mais il travaille avec méthode et sûreté. Il étend au maximum ses indications opératoires, mais formé à l'ancienne, il reste attaché aux techniques éprouvées, confirmées par le temps et il ne s'aventure pas au-delà. Il dévie rarement des normes reconnues; la cholécystostomie, pour ne citer qu'un exemple, lui paraît supérieure à la cholécystectomie; sa conviction est basée sur l'analyse des risques anesthésiques, sur la plus grande durée de l'agression chirurgicale, sur l'expérience de suites opératoires orageuses.

Professeur clair et systématique, il a un débit froid, mais il émaille ses dissertations théoriques de faits cliniques judicieusement choisis. Si le professeur est écouté, le chirurgien est un chef ferme et digne; il est respecté de ses troupes qui savent compter sur son appui en toutes occasions. Il est le maître incontesté dans son royaume de Notre-Dame. L'œil toujours aux aguets, l'attention constamment en éveil, nul n'arrive à prendre sa vigilance en défaut. Il connaît à fond tous ses assistants et il les aime comme des fils.[3]

B.-G. Bourgeois fait ses débuts de chirurgie au vieil hôpital de la rue Notre-Dame, voisin de l'ancienne Gare Viger. Il déploie donc ses énergies des premiers vingt ans dans un centre où le provisoire est devenu permanent, où les locaux sont incontestablement insuffisants. Il aurait été le chirurgien de garde chaque jour de 1908 à 1920, si l'on en croit son successeur, le docteur Urgel Gariépy.[2]

L'emménagement dans le nouveau Notre-Dame, face au Parc Lafontaine, lui apporte des facilités de travail, mais il augmente le fardeau de ses responsabilités. Télesphore Parizeau quitte à ce moment le service de chirurgie pour devenir directeur des études à la Faculté et, en 1933, doyen. La mort en 1929 de Mercier n'est pas non plus de nature à diminuer les charges du nouveau chef; il est débordé: clientèle privée nombreuse, séances opératoires longues, consultations astreignantes, enseignement clinique, participation active à tous les actes chirurgicaux des malades publics, présence assidue aux multiples comités universitaires, hospitaliers et administratifs. Heureusement O. A. Gagnon, Albert Demers, Eugène Dufresne, Léo Blagdon et Urgel Gariépy répondent à l'appel de Notre-Dame. L'aide arrive; mais elle ne suffit pas à la tâche; les nouveaux venus apportent la pensée du jour et des conceptions parfois divergentes, mais si B.-G. Bourgeois écoute, il ne se rend pas toujours.[2, 3, 7]

Homme convaincu de détenir la vérité, il a la vigueur du croisé. Excellent chirurgien, sa longue expérience lui vaut d'être chaque année invité aux congrès importants. Le dernier congrès, auquel il est appelé à parler, est celui de 1942 tenu aux Trois-Rivières. Il donne alors un remarquable travail basé sur son expérience de la conduite à tenir en face de l'ulcère digestif.[8]

Malgré les crises cardiaques qui le minent, le chef de Notre-Dame reste fidèle aux obligations de ses charges; il ne se résigne pas à ralentir ses activités; sans l'épreuve, inconsciemment redoutée, survenue à l'automne 1942, il n'aurait pas freiné l'élan qui l'emportait.

Le 11 décembre 1942, la dépêche, annonciatrice de malheur, lui apprend que son fils Jacques, le successeur qu'il s'était désigné, cet élève brillant d'Edward Gallie, devenu, depuis la guerre, un officier de marine qui parcourait les mers en service commandé, est porté disparu (Fig. 3).[4]

C'est l'aube de l'invasion nord-africaine par les forces alliées. Le port d'Oran, objectif du débarquement, est pris d'assaut; malheureusement l'effet de surprise rate, car les troupes défensives sont aux aguets

Fig. 3.—Le lieutenant-chirurgien Jacques de L. Bourgeois, fils du professeur B.-G. Bourgeois, porté disparu le 8 novembre 1942 lors de l'invasion par les alliés du Port d'Oran.

et dès le premier mouvement d'attaque, l'alerte est donnée.

Deux vaisseaux anglais, le H.M.S. Hartland et le H.M.S. Walney foncent à pleine vitesse sur l'embâcle dressé pour fermer la rade. Les obus défensifs pleuvent et sous les coups de la mitraille, les deux croiseurs donnent de la bande et rapidement coulent. Peu de survivants. Le lieutenant-chirurgien de marine Jacques de L. Bourgeois, en devoir à bord du H.M.S. Hartland est cloué à son poste de la salle d'opération. Il n'est plus revu.

B.-G. Bourgeois accueille avec stoïcisme la nouvelle de cette profonde et aussi rapide tragédie; il surmonte sa douleur et tente de faire bonne contenance, alors qu'il fait part du désastre aux membres de sa famille et qu'il leur exprime les mots d'espoir auxquels il ne croit plus.

"Il avait un tempérament d'une sensibilité extrême qu'il cachait sous un masque d'emprunt. C'est quand il était le plus ému qu'il se cuirassait davantage contre les extérieurs de cette émotion."[2]

Tout caractère, fut-il le mieux trempé, ne peut indéfiniment résister et se défendre contre les chocs répétés. Aussi, au matin du 28 janvier 1943, le fil de la vie est-il brisé. B.-G. Bourgeois n'est plus. Il a quitté ce monde discrètement, s'excusant presque de la peine immense qu'il cause à ceux qu'il aime et dont il se sait profondément chéri.

La Cathédrale de Montréal est, le 1er février 1943, le lieu des funérailles solennelles et émouvantes de B.-G. Bourgeois et de son fils Jacques.

Les professeurs de la Faculté de Médecine, revêtus de la toge universitaire ornée d'hermine, font une haie d'honneur et ils escortent dans l'allée centrale le cercueil de leur collègue. La tombe symbolique du lieutenant-chirurgien Jacques de L. Bourgeois est portée dans l'avant-chœur par des compagnons d'arme. Côte à côte, père et fils reposent sur de modestes socles entourés d'une garde d'honneur.[3]

Cette cérémonie touchante associe en un suprême hommage à Dieu les hauts faits de deux hommes qui ont donné leur vie, l'un au service de l'humanité souffrante, l'autre pour le Canada, sa patrie et pour la libération du monde asservi.

La figure de B.-G. Bourgeois mérite une place de choix dans la galerie des chirurgiens remarquables du Canada.

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ORIGINAL ARTICLES

THE NATURE OF THE VASODILATION WHICH FOLLOWS ARTERIAL GAS EMBOLIZATION*

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THE prolonged increase in blood flow which follows the passage of gas emboli through the arteries of a limb, was first described by Chase[4] in 1934 and by Lemaire *et al.*[29] in 1948. Investigations into the mechanism of the vasodilation have been reported by Judmaier,[16] Wernitz and Dorken,[28] Hasse, Köble and Linker,[15] Duff, Greenfield and Whelan,[6, 7] Marshall and Whelan,[21] Fries, Fries and Wesolowski[9] and Oppenheimer, Durant and Sherivin.[23]

The purpose of the present investigation was to determine the characteristics of the vasodilation which follows arterial gas embolization and to determine, if possible, its physiological mechanism.

METHOD

Sixty-three mongrel dogs weighing from 15 to 25 kg. were anesthetized with 35 mg./kg. of sodium pentobarbital. They breathed room air *via* an endotracheal tube. Clotting was controlled by 2 mg./kg. of heparin with an additional 1 mg./kg. every 90 minutes thereafter. The dogs rested on a constant-temperature blanket and their mid-esophageal temperature remained between 37° C. and 39° C. throughout the experiment.

Vascular resistance.—The left common femoral artery was cannulated and the blood led via a ¼ in. tube under screw-clamp control to an open reservoir containing 200 ml. of unmatched, fresh, homologous blood. The blood was then pumped by a roller pump (occlusive to 200 cm. water) through a heat exchanger maintained at 100° F. to a second cannula pointing distally in the common femoral artery. Collateral circulation to the leg was occluded by an extremely tight tourniquet of umbilical tape encircling the thigh at the level of the cannulas: the femoral artery and vein were the only structures not included in this tourniquet.

Arterial and venous pressures in both legs were measured *via* polyethylene catheters (P.E.160) inserted through side branches of the femoral artery and vein so that their tips lay within the lumen of the main vessel: the arterial catheter pointed upstream, the venous catheter pointed downstream. These pressures were transmitted *via* Statham strain gauges (P 23^{AC}, P 23^{BC}) to a four-channel Grass recorder with a paper speed of 0.25 mm./sec.

The pump speed was regulated until the perfusion pressure in the left leg was steady at approximately 150 mm. Hg. The flow rate, which varied between 50 ml. and 150 ml./min. in different dogs, was kept constant throughout each experiment. The changes occurring in the peripheral vascular resistance following the intra-arterial injection of a gas or drug were measured in three ways: (1) Maximal change in mm. Hg in arterial perfusion pressure; (2) Duration of the change in minutes; and (3) the "area of vasodilation". The area of vasodilation (the area encompassed by the fall in mean perfusion pressure until the base-line pressure was again achieved) measured in square centimetres with a planimeter, gave the most accurate overall measure of the change in vascular resistance.

Immediately before injection, the gases to be tested were drawn up in saline-sealed syringes. Injections were made over

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a two to three-second interval through a self-sealing rubber tube situated just proximal to the distal cannula. The following gases were tested: oxygen, carbon dioxide, nitrogen, and air. All injections consisted of 5 ml. of gas measured at room temperature and pressure.

The effect of the gas emboli on the "receptors" of the vascular system of the canine leg was determined by comparing areas of vasodilation obtained before and after the receptor had been specifically blocked. For example, in the investigation of the role of the beta-adrenergic receptor, areas of vasodilation resulting from: (a) 5 ml. of oxygen and (b) 2 μg./kg. of isoproterenol were obtained. The receptor was then blocked by 10 mg./kg. of dichloroisoproterenol, sufficient time (25 to 30 minutes) was allowed to elapse until the perfusion pressure had stabilized at or near the baseline pressure, and then a second set of areas of vasodilation from (a) 5 ml. of oxygen and (b) 2 μg./kg. of isoproterenol were obtained.

All gas and drug injections were given intra-arterially. The doses of the stimulator and blocker for the receptor (given below) were accepted as given in the literature. The drug solutions for injection were made up daily, using sterile pyrogen-free saline as diluent. Histamine phosphate was given in a dose of 1.7 μg./kg. in five dogs and was blocked by 5 mg./kg. of tripelennamine hydrochloride.[21, 26] A dose of 0.01 ml./kg. of 1:1000 dilution of l-epinephrine, was given in two dogs and was blocked by 10 mg./kg. of phenoxybenzamine (Dibenzyline).[12, 22] Acetylcholine was given in a dose of 5 μg./kg. in four dogs and was blocked by 0.065 mg./kg. of atropine sulfate.[11] Isoproterenol in a dose of 2 μg./kg. was given in five dogs and was blocked by 10 mg./kg. of dichloroisoproterenol (DCI).[19, 20] Serotonin was given in a dose of 5 μg./kg. in two dogs.[12, 13]

In an attempt to assess the effect of denervation, the leg of one dog was amputated at the thigh except for the femoral vein and the femur.

In order to assess the mechanical effect of gas emboli in a fluid system, a mock vascular system of arteries, arterioles, and capillaries was constructed of polyethylene, perfused with blood at a constant flow rate and the response of the arterial perfusion pressure to gas injection observed.

Flow studies.—In 10 dogs, the changes in flow following gas injection in the femoral artery of a leg perfused by the dog itself, rather than changes in the blood pressure of a perfused leg were studied. Femoral artery flow was measured by a square-wave electromagnetic flow meter* and a 3 mm. non-cannulating probe. Calibration curves were obtained with canine blood at 37° C. Zero flow was obtained by occluding the femoral artery proximal to the probe before and after each measurement. The gases and chemicals were injected *via* a small branch of the femoral artery just distal to the probe. The femoral arterial and venous pressures were recorded in a manner identical to those described above. A tourniquet was again used to isolate the leg.

Simultaneous gas injections into the pump-perfused left leg and the flow-monitored right leg were made in five dogs. Studies of the magnitude of reactive hyperemia secondary to mechanical occlusion of the flow-monitored femoral artery for periods of 0.25 seconds to five minutes were performed in another five dogs.

Tissue-oxygen tension.—Oxygen tension in the perfused leg was monitored by a Beckman oxygen micro-electrode and a Beckman recorder.† The electrode was calibrated by immersion first in "Dee-O" solution in 5% dextrose and water at 38° C., and then in water at 38° C. through which air was gently bubbled. Oxygen tensions were then recorded in the popliteal artery, the femoral vein, and the muscles and subcutaneous tissue of the calf.

Microcirculation.—The behaviour of gaseous emboli in small blood vessels was assessed by microscopic observation of the mesenteric vessels in 10 rabbits. The gases, (oxygen, carbon dioxide, nitrous oxide, and air) were injected in amounts of 2 ml. *via*

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*Square-Wave Electromagnetic Flow Meter, Catolina Medical Electronics Inc., Winston-Salem, North Carolina, U.S.A.

†Beckman Physiologic Gas Analyser and Oxygen Electrode, Beckman Instruments, Inc., South Pasedena, California, U.S.A.

a small catheter situated in the aorta near the origin of the superior mesenteric artery; the mesenteric vessels were then observed under quartz-rod illumination. Moving pictures at 16 frames per second were taken to provide a more accurate record of the changes observed.

Results

(A) The Effect of an Intra-Arterial Injection of 5 ml. of Gas on Vascular Resistance

The pattern of the response to oxygen.—In the dog's hind leg perfused at a constant arterial flow, the gas emboli produced a biphasic change in femoral arterial pressure (Fig. 1). With a constant arterial blood flow and an unchanged venous pressure, the changes in arterial pressure were indicative of changes in vascular resistance. The initial phase, one of increased arterial pressure (increased vascular resistance) lasted for one to two minutes. It was followed by a phase of decreased arterial pressure (decreased vascular resistance) lasting for 11 to 20 minutes. In the second phase, the arterial pressure fell rapidly in a smooth curve until it reached approximately two-thirds of the base-line pressure. It then rose slowly to its original level. The area between the base-line and the curve below it was measured in square centimetres and recorded as the area of vasodilation. In Fig. 1, the response of the arterial pressure of the perfused leg to 5 ml. of intra-arterial oxygen is compared to the response to 25 mg. of intra-arterial tolazoline, and to two minutes of femoral arterial occlusion.

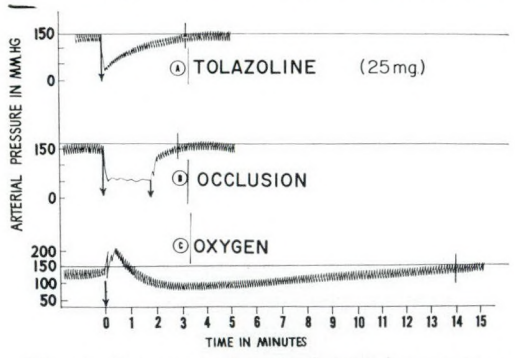

Fig. 1.—Response of arterial perfusion pressure to tolazoline, zero flow and gaseous oxygen. Isolated hind limb of a 15 kg. dog perfused at 100 ml./min. to: (1) 25 mg. of intra-arterial tolazoline, (2) two minutes of zero flow, and (3) 5 ml. of intra-arterial oxygen.

As shown in Table I, the magnitude of the vasodilation which followed the injection of 5 ml. of oxygen into the femoral artery of 19 different dogs, was relatively constant. The arterial pressure decreased to 67% of normal (SD 5.7) and returned to normal in 15 minutes (SD 2.3). An area of vasodilation of 15.2 sq. cm. (SD 3.0) was produced.

TABLE I.—Response to 5 ml. of Oxygen Injected into the Perfused Femoral Artery of 19 Dogs

	Base-line pressure (mm.Hg)	*Fall in pressure (mm.Hg)*	*% fall in pressure*	*Duration of response (min.)*	*Area of dilation (sq. cm.)*
Mean....	155.3	15.3	33.1	15.4	15.2
SD.....	10.7	9.3	5.7	2.3	3.0

Tachyphylaxis did not occur with repeated doses of 5 ml. of oxygen in the perfused canine leg. The results of eight separate injections in one dog are shown in Table II.

TABLE II.—Injections of 5 ml. Oxygen into the Perfused Femoral Artery of One Dog (17 kg.).

	Base-line pressure (mm.Hg)	*Fall in pressure (mm.Hg)*	*% fall in pressure*	*Duration of response (min.)*	*Area of dilation (sq. cm.)*
1	165	40	24	11	9.9
2	155	45	29	12	9.8
3	150	40	27	13	10.6
4	175	50	29	14	12.4
5	175	45	26	13	10.1
6	175	50	27	12	9.6
7	165	45	27	14	11.6
8	190	25	13	12	8.1

Response to eight injections of 5 ml. of oxygen at 20-minute intervals into the femoral artery of a 17 kg. dog perfused at a constant flow of 110 ml./min.

The "optimal" dose of gas.—Dose-response curves obtained from oxygen injections into the perfused legs of two dogs (15 kg. and 21 kg.) are shown in Fig. 2.

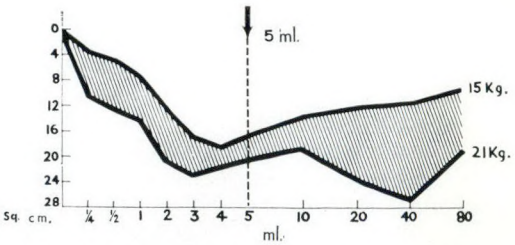

Fig. 2.—Dose-response curve to oxygen. The areas of vasodilation in sq. cm, resulting from injections of 0.25 ml. to 80 ml. of oxygen into the perfused femoral arteries of the isolated limbs of two dogs (15 kg. and 21 kg.).

The area of vasodilation increased with increasing doses up to 3 to 5 ml. and then became unpredictable or decreased. With larger volumes of gas, the femoral vein contained small gas bubbles and the animal's systemic pressure dropped as pulmonary gas embolization presumably occurred. No change in systemic arterial pressure or heart rate was noted with femoral intra-arterial injections of 5 ml. of oxygen.

Comparison of various gases.—The effect of 5 ml. doses of carbon dioxide, nitrogen, and air was also tested. With each gas, the first phase of increased arterial pressure was similar in duration and magnitude to that seen with oxygen, while the second phase of decreased arterial pressure was different. In the second phase, the arterial perfusion pressure always decreased to approximately 2/3 of the control value, but the duration of the response was variable. The mean duration of vasodilation following carbon-dioxide emboli was seven minutes; oxygen, 15 minutes; air, 18 minutes; and nitrogen, 21 minutes (Table III). The longer duration of vasodilation was also reflected in the area of vasodilation: carbon dioxide, 5.22 sq. cm.; oxygen, 15.2 sq. cm.; air, 19.5 sq. cm.; and nitrogen, 19.3 sq. cm.

Similarly, when gas emboli were observed in the microcirculation of the rabbit mesentery, they disappeared from the small vessels at varying rates. Complete clearance of 2 ml. of nitrous oxide occurred in one to two minutes, carbon dioxide in two to seven minutes, oxygen in 10 to 15 minutes, and air in 20 to 25 minutes.

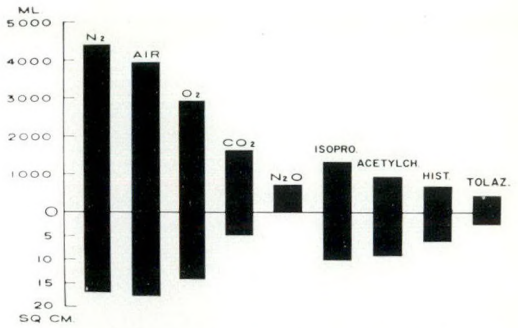

Fig. 3.—Comparison of gas-induced vasodilation to that resulting from several drugs. In the top half of the figure are depicted the total increases over baseline flow in the self-perfused flow-monitored leg. In the bottom half of the figure, are depicted the areas of vasodilation in the pump-perfused leg. All gases and drugs were injected intra-arterially. Doses are given in text.

Comparison of vasodilation due to gas emboli to that following standard chemical vasodilating agents.—The magnitude of vasodilation resulting from gas emboli in both flow-variable (perfused by the dog itself and pressure-variable (pump-perfused) systems was compared to that resulting from several compounds commonly used to produce vasodilation (Fig. 3). The increased flow in the flow-variable system and the area of vasodilation in the pressure-

TABLE III.—Vascular Response in the Hind Limb of the Dog Following Intra-Arterial Insufflation of 5 ml. of Various Gases.

Gas	*Base-line pressure (mm.Hg)*		*Fall in pressure (mm.Hg)*		*% Fall in pressure*		*Duration of response (min.)*		*Area of dilation (cm.²)*	
5 c.c.	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
Nitrogen* (6 injections in 3 dogs)	173	17.6	48	18.9	29	14	21	1.0	19.3	4.6
Air (12 injections in 6 dogs)	164	10.2	57	16.3	35	11	18	3.6	19.5	6.9
Oxygen* (19 injections in 19 dogs)	155	10.7	51	9.3	33	5.7	15	2.3	15.2	3.0
Carbon dioxide* (10 injections in 5 dogs)	167	13.0	41	9.6	25	8.1	7.2	1.1	5.22	1.1

*Absorption coefficients of nitrogen, oxygen and carbon dioxide at 35° C. are 0.01256, 0.02440 and 0.592.[17]

variable system following the injection of gas emboli was larger than that resulting from a single injection of a standard dose of such drugs. The major difference between the response to gas and to vasodilating solutions was the long duration of the vasodilation which followed gas injections.

B. The Cause of the Response

The short initial phase of increased arterial pressure was of the same duration and magnitude irrespective of the gas injected into the perfused leg. Its characteristics were not changed by amputation of the leg or blockade of the alpha-adrenergic receptors by 10 mg./kg. of intra-arterial dibenzyline. A similar phase, although of shorter duration, occurred when gas was injected into a perfused circulation model of polyethylene tubes. The initial phase of increased resistance would seem to result from a blockage of small vessels with gas "slugs", and to be unrelated to increased vascular tone or arterial spasm.

There seemed to be several possible mechanisms by which the second phase of decreased vascular resistance may have resulted from the passage of the gas emboli:

1. Reduction of sympathetic tone.
2. Reactive hyperemia.
3. Mechanical dilation.
4. Stimulation of vascular receptors.

1. Reduction of sympathetic tone.—It has been reported that sympathectomy does not affect the response to gas emboli.[7] In order to ensure complete sympathetic denervation, the leg of one dog was severed at the thigh leaving intact only the femur and the femoral vein. This leg was then perfused in the standard fashion. The responses to four injections of 5 ml. of oxygen in this leg are shown in Table IV.

TABLE IV.—A Comparison of the Response to 5 ml. of Oxygen Injected into the Intact Perfused Hind Limb (19 Injections in 19 Dogs) and the Amputated Perfused Hind Limb (4 Injections in One 20-kg. Dog.)

	Intact hind limb		*Denervated hind limb*	
	Mean	*SD*	*Mean*	*SD*
Base-line pressure mm.Hg	155.0	10.7	163.0	5.8
Fall in pressure mm.Hg	51.0	9.3	51.7	2.9
% decrease in pressure	33.1	5.7	31.3	2.5
Duration of response (min.)	15.4	2.3	19.7	.58
Area of dilation	15.2	3.0	18.7	.58

There was no significant difference between the magnitude, the duration, or the area of vasodilation in the denervated leg as compared to the standard response.

2. Reactive hyperemia.—It seemed probable that the initial phase of decreased flow and increased vascular resistance from embolic blockage of small vessels would be a stimulus to reactive hyperemia. An estimate of the proportion of the increased flow which was contributed by reactive hyperemia was obtained by occluding the dog-perfused, flow-monitored femoral artery for periods of 15 seconds to five minutes, and then observing the extent to which the calculated "flow debt" was "repaid". "Flow debt" was calculated by multiplying base-line flow by the duration of occlusion. The maximum extent to which the "flow debt" was repaid by reactive hyperemia was only 14%; this occurred following 25 seconds of occlusion. In contrast, the "flow debt" incurred during the first phase of the response to gas emboli was always "repaid" 15 to 20 times during the second phase. It seemed that reactive hyperemia played only a small part in the phase of decreased vascular resistance which occurred following arterial gas emboli.

3. Mechanical dilation.—When the rabbit mesentery was observed during gas embolization, small "slugs" of gas were seen to arrive in and plug small arteries and arterioles. As they passed through these vessels there was a widening of the vessel at the site of the embolus and proximally to it, while distally the vessel was narrowed. Two explanations of the increased vessel-width seemed possible: (1) The gas, expanding because of the decreased pressure in the lumen of these small vessels and from its slow warming to body temperature, dilated the vessel; (2) The decreased velocity of flow behind the gas embolus caused increased lateral pressure on the vessel wall.

The arteriole distal to the gas "slug" was narrowed and many previously visible capillary beds emptied and became invisible. A decrease in tone may have occurred in the muscle of the wall of the collapsed non-perfused vessels, allowing increased flow after passage of the obstructing slug as suggested by Folkow.[10] From the re-

ported observations, it would seem that the mechanical effect of the passing gas embolus may have played some part in the production of the phase of decreased vascular resistance.

4. Stimulation of vascular smooth-muscle receptors.—As the vasodilation following gas emboli did not seem to be adequately explained by either reactive hyperemia or mechanical dilation alone or in combination, the role of the various vascular "receptors" was investigated.[1, 14, 18, 20]

(a) Histaminergic "receptors".—In five dogs, areas of vasodilation in response to 1.7 μg./kg. of histamine phosphate and to 5 ml. of oxygen were measured before and after blockade of the histaminergic "receptors" with 5 mg./kg. of tripelennamine HCl. The antihistamine reduced the vasodilation response to histamine by 60% ($P < .001$), but did not significantly affect the response to 5 ml. of oxygen (Figs. 4 and 5). Thus the gas emboli did not cause vasodilation by a mechanism inhibited by the antihistamine tripelennamine hydrochloride.

(b) Cholinergic "receptors".—In four observations in four dogs, areas of vasodilation in response to 5 μg./kg. of acetylcholine and to 5 ml. of oxygen were measured before and after blockade of the cholinergic "receptors" with 0.065 mg./kg. of atropine sulfate. Atropine sulfate reduced the vasodilation response to acetylcholine by 71% ($P < .001$) but did not significantly affect the response to 5 ml. of oxygen (Figs. 4 and 5). Thus the gas emboli did not produce vasodilation through a mechanism inactivated by atropine sulfate.

(c) Serotonin release.—Serotonin may produce an increase or decrease in vascular tone in perfused canine hind limb depending on the initial resistance.[13] In our preparation, 5 μg./kg. of serotonin injected intra-arterially, produced an increased vascular resistance. Thus serotonin release cannot explain the vasodilation response after gas emboli.

(d) The "alpha-adrenergic receptor".—As stated above, 10 mg./kg. of intra-arterial dibenzyline did not significantly change the initial phase of increased vascular resistance following oxygen injection. A normal vasoconstrictive response to epinephrine was observed in five experiments in which 0.01 ml./kg. of 1:1000 dilution of epinephrine was injected at the peak of the phase of gas-induced vasodilation. Stimulation or blockade of "alpha-adrenergic receptors" did not seem to be involved in the response to gas emboli.

(e) The "beta-adrenergic receptor".—In five observations in five dogs, blockade of the "beta-adrenergic receptors" with 10 mg./kg. of dichloroisoproterenol (DCI) caused a 92% reduction in the vasodilation response to 2 μg./kg. of isoproterenol ($P < .001$). DCI also caused a 42% reduction in the vasodilation response to 5 c.c. of oxygen ($P < .001$) (Table V and Figs. 4 and 5).

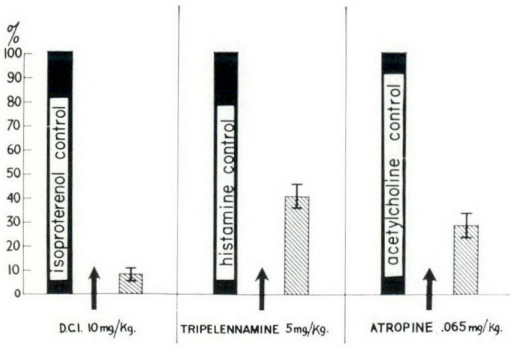

Fig. 4.—Reduction of drug-induced vasodilation. The percentage reduction in the area of vasodilation which follows the intra-arterial injection of isoproterenol (when blocked by DCI), histamine (when blocked by tripelennamine), and acetylcholine (when blocked by atropine). Standard deviations are shown.

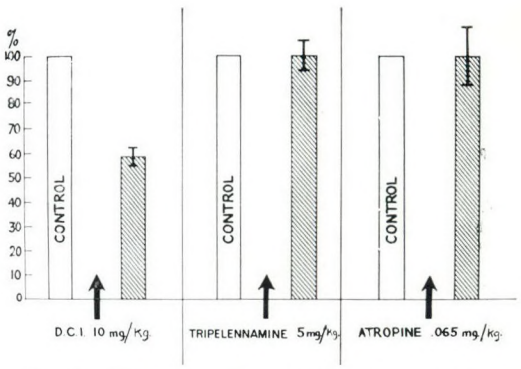

Fig. 5.—Reduction of gas-induced vasodilation. The percentage reduction in the area of vasodilation which follows the intra-arterial injection of 5 ml. of oxygen after blockade with DCI, tripelennamine, and atropine. Standard deviations as shown.

TABLE V.—Data from the Five Experiments in which 10 mg./kg. of DCI was Employed to Affect the Vasodilation Response to 2 μg.kg of Isoproterenol and 5 ml. of Oxygen

		Isoproterenol					*Oxygen*			
		Base-line pressure mm.Hg	*Fall in pressure mm.Hg*	*Duration in minutes*	*Area cm.²*		*Base-line pressure mm.Hg*	*Fall in pressure mm.Hg*	*Duration in minutes*	*Area cm.²*
Dog 4	A*	130	85	20	24.2	A	125	40	13	8.7
Wt. 16 kg.	B*	120	50	1.5	1.0	B	120	25	12	5.5
Dog 6	A	170	85	3	4.2	A	155	50	17	18.4
Wt. 13 kg.	B	150	15	40 sec.	0.3	B	150	45	11	9.9
Dog 8	A	145	90	23	19.1	A	150	60	17	17.7
Wt. 23 kg.	B	175	55	5	3.0	B	170	50	12	11.5
Dog 10	A	160	70	7	8.6	A	160	30	11	8.4
Wt. 17 kg.	B	160	30	1.0	1.1	B	155	25	8	4.8
Dog 11	A	170	90	8	7.5	A	170	60	16	18.1
Wt. 18 kg.	B	175	15	40 sec.	0.8	B	165	40	10	9.2

Mean % decrease in area of 90% (P <.001) SD—4.5 cm.²; SE of mean—2.0 cm.²

Mean % decrease in area—42% (P <.001) SD—6.0 cm.²; SE of mean—2.7 cm.²

*A—Before injection of DCI. *B—After injection of DCI.

Fig. 6 shows dose-response curves of gas-induced (5 ml. oxygen) and drug-induced (2 μg./kg. of isoproterenol) vasodilation to blockade by DCI in doses from 1 to 64 mg./kg. Ten mg./kg. was the most effective dose of DCI for blocking gas-induced vasodilation in the hind limb of dogs. Vasodilation which occurred after arterial gas emboli appeared to be referable, in part, to stimulation of receptors that could be blocked by DCI.

Injection of 2 μg./kg. of isoproterenol at the maximum point of gas vasodilation caused further vasodilation of slight degree. The random distribution of the gas emboli may have stimulated only part of the "beta receptors" potentially susceptible, whereas an injected chemical solution reached all these receptors.

C. The Course of the Emboli

Experiments of Burns, Robson and Smith,[3] Sabiston *et al.*[25] and Talbert *et al.*,[27] which have shown that an organ such as the heart can continue to function for several hours when the fluid in the coronary tree has been totally replaced with a moist gaseous mixture of 95% oxygen and 5% carbon dioxide, suggest that a gas can perfuse the capillary bed. In extensive observations of the microcirculation of the rabbit mesentery, we were never able to see a gas embolus actually pass through the capillary bed. The small sausage-shaped emboli blocked the small arteries, arterioles and metarterioles. They gradually grew smaller and smaller and then either disappeared completely or else passed rapidly through arteriovenous communications into the veins. Previously invisible capillary beds then opened and a marked hyperemia of the total area occurred. Others have reported a similar sequence of events.[28]

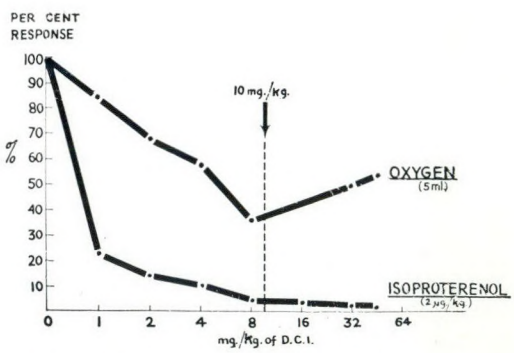

Fig. 6.—Dose-response curve to DCI. Reduction in the areas of vasodilation which follow 2 μg./kg. of isoproterenol and 5 ml. of oxygen when DCI was given in doses from 1 to 64 mg./kg.

When a polarographic oxygen electrode was inserted into an artery or vein, there was always a marked rise in recorded oxygen tension when a bubble of oxygen passed. The intra-arterial pO_2 often exceeded 300 mm. Hg. Measurements of pO_2 in calf muscle and subcutaneous tissue, however, were variable. There was an occasional rise in muscle and subcutaneous tissue pO_2 during the first five to 10 minutes while the oxygen bubbles were still in the leg but frequently tissue pO_2 fell during this period and then rose to normal levels with the phase of vasodilation. With carbon-dioxide emboli there was always a fall in tissue pO_2 in the first five to 10 minutes. The results seemed compatible with a random distribution of gas emboli producing a blockade of small arterioles and arteries and suggested that there was not a generalized perfusion of the capillary beds of the leg.

Discussion

There is much that is unknown about both the behaviour and fate of small gas bubbles in the vascular tree, and also about the response of the blood vessels to the presence of such gas bubbles. When the vascular system was completely filled with a gas, as in the experiments of Sabiston *et al.*,[25] Talbert *et al.*,[27] and Burns, Robson and Smith,[3] the continued function of the organ suggested that the capillaries were perfused. When a small volume of gas was injected into the mesenteric artery of the rabbit in our experimnts, the capillary bed was not perfused, but instead the gas bubbles acted as temporary emboli and blocked the small arteries, arterioles, and metarterioles. The bubbles never became small enough to perfuse the capillary bed.

The size of the gas bubble will be affected by its temperature and the intraluminal pressure of the vessel it inhabits. The rate at which its volume shrinks will also be determined by its solubility and diffusability. The gas emboli resulting from an injection of N_2O disappeared in two minutes while those from nitrogen were visible for 20 to 25 minutes.

Eiseman, Baxter and Prachuabmoh[8] have stated that the resistance to blood flow caused by a gas embolus will be determined by whether it is moving or stationary. They observed no increased resistance while the bubble was moving, but once it was stationary and had created a "slug flow system," resistance rose rapidly. The creation of "slug flow systems" from complete blocking of some vessels would explain the first phase (the phase of increased vascular resistance) seen after the injection of 5 ml. of gas into the femoral artery. Denervation of the leg or blockade of "alpha-adrenergic receptors" with dibenzyline did not affect this phase of the response to gas emboli.

The second phase (the phase of decreased vascular resistance) was less easily explained. From the observations reported in this article, several deductions were possible. The magnitude of the vasodilation was the same in the amputated as in the intact extremity, so it was unlikely that a reduction of sympathetic tone had occurred. The vasodilation was not blocked by tripelennamine hydrochloride, so it was unlikely that histamine release by irritated vascular endothelium was involved. The vasodilation was not blocked by atropine sulfate so it was unlikely that the emboli activated a cholinergic mechanism of vasodilation. In the experimental preparation, serotonin acted as a vasoconstrictor and so did not seem to be involved. Adrenaline still acted as a potent vasocontricting agent at the height of gas-induced vasodilation so it was unlikely that there was any significant blockade of "alpha-adrenergic receptors".

It seemed likely that three phenomena were involved in the production of the phase of decreased vascular resistance which followed the injection of a gas. (1) The tissue ischemia present during the initial phase of increased resistance would result in reactive hyperemia due to the accumulation of acid metabolites and changes in tone of collapsed blood vessels distal to the embolus. (2) The tendency of a gas bubble to expand as it reached the lumens of vessels with lower pressure, and as it warmed from room temperature to body temperature may have mechanically dilated the vessel. (3) The finding of a 42% decrease in the magnitude of the vasodilation following blockade of the "beta-adrenergic receptors" with DCI suggests that a large

proportion of the response was dependent upon activation of this mechanism of vasodilation.

Furchgott[12] and Dresel[5] have recently shown that DCI in large doses will act as a sympathomimetic agent and produce vasoconstriction. The arterial perfusion pressure and the venous pressure and consequently the calculated vascular resistance in the pump-perfused leg were the same before the control oxygen injection, and before the oxygen injection given 30 to 40 minutes after the intra-arterial administration of 10 mg./kg. of DCI. It is unlikely, therefore, that the vasoconstrictive properties of this drug could account for the observed decrease in the vasodilation following arterial gas embolization.

Summary

When 5 ml. of gaseous oxygen was injected into the femoral artery of the dog, there was a biphasic response consisting of a short period of increased vascular resistance followed by a prolonged period of decreased vascular resistance. The phenomenon of decreased vascular resistance after gas embolism was investigated with constant-flow perfusion, measurement of femoral-artery flow with the electromagnetic flow-meter, measurement of tissue-oxygen tension, and observation of the behaviour of gas emboli in the rabbit mesentery. The vasodilation was of greater magnitude than could be explained by reactive hyperemia. It was not affected by an antihistaminic (tripelennamine hydrochloride) or atropine sulfate, but was decreased 42% by a blockade of "beta-adrenergic receptors" with dichloroisoproterenol. The gas emboli did not perfuse the capillary bed.

Intra-arterial gas embolism produces peripheral vasodilation. Stimulation of the "beta-adrenergic receptors" of the vascular musculature would appear to account for a portion of this reaction.

We wish to thank Professor D. B. W. Reid and Miss B. Clarkson for their assistance in the statistical analysis of the results.

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Résumé

L'embolie gazeuse produit dans un membre une augmentation marquée et prolongée du débit sanguin. Les auteurs ont voulu, dans leur processus expérimental, décrire les caractères de cette vasodilatation et en expliquer le mécanisme physiologique. Par des injections de gaz (oxygène, azote, gaz carbonique, air) intra-artérielles, ils ont étudié la réaction à l'embolie gazeuse de la résistance vasculaire périphérique, du débit sanguin, de la tension tissulaire d'oxygène et de la micro-circulation. Ils ont également étudié la réponse après blocage des récepteurs vasculaires adrénergiques.

L'embolie gazeuse provoque au niveau de la résistance vasculaire périphérique une double réaction. Elle produit d'abord une résistance accrue qui se manifeste par une courte période d'hypertension vasculaire d'une durée de une à deux minutes, suivie d'une période de résistance diminuée, traduite par une hypo-tension et qui dure de 11 à 20 minutes. L'explication physiologique de la seconde phase n'est pas facile à déterminer. Il semble qu'elle soit consécutive à plusieurs phénomènes. L'ischémie tissulaire de la première phase semble provoquer une hyperhémie réactionnelle due à l'accumulation de métabolites acides. Elle serait également en relation avec l'activation du mécanisme de vaso-dilatation que les auteurs ont pu mettre en évidence après blocage des récepteurs adrénergiques avec le dichloroisoprotérénol.

SELECTIVE CELIAC AND SUPERIOR MESENTERIC ARTERIOGRAPHY*

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Catheterization of the celiac and superior mesenteric arteries for the purpose of selective arteriography was first reported in 1951 and a practical method was descibed in 1956. A "selective" arteriogram provides better vascular detail than is possible with aortography because of the higher concentration of contrast medium in the arteries and because of the absence of overlying vessels. Selective arteriography also provides a method for detecting collateral flow from one major artery system to another. For the past 2½ years we have been using this technique as part of the clinical investigation of patients and our findings are the subject of this report. A particular interest was the arterial system of the pancreas because of the inaccessibility of this organ to the usual diagnostic methods.

History

In 1951 Bierman *et al.*[1] reported the use of a cardiac catheter introduced by carotid or brachial arteriotomy to catheterize selectively the celiac or super mesenteric artery. In 1952 Rappaport[2] described a technique for selective catheterization of the celiac artery, using a femoral arteriotomy. In 1956 Odman[3, 4] described a preformed catheter which could be introduced percutaneously after the method of Seldinger *via* a femoral artery for selective arteriography of the celiac artery. He described in detail the anatomy of the celiac arteriogram and in 1959 he described the technique and ana-

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tomy of selective arteriography of the superior mesenteric artery.[5,6] Odman's method has proven to be satisfactory and forms the basis of all subsequent reports including the present one. In 1965 Baum *et al.*[7] reported on the value of selective arteriography in diagnosing tumours of the liver and pancreas. In 1965 Paul *et al.*[8] and Rosch and Bret[9] reported its value particularly in the diagnosis of pancreatic disease.

Selection of Patients

The patients studied were 30 to 79 years of age. Each was examined because of signs or symptoms suggesting disease of the pancreas or adjacent organs. Fifty-seven examinations were attempted and 53 were completed, the others being unsuccessful owing to extreme tortuosity of the arteries or aorta, or to technical breakdowns.

Before the examination each patient was questioned and examined with particular reference to allergy, cardiovascular or renal disease, or bleeding tendencies, and laboratory tests were obtained as indicated. A history of allergy was considered to warrant the use of premedication with an antihistaminic agent, but apart from mild urticaria there were no allergic reactions. Arteriography was performed on patients with varying degrees of renal failure without aggravating their condition, and renal insufficiency was not considered a contraindication. Other relative contra-indications such as angina pectoris were weighed against the necessity of the diagnostic procedure. Sedation was usually by morphine, 1/6 grain, given intramuscularly one hour before the examination.

Method

A 70 cm.-long green radiopaque Kifa catheter with a 3 cm.-diameter semicircular curve of the end was used. The tip was tapered to fit a No. 160 spring guide-wire and a side hole served to minimize recoil during injection. The catheters were sterilized chemically and discarded after use.

Seldinger's technique was used with the usual sterile precautions to introduce the catheter *via* a femoral artery into the aorta. With the curve of the catheter temporarily eliminated by the spring guide-wire in its lumen, the tip was passed to the level of the 12th thoracic vertebral body under fluoroscopic control. The guide was then withdrawn allowing the catheter to recover its curvature and the tip was manipulated into the celiac artery. Occasionally, severe atherosclerosis of the iliac or femoral arteries made retrograde catheterization impossible and in these cases an appropriately shaped catheter was introduced *via* the axillary artery and guided into position by fluoroscopy. The position of the tip was in any event confirmed by observing fluoroscopically a 10 c.c.-test injection of contrast medium and the patient was then moved to the Schoenander film changer. For arteriography between 25 and 35 c.c. of diatrizoate methylglucamine (Renovist) were injected in two seconds, radiographic films were exposed for the first three seconds at the rate of two per second and for the next 18 seconds at the rate of one every six seconds. The catheter was then repositioned for selective injection of the superior mesenteric artery. A heparin solution was used for periodic flushing of the catheter between injections. Where it was felt to be desirable, selective catheterization was preceded by aortography.

After completion of the examination and removal of the catheter, the puncture site was compressed manually until there was no further evidence of bleeding, care being taken not to obliterate the pulse for a long period. The patient was instructed to remain flat in bed for the remainder of the day, and the blood pressure and pulse were observed for six hours.

Complications

The most serious complications reported have been occlusion of the artery at the puncture site and perforation of an iliac artery by the catheter. Other complications which have been reported include dissecting aneurysm of the arterial wall, subintimal injection of contrast medium and false aneurysm formation at the puncture site.[10] An awareness of these possibilities together with gentleness in the manipulation of the catheter and guide wires, the use of heparin, and careful fluoroscopic control at all times will prevent most com-

plications.

No complications occurred in this series of examinations, nor have we encountered any serious complications in over 400 other recent catheter aortograms and arteriograms.

Anatomy

The celiac artery normally gives rise to the splenic and left gastric arteries and the common hepatic artery, which in turn gives rise to the gastroduodenal artery. The pancreaticoduodenal arcades to the head of the pancreas arise from the gastroduodenal artery, the posterior arcade arising proximally and usually lying medially, and the anterior arcade arising more distally and lying more laterally, as seen in the anteroposterior (AP) arteriogram. The body of the pancreas receives branches from any of the adjacent major vessels usually by way of the dorsal pancreatic artery. The tail is usually supplied by small branches from the splenic artery and sometimes by the large pancreatica magna artery. The major pancreatic arteries are about 1 mm. in diameter and the clarity with which they are visualized is a convenient measure of the quality of the arteriogram (Fig. 1).

From the superior mesenteric artery, jejunal and ilial branches fan out to the left before the main artery continues to the ileocecal region. Other branches are one or several right colic arteries and a transverse colic artery which can be seen crossing the abdomen with the transverse portion of the colon. Proximally the inferior pancreaticoduodenal artery ascends to join the anterior and posterior arterial arcades of the head of the pancreas and the dorsal pancreatic artery may pass to the body of the pancreas. The pancreatic arcades may be opacified either from the celiac or superior mesenteric artery, a feature which assists in their identification and also demonstrates the value of this route as a potential anastomosis between the two major arterial systems (Fig. 2).

Anatomical variations of the major vessels occurred in 10 of our patients usually affecting the site of origin of an artery, the commonest being a hepatic artery arising from the superior mesenteric artery. Innumerable variations occurred in the minor vessels.[11]

Pancreatic Disease

Five patients with chronic relapsing pancreatitis were examined. In three the ar-

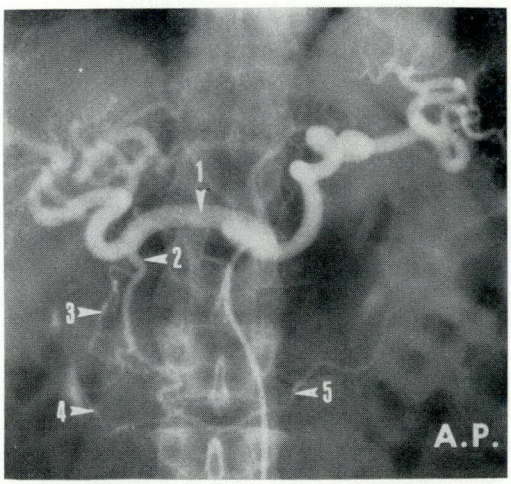

Fig. 1.—Selective celiac arteriogram: (1) hepatic artery, (2) gastroduodenal artery, (3) retroduodenal artery and posterior arterial arcade of the head of the pancreas, (4) superior pancreaticoduodenal artery and anterior arterial arcade of the head of the pancreas, (5) right gastroepiploic artery continuing from the gastroduodenal artery.

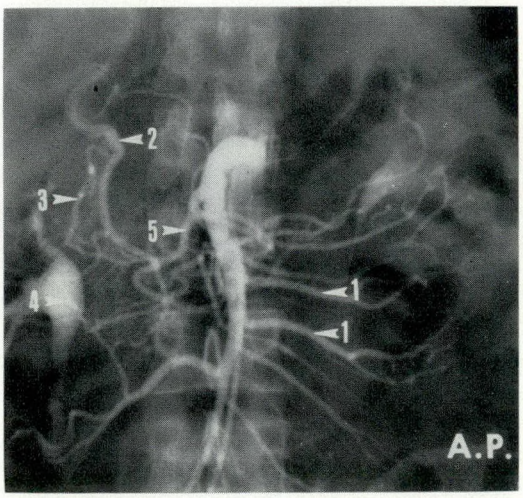

Fig. 2.—Selective superior mesenteric arteriogram (same patient as Fig. 1): (1) jejunal branches, (2) gastroduodenal artery, (3) retroduodenal artery, (4) superior pancreaticoduodenal artery, (5) inferior pancreaticoduodenal artery. The arterial arcades of the head of the pancreas are filled equally well from the superior mesenteric or celiac injection.

teries supplying the pancreas were dilated. Consequently there was a more than usually free communication between the celiac and superior mesenteric arteries, an appearance which Odman has described in chronic pancreatitis. In acute pancreatitis, Rosch and Bret[9] described a diffuse increase in vascularity and some irregularity of the arteries of the gland, and this appearance was present in one patient in this series who had subsiding acute pancreatitis. While these arteriographic changes may be found in pancreatitis, they are scarcely sufficiently characteristic to be of diagnostic value.

Pseudocysts of the pancreas were present in two of these patients but did not produce an arteriographic abnormality.

Nine patients with carcinoma of the pancreas were examined. Complete or partial obstruction of a major artery was found in six. Arterial obstruction occurred at the site of the tumour and appeared to be due to compression or invasion of the artery. Because most of the tumours involved the head of the pancreas, the artery most commonly obstructed was the gastroduodenal; however the superior mesenteric and celiac arteries were also affected. In one patient the celiac artery and its branches were completely obstructed.

CASE REPORT. — M.O., a 58-year-old woman, was first admitted with severe right upper quadrant pain of three-days duration. A pulsatile mass with a thrill was felt in the epigastrium. Laboratory investigation was normal. A selective arteriogram showed obstruction of the celiac artery with collateral flow to the liver and spleen from the superior mesenteric artery and it appeared that the pulsatile mass was the dilated superior mesenteric artery (Figs. 3 and 4). The patient had become asymptomatic in hospital and was discharged. Two weeks later she was readmitted with painless obstructive jaundice. At laparotomy the entire pancreas was found to be massively enlarged by a carcinoma, which enveloped the celiac axis and obstructed the common bile duct.

Arterial obstruction is not usually considered to be a complication of pancreatic carcinoma but we have found this to be the most common arteriographic feature and Rosch and Bret[9] reported finding arterial narrowing or obstruction in each of their 17 cases. Arterial obstruction also occurred with other disease processes, but the site and extent of the obstruction often suggested its nature.

In the majority of our patients carcinoma of the pancreas was remarkably "avascu-

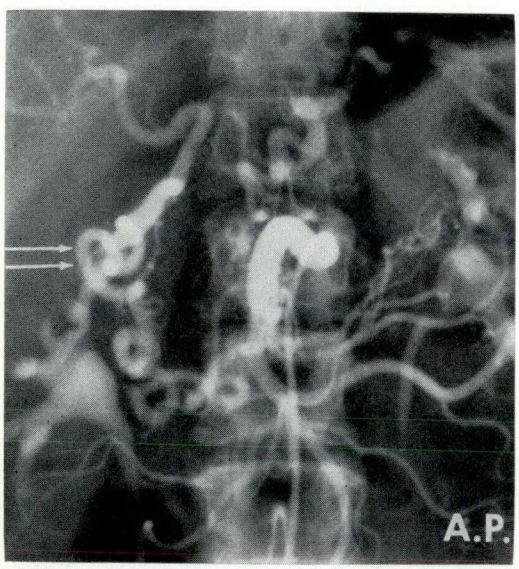

Fig. 3.—Case 1. Carcinoma of the pancreas causing obstruction of the celiac artery. Selective superior mesenteric arteriogram with tortuous dilated collateral vessels (double arrows) supplying the hepatic artery, and other smaller collaterals supplying splenic artery.

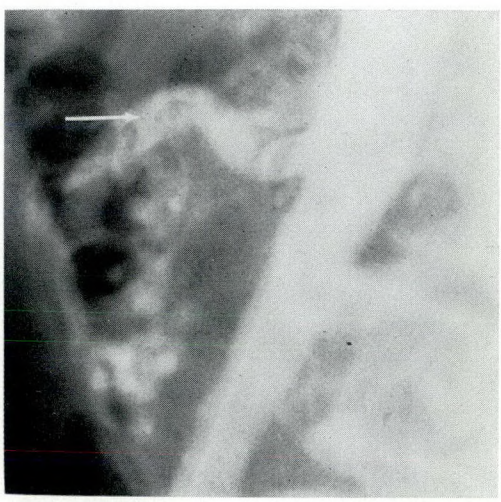

Fig. 4.—Case 1. Lateral aortogram showing dilated superior mesenteric artery (arrow) and failure to fill the celiac artery which normally arises 1 cm. above this.

lar", that is, even large tumour masses were present without evidence of any additional vascularity due to the tumour. In only four patients was some degree of excessive vascularity observed at the site of the tumour and in only two was this at all striking.

CASE REPORT.—J.L., a 71-year-old Chinese man complained of mid-abdominal pain of many months' duration. Slight tenderness was found in the mid-abdomen. Investigations, including gastroscopy, barium series and barium enema, were normal. Selective arteriography, however, showed tumour vessels in the head of the pancreas (Fig. 5). At laparotomy, an adenocarcinoma was found extensively infiltrating the head of the pancreas and the posterior wall of the stomach and duodenum.

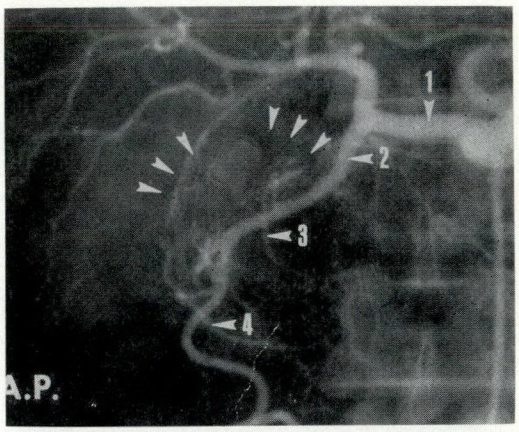

Fig. 5.—Case 2. Selective celiac arteriogram: (1) hepatic artery, (2) gastroduodenal artery, (3) retroduodenal artery and posterior arcade of the head of the pancreas, (4) superior pancreaticoduodenal artery and anterior arcade. The arrows outline a region of tumour vessels due to a carcinoma of the head of the pancreas which was beginning to invade the porta hepatis.

Even in this case the tumour vessels scarcely indicated the extent of the tumour. During arteriography, carcinoma can only be diagnosed positively by the presence of tumour vessels and our experience is in agreement with that of Rosch and Bret who observed arterial changes in each of their 17 patients but increased vascularity at the site of the tumour in only six. It must be concluded that arteriography is not altogether reliable in the diagnosis of carcinoma of the pancreas, but the accuracy of diagnosis has improved with the quality of the examination as we have gained experience in the assessment of the films and is now at least as good as with any other diagnostic method. In our experience a technically satisfactory normal arteriogram is fairly strong evidence against carcinoma of the pancreas. In the presence of carcinoma there will usually be arteriographic abnormalities which may be non-specific but which are sometimes diagnostic.

Benign adenomyomas and pancreatic islet-cell tumours may frequently be detected by arteriography. However, in our experience with a single 2 cm. insulinoma in the tail of the pancreas there appeared to be no arteriographic abnormality.

ABDOMINAL MASSES

Three patients with abdominal masses of unknown nature were examined by arteriography. One large mass, clinically thought to be an aortic aneurysm, was diagnosed by arteriography as a neoplasm of the mesentery or retroperitoneum, and proved at laparotomy to be a retroperitoneal liposarcoma. In the other two patients, a mass was felt in the right upper quadrant and was thought in one to arise in the right kidney. In each case selective arteriography showed a collection of tumour vessels in the liver; in one the lesion proved to be a primary hepatoma and the other a metastatic leiomyosarcoma of the liver (Fig. 6). The recognition of the site and blood supply of these tumours in the liver was of value not only in preoperative diagnosis but also in planning chemotherapeutic infusion. This information may prove to be of even more value in the future if segmental liver resection becomes an acceptable procedure.

Ten patients with diffuse liver disease due to metastases (seven) or cirrhosis (three) were examined. In these the arteriogram often showed a loss of normal branching of the intrahepatic arteries and a reduction in the flow in the main hepatic artery and, in a few cases, patches of "neovascularity" in the liver tissue, but these changes were rarely sufficiently distinctive to allow a positive diagnosis to be made.

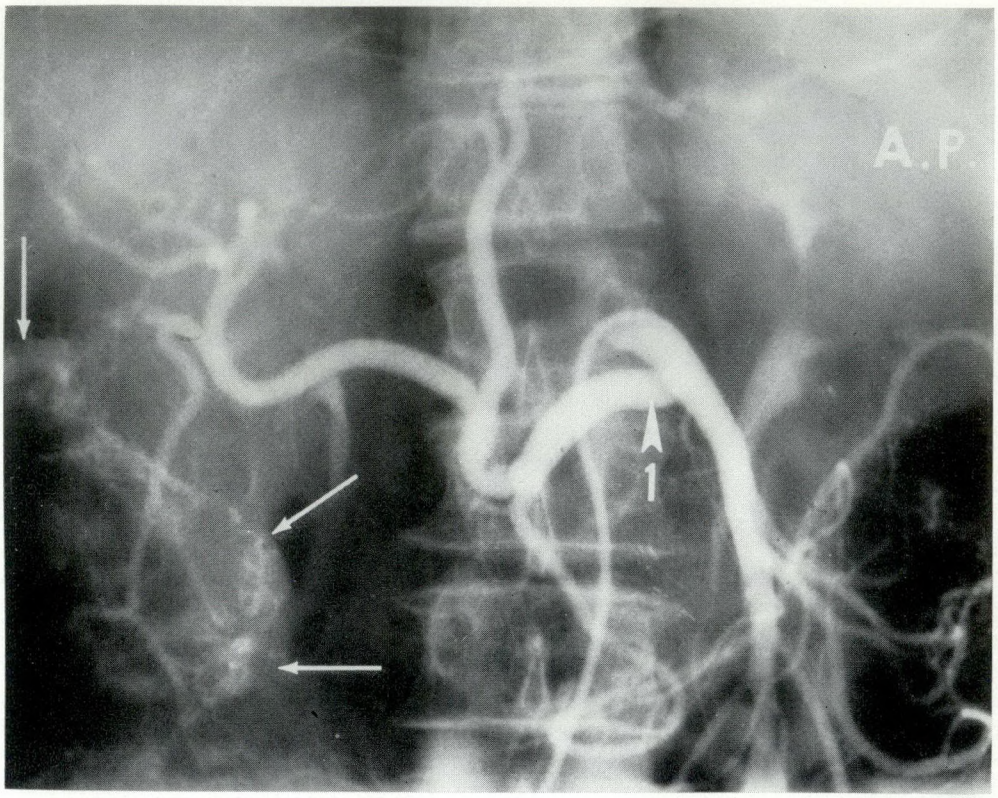

Fig. 6.—Leiomyosarcoma of the liver. Selective superior mesenteric arteriogram showing common hepatic artery (1) arising from the superior mesenteric and supplying a mass of tumour vessels (arrows) in the right lobe of the liver.

Carcinoma of the Stomach

Three patients with carcinoma of the stomach were examined. "Tumour" vessels were seen in only one case, and this agrees with other reports that gastric carcinoma is usually arteriographically avascular. In one patient, thickening of the wall of the stomach was recognized because the gastroepiploic artery was displaced from the gastric lumen. In another the tumour caused partial obstruction of the hepatic artery.

Selective arteriography appears to be of only occasional value in the diagnosis of carcinoma of the stomach.

Infusion Therapy

In 14 patients selective arteriography was used to determine the blood supply of a tumour for the purpose of chemotherapeutic infusion. The arterial supply of the tumour was unexpected in several of these patients owing, in some cases, to congenital anomalies and in others to neoplastic obstruction of the arteries. This obstruction was due to carcinoma of the pancreas in most cases but also occurred with carcinoma of the stomach. After the blood supply to the tumour had been identified, the catheter was left in the appropriate artery for infusion therapy. This has been well tolerated for treatments lasting up to 12 days.

Vascular Disease

The patient with acute mesenteric vascular occlusion may be too ill to tolerate investigation but, in selected cases, arteriography can be useful in determining the site of an arterial occlusion and the possibility of surgical repair of the artery. In a patient with embolic mesenteric ischemia where frank infarction had not occurred,

the site and possibility of removal of the emboli were determined by arteriography.

CASE REPORT.—W.R., a 79-year-old man, was found in a semi-comatose state with evidence of acute abdominal pain and vomiting. The pulse was rapid, faint and irregular and the abdomen was moderately rigid with reduced peristalsis. The clinical impression was cardiac fibrillation and an acute abdominal catastrophe. However, his poor general condition made surgery impossible and treatment was supportive with antibiotics and digitalis. His condition improved and 36 hours later a selective arteriogram revealed multiple emboli in the superior mesenteric artery (Fig. 7) and embolic occlusion of the left renal artery. It was not believed that the emboli could be surgically removed so anticoagulants were added to his treatment. His recovery was uneventful and complete.

Three other patients had obstruction of the celiac artery presumably due to atherosclerosis; however there were no signs or symptoms of arterial insufficiency. In each the obstruction affected the origin of the artery as is characteristic of atherosclerosis of the branches of the abdominal aorta. An abundant collateral blood supply from the superior mesenteric artery usually by the pancreaticoduodenal arterial arcades appeared to have compensated for the celiac obstruction (Fig. 8).

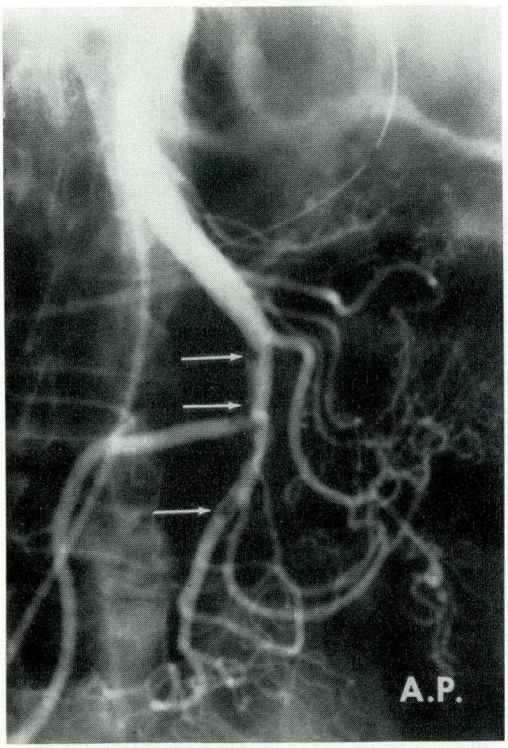

Fig. 7.—Case 3. Superior mesenteric arteriogram revealing multiple emboli (arrows) as filling defects in the main vessel and its branches. The arteries have filled distally indicating that the emboli have not caused complete obstruction.

DISCUSSION

With good radiological facilities and experienced personnel, selective arteriography has not proved to be technically difficult and a satisfactory examination can usually be obtained except where severe arterial disease is present. As our own experience and that of others indicates, selective arteriography can be done without complications. However, it must be emphasized that without facilities and experience in this area serious complications may be encountered which may outweigh the diagnostic value of the examination.

The interpretation of the arteriogram must be approached with caution. Familiarity with the anatomy of the region and an appreciation of the great variability which is normally possible are both necessary. A source of variability has been the occasional appearance of a "pancreatogram" because of contrast medium in the vessels of the pancreatic parenchyma. It may eventually be possible to produce this effect reliably and obtain a diagnostic "pancreatogram" of value. Other attempts to increase the utility of the examination have included "superselective" catheterization of branches of the celiac artery in an effort to provide improved arterial detail and also improved venous opacification.

Although selective arteriography has not proved to be as accurate in the diagnosis of tumours of the pancreas and liver as we had hoped, it has been of value in a sufficient number of cases and so it continues to be part of our diagnostic investigation of patients suspected of having these conditions.

CONCLUSION

Fifty-three patients have been examined

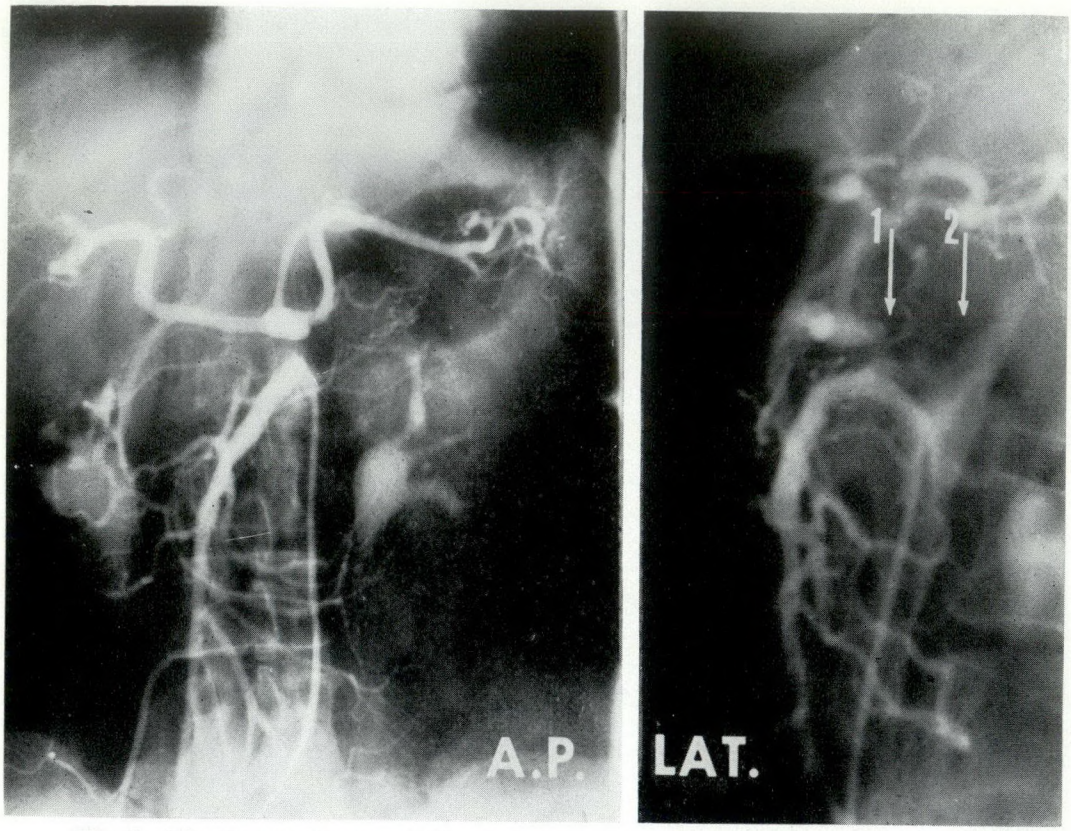

Fig. 8.—Selective superior mesenteric arteriogram in a patient who was being investigated for hypertension and had no abdominal symptoms. In the anteroposterior view the entire celiac system is seen to be supplied from the superior mesenteric artery by way of the pancreaticoduodenal arteries. In the lateral view of the same examination the obstructed portion of the celiac artery can be identified as the defect between the celiac artery opacified by collaterals distally (1) and the aorta visualized by contrast medium reflux (2).

by selective celiac and superior mesenteric arteriography. The examination has proven to be useful in: (1) The diagnosis of pancreatic tumours. (2) The diagnosis of masses arising in the liver and retroperitoneum. (3) The diagnosis of acute and chronic arterial obstruction. (4) The identification of the blood supply of inoperable tumours and as a method of instituting chemotherapeutic infusion in the appropriate artery.

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RÉSUMÉ

L'artériographie sélective par cathétérisation des artères cœliaque et mésentérique supérieures a été rapportée pour la première fois en 1951, par Bierman. Les auteurs rapportent leur expérience avec cet examen de diagnostic qu'ils ont utilisé durant les deux dernières années. Utilisant la méthode de Odman, ils ont pratiqué 53 artériographies, à l'aide du long cathéter Kifa radio-opaque dont l'extrémité a la forme d'une courbe semi-circulaire. Le cathéter est introduit dans l'artère fémorale selon la technique de Seldingers. Avec gentillesse, le bout du cathéter, monté jusque dans l'aorte abdominale est introduit soit dans l'artère cœliaque, soit dans l'artère mésentérique supérieure. Les auteurs signalent que l'expérience et la délicatesse des manœuvres permettent de diminuer au minimum les complications de la technique. Après avoir décrit quelque peu l'anatomie des troncs artériels cœliaque et mésentérique, ils repassent leur expérience clinique. Par exemple, ils ont pratiqué des artériographies dans cinq cas de pancréatite chronique récidivante, neuf cas de cancer du pancréas. Les auteurs analysent les clichés radiologiques du circuit artériel dans les diverses pathologies et rapportent quelques cas démonstratifs. Ils ont également utilisé cette méthode de diagnostic pour des masses abdominales, pour des cancers d'estomac et des maladies vasculaires, telle l'occlusion mésentérique. Il signalent l'utilité de cette technique dans un but thérapeutique d'une chimiothérapie régionale intra-artérielle, le cathéter étant placé dans l'artère principale du lit tumoral à infuser.

En conclusion, les auteurs sont d'avis que l'artériographie cœliaque et mésentérique sélective leur a paru utile pour le diagnostic des tumeurs pancréatiques, des masses tumorales hépatiques et rétro-péritonéales ainsi que pour le diagnostic des obstructions artérielles chroniques et aiguës. Enfin, cette technique permet d'étudier la vascularisation des tumeurs inopérables et d'instituer une infusion chimiothérapique.

RADICAL SURGERY FOR GASTRIC CARCINOMA*

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IN 1947 and a few years thereafter some surgeons[2-4] suggested that the standard procedure for carcinoma of the stomach should be radical total gastrectomy. The premise appeared to be a logical one. In order to test this premise, 46 patients were subjected to radical total gastrectomy at the Toronto General and the New Mount Sinai Hospitals, Toronto, between the years 1947 and 1958.

Judgment with respect to how much should be resected following gross inspection of the lesion is not reliable because wide permeation is often present well beyond it. In a study done in 1953 in cooperation with the Department of Pathology of the Toronto General Hospital,[1] I found evidence which confirmed the advisability of wider resection. Thirty-six patients operated upon by several surgeons, were studied because in these a partial resection had been performed in an attempt to obtain a cure. Examination of the removed gross specimens was startling in itself. In several, the proximal lines of resection were extremely close to gross disease, yet 33 of the pathological specimens had been reported as negative for residual carcinoma on the basis of one or two microscopic sections at each resection line. Ten new random sections were taken of the proximal cut-edge of these 33 "favorably reported" specimens and microscopic examination revealed carcinoma in 16 of them. If serial sections had been taken from both the proximal and distal ends of the specimen, probably more cancer would have been found. These findings convinced me of the futility of limited resection.

On this point, Berkson *et al.*,[2] in advocating partial resection, remarked: "We have found . . . that from three to five cm. beyond the area of extension of the lesion as indicated by immediate microscopic section, is a sufficient distance to carry the resection." However, the entire lesser curvature of stomach measures about 15 to 16 cm. and one rarely encounters a carcinoma smaller, in the gross, than five to seven cm. By their own standards therefore, except

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for those tumours which are very distally placed, the entire lesser curvature should be sacrificed in order to get well beyond the gross margins of tumour.

Furthermore, partial removal of the stomach is contrary to accepted principles concerning the surgical extirpation of carcinoma. Because mortality figures for extensive surgery in general were decreasing, it seemed reasonable that as this rate lessened even more, and cures from total gastrectomy increased, the case for radical total gastrectomy would be firmly established.

It is perhaps unfortunate that shortly after 1947, highly respected observers[8, 9] made statements that tended to discredit the radical procedure. Though their observations of increased morbidity and mortality from this procedure were correct, their conclusions influenced others to abandon the operation, or a modification of it, completely. These same observers stated that survival rates were also discouraging. It appears, however, that these rates were based on a series of cases in which the surgeons tended to choose total gastrectomy for large or proximally placed lesions. It is recognized that these cases always have a poorer prognosis. In my opinion and that of others[6, 7] no large group of cases has yet been treated by radical gastrectomy without previous selection. The reason for this will become evident later.

Material and Methods

In the present series, radical total gastrectomy, with block excision of the entire stomach, lymph-node-containing omenta, spleen, tail of pancreas, was carried out in 46 consecutive unselected patients. More pancreas or a portion of transverse colon were removed when indicated. All the cases were operated upon by myself or by a resident surgeon with my assistance.

Results

From 1947 to 1958, 137 patients with adenocarcinoma were seen and 111 were operated upon, the remainder being beyond even temporary palliation (Table I). Of those patients operated upon, 29 proved to be beyond help and only biopsies were performed. In 36 only palliative procedures were performed; these included partial gastrectomy, esophagogastrostomy, and gastroenterostomy. None of these latter patients lived more than three years and most died within a year. Forty-six patients remained in whom a radical attempt at cure could be made.

TABLE I.—Operable Patients with Adenocarcinoma of the Stomach

No. of patients seen	137
No. of patients operated upon	111
Exploration and biopsy only	29
No. of cases—palliative	36
No. of cases—total gastrectomy	46

For the most part, the technique used was end-to-end esophagojejunostomy with jejuno-jejunostomy (Table II). In this series, patients in whom this technique was used had less early and late complications than when other techniques were used. Nine patients had a Roux-en-Y procedure, one had esophagoduodenostomy and three had interposition of a length of small bowel.

TABLE II.—Techniques of Radical Total Gastrectomy

End-to-side esophagojejunostomy	7
with enteroenterostomy	26
Roux-en-Y esophagojejunostomy	9
Esophagoduodenostomy	1
with interposition of small bowel	3
	46

There were two deaths. One patient died of peritonitis following a massive leak in the upper suture line following an interpositional procedure. Death in the other was the result of myocardial infarction. Complications (Table III) ran the gamut of those encountered in such a lengthy and extensive procedure. Subhepatic or subphrenic abscess occurred in three, and required drainage; these probably represented additional suture-line leaks. Nonfatal pulmonary embolism occurred twice, and small intestinal obstruction, which required operation, occurred once.

None of these patients regained their pre-

TABLE III.—Complications and Deaths from Radical Total Gastrectomy

Early	
Suture-line leaks	4 (1 death)
Wound infection	6
Wound dehiscence	2
Intestinal obstruction	1
Subphrenic or subhepatic abscess	3
Pulmonary embolism	2
Cardiac infarction	1 (1 death)
Thrombophlebitis	5
Atelectasis	7
Late	
Cardiac infarction	4 ?
Dumping syndrome	31
Anemia—megaloblastic	8

operative weight. Of the 44 who survived to leave hospital, 31 complained of mild, moderate or severe dumping. In several, who lived only a few months to a year, it was difficult to determine when the effects of severe dumping blended into the results of residual disease, because they passed into a state of severe malnutrition and died. It is quite possible that one or several of these had no residual malignant disease, but died of malnutrition.

Of the 11 who have survived five years or more, four had moderate or severe dumping symptoms, but within two years these had subsided sufficiently to make life more comfortable (Table IV). Two patients developed strictures of the suture line and had to be operated upon after attempted bouginage. Of those that survived for more than three years, most developed anemia. In the early period, many had a mild hypochromic anemia. Four of the surviving patients have already developed a megaloblastic anemia and more will likely develop it.

TABLE IV.—Five-Year Results of Total Gastrectomy—46 Patients

Died in postoperative period	8
Died of cancer or malnutrition	22
Died of other causes	2
Lost to follow-up (dead?)	2
Living—with cancer	1
Living	11

In 1959, the procedure was modified for the following reasons:

First, the small number of survivors at that time was not encouraging, and the literature was not optimistic about survival. Second, the severe nutritional problems, with or without the dumping syndrome, faced in the first months after resection, were disheartening both to surgeon and patient. Several patients said that they preferred death to the constant severity of the symptoms. It is true however that the patients who lived on became more comfortable as a rule in the second year, and were helped by adjusting their diet and way of life.

It was decided that the operation was too radical. The physiological deficit created by removal of the whole stomach overbalanced the possible increased curative effects obtained. Probably, if the tissues could be removed with the same degree of completeness retaining a small amount of stomach, increased cures might still accrue, without the physiological defects. Therefore, it was decided to continue to remove the lesser curvature and other structures, but a small portion of cardia measuring about 2 x 5 cm. was preserved.

This small portion of cardia had proved to be invaluable in avoiding, to a great extent, many of the severe nutritional problems of total gastrectomy. Some of the patients have had moderate dumping symptoms, consistent with a high gastrectomy. A large enough series has not yet been collected to present more than our impressions of the value of this modified procedure.

Discussion and Conclusions

Two recent reviews[6, 7] assess fairly the controversy regarding the best surgical procedure for the treatment of carcinoma of the stomach. Most surgeons have discarded radical total gastrectomy because of associated nutritional problems, and because available data regarding cure are discouraging. No series has yet been reported which provides a clear answer to this problem: Does the probable increased rate of cure from radical total gastrectomy justify the serious complications of the procedure and grave long-term morbidity from which the survivors suffer? In many centres, surgeons are still choosing total gastrectomy

for patients with large tumours or high lesions, and this selection lowers survival figures. Those observers who have operated on consecutive cases have assembled a small series, such as this one, and the interpretation of their results has been modified by extraneous factors such as poor nutrition and anemia. Improvement in technique and the care of the patient requiring such a radical procedure has lowered mortality figures to less than 5%. Our knowledge of the treatment of the nutritional problems involved has improved as well. However, many patients who have been cured by radical total gastrectomy are still unhappy, because we cannot yet completely cope with the physiological changes which total gastrectomy produces.

The essence of any successful operation for cancer is complete removal of all malignant tissue and the preservation of the patient in a reasonable state of well-being. Conservative partial gastrectomy is quite inadequate in fulfilling the first condition. However, until associated nutritional problems can be solved, radical total gastrectomy does not satisfy the second. Thus far, one can more nearly approach these standards by radical subtotal gastrectomy, which leaves a small remnant of cardia, so that the patient's nutritional state remains satisfactory, yet more nearly fulfills the rules of good cancer surgery.

I would like to express my appreciation to Dr. F. G. Kergin for valuable suggestions.

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Résumé

Depuis 1947, la gastrectomie radicale totale a semblé rallier l'opinion chirurgicale comme traitement du cancer gastrique. Certains paramètres semblent d'ailleurs militer en ce sens. D'abord, l'impossibilité pour le chirurgien de délimiter l'extension macroscopique de la lésion. Au département de pathologie du "Toronto General Hospital", en 1953, une étude histologique sur 33 cas de gastrectomie, a montré la persistance de tissu néoplasique au niveau de la ligne proximale de section chirurgicale dans 16 cas; deuxièmement, la difficulté de conserver une partie de la petite courbure, si l'on veut amputer de 3 à 5 cm. au-delà des limites macroscopiques; enfin, la diminution notable de la morbidité et de la mortalité post-opératoires dans la gastrectomie totale.

Même si aucune série à date n'offre des statistiques significatives sur le meilleur traitement dans le cancer de l'estomac, l'auteur en 1947, avait décidé de faire une série personnelle de gastrectomies totales radicales. De 1947 à 1958, il a traité 137 cas de cancers gastriques, dont 46 ont subi une chirurgie extensive radicale. Sur 46 cas, on compte 11 survivants après cinq ans, deux cas de mortalité opératoire, et six cas de morbidité post-opératoire. Dans l'évaluation des résultats lointains, on remarque 31 malades qui se plaignent de "dumping". Chez les malades qui vivent plus de trois ans, la plus part développe une anémie et la majorité n'a pas rejoint son poids initial. En 1959, on décida de modifier la technique de gastrectomie radicale de sorte qu'il serait conservé une partie de la poche gastrique et du cardia, mesurant de 2 à 5 cm. Ce petit segment d'estomac a semblé avoir des résultats incalculables sur les problèmes nutritionnels consécutifs à la gastrectomie totale. En discussion, les auteurs remarquent qu'en principe, la chirurgie cancéreuse vise d'abord à exciser tout le tissu néoplasique et secondairement à préserver la vie du malade dans un état qui lui permettra de faire une vie normale. La chirurgie gastrique conservatrice ne remplit pas le premier point tandis que la chirurgie gastrique radicale manque au second. Aussi l'auteur pense-t-il que la modification de cette gastrectomie totale radicale en conservant une partie du cardia, pourra préserver l'état de nutrition de façon satisfaisante chez ceux qui survivront à l'opération tout en respectant les exigences de toute chirurgie cancéreuse.

THE MANAGEMENT OF HYPOPHARYNGEAL DIVERTICULUM

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DIVERTICULA of the esophagus can be classified as pulsion and traction types. The pulsion diverticula are further subdivided according to location, as (a) hypopharyngeal, (b) epiphrenic, and (c) those involving other sites.

Traction diverticula are curiosities found incidentally on radiographic studies of the esophagus.[6] They are usually situated near the tracheal bifurcation or left main bronchus, and most are said to result from adherence of the esophageal wall to inflamed lymph nodes, although some may be congenital. Pulsion diverticula are almost always juxtasphincteric, that is, hypopharyngeal or epiphrenic, but are occasionally seen in different sites in association with other esophageal disease, such as diffuse spasm. By far, the commonest form of diverticulum encountered in the esophagus is the hypopharyngeal type.

Hypopharyngeal diverticulum is usually described as a pulsion sac developing from the posterior wall of the pharyngoesophageal junction. The mucous membrane in this area bulges between the oblique and transverse fibres of the inferior pharyngeal constrictor muscle. The transverse muscle bundles are referred to as the cricopharyngeus muscle, and spasm or incoordination of this structure is believed to be the underlying cause of the diverticulum.[1] Three stages in the natural history of the diverticulum were described by Lahey[2] and symptoms were correlated with these stages. Recent work, notably that of Sutherland,[3] suggests that a stage "zero" could be termed "cricopharyngeal achalasia" (Fig. 1). Accordingly, hypopharyngeal diverticulum is one of the complications of the disease entity "cricopharyngeal achalasia". Another less common complication is sideropenic dysphagia.[4]

This paper reviews 37 consecutive cases of hypopharyngeal diverticulum seen at a university hospital over a period of 13 years (Table I). During the period of the review, three mid-thoracic diverticula and one epiphrenic diverticulum were encountered.

TABLE I.—FREQUENCY OF THREE TYPES OF DIVERTICULA

Type	*Cases*
Hypopharyngeal	37
Mid-thoracic	3
Epiphrenic	1

CLINICAL FEATURES

Age, Sex and Duration of Symptoms.—Among the patients investigated in this study, there were 22 men and 15 women. The average age for patients in this series

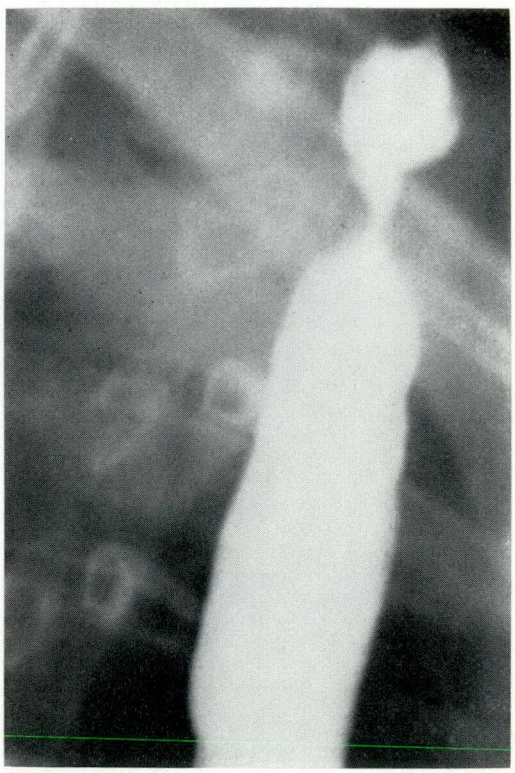

Fig. 1.—Barium swallow showing spasm of the cricopharyngeus muscle.

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was 66 years; the youngest was 48 years of age and the oldest was 83. The average age for men was 65 years and for women 67 (Table II). In the group as a whole symptoms were present for an average of five years before treatment. Among men the duration of symptoms averaged four years, while in women it averaged nine. The women in this series had endured the condition for a longer time than had the men.

TABLE II.—Age and Sex of Patients with Hypopharyngeal Pouches

	All cases	*Males*	*Females*
Number	37	22	15
Average age	66 yrs.	65 yrs.	67 yrs.
Average duration of symptoms	5 yrs.	4 yrs.	9 yrs.

Symptoms (Table III).—All patients had dysphagia. This varied from a delay in the swallowing of liquids to complete obstruction for liquids. Most patients also had other symptoms related to the presence of the pouch, such as gurgling in the neck, regurgitation of undigested food, a swelling in the neck, or excessive oral mucus. Many patients avoided eating in public places, a symptom common to other forms of obstructive esophageal disease. The degree of dysphagia did not correlate well with the size of the pouch.

Twenty-one patients had weight loss of some degree, and none had gained weight after the onset of symptoms.

Nineteen patients had symptoms which were regarded as due to aspiration of pouch contents into the lungs. This was considered probable where there was a history of choking with meals, coughing spells on lying down, or the postoperative clearing of radiologic evidence of aspiration pneumonitis.

TABLE III.—Presenting Symptoms

	No. of patients
Dysphagia, etc.	37
Weight loss	21
Lung symptoms	19

One patient had a family history of hypopharyngeal diverticulum. Minimal neck pain was present in one patient.

Diagnosis

The diagnosis was made by history and radiologic examination. Esophagoscopy was not performed routinely, and in recent years has been considered unnecessary and dangerous. Eleven patients had had other diseases of the upper gastrointestinal tract at some time in the past (Table IV). Duodenal ulcer antedated the diverticulum in four patients, and a fifth patient died of a perforated duodenal ulcer three years after successful excision of a hypopharyngeal diverticulum. Perforation of a gastric ulcer had occurred some years before in one patient, and a prepyloric ulcer was present in another patient at the time the hypopharyngeal diverticulum was diagnosed. Carcinoma of the cardia was discovered in one man only six months after unsuccessful treatment of a hypopharyngeal diverticulum by dilatation. Chronic anemia was present in only one patient; this was found to be associated with a carcinoma of the stomach three years after excision of a hypopharyngeal diverticulum. Hiatal hernia was seen in two patients. In one, symptoms of hiatal hernia were of at least 10 years' duration, while the symptoms due to hypopharyngeal diverticulum had been present for only four years. The hiatal hernia was first repaired, and two weeks later the diverticulum was excised. A second patient was known to have a hiatal hernia at the time of excision of a hypopharyngeal diverticulum. Four years later she again presented with dysphagia which responded to surgical correction of the hiatal hernia.

TABLE IV.—History of Upper Gastrointestinal Lesions in Patients with Hypopharyngeal Diverticula

	No. of patients
Duodenal ulcer	5
Gastric ulcer	2
Cancer of the stomach	2
Chronic anemia	1
Hiatus hernia	2

Operative Findings

The sac was described as small in seven patients, and moderate to large in 23. No mention of its size was made in seven. The radiologic appearance of a large diverticulum is shown in Fig. 2. The postoperative barium swallow of the same patient is shown in Fig. 3. The pouch was midline in eight patients, in the left side of the neck in 10, in the right side of the neck in seven, and the location was not indicated in 12.

At operation the sac was readily dissected from the surrounding tissues except in one patient who had previously had a thyroidectomy and in another patient with a recurrent pouch. There was no suspicion of malignant change in any specimen. Histological examination disclosed signs of chronic inflammation in the submucosa, and in a few cases showed superficial ulceration of the mucosa.

Management

All patients were thought to require operative treatment of some sort (Table V). Many needed a period of preoperative preparation, usually lasting a few days, for such reasons as correction of dehydration, improvement of pulmonary infection and so on. One man required gastrostomy feeding for three weeks before he was fit for surgery.

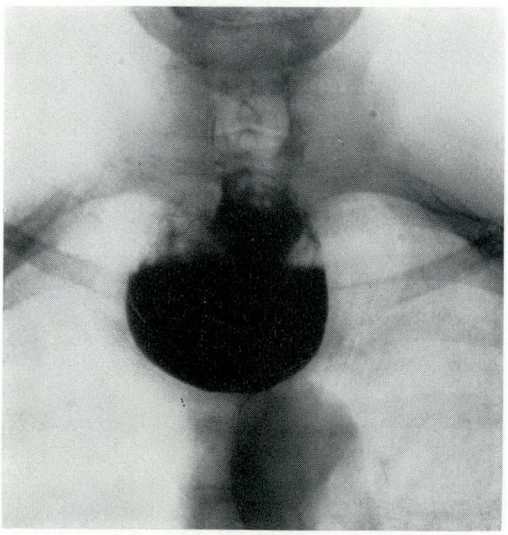

Fig. 2.—Barium filling a large hypopharyngeal diverticulum.

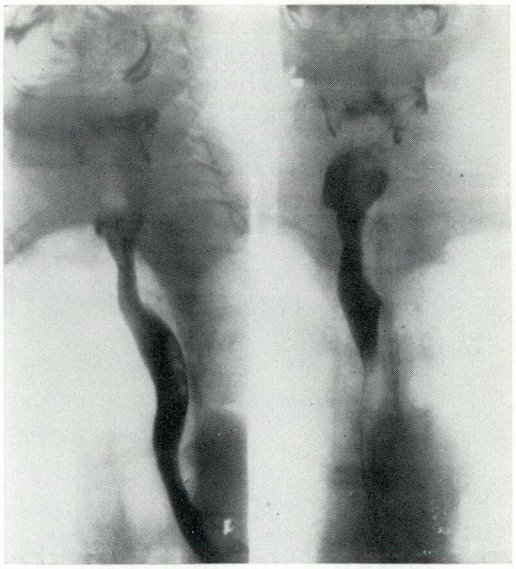

Fig. 3.—Postoperative barium swallow of patient shown in Fig. 2.

One man died from aspiration pneumonia soon after admission, before specific treatment for the diverticulum could be given. He had been known to have a symptomatic hypopharyngeal diverticulum for at least five years but had refused treatment.

Two patients with very small pouches were managed solely by dilatation of the cricopharyngeus muscle. Two patients were treated by Dohlman's technique[5] of endoscopic diathermy of the cricopharyngeus. The remaining 32 patients had a one-stage excision of the sac. One recurrent pouch was excised, making a total of 33 excision operations.

TABLE V.—Treatment

	No. of patients
Dohlman's operation	2
Excision	32
Dilatation	2
None	1

Technique of Excision Operation

The technique, which has been modified over the years, is as follows: Endotracheal anesthesia is used in all cases and is considered safer than local anesthesia. In re-

cent years a standard collar incision has been used rather than an incision along the anterior border of sternomastoid. Access to the sac is good irrespective of its location. Skin and platysma flaps are reflected and the fascia in front of the sternomastoid is opened. The omohyoid muscle and middle thyroid vein are divided between clamps and ligated. The thyroid gland is retracted forward and the carotid sheath and sternomastoid laterally. The inferior thyroid artery is usually divided after identifying the recurrent laryngeal nerve.

The pouch is then found lying on the cervical vertebrae and prevertebral muscles and is readily delivered from surrounding tissues by blunt dissection.

The areolar tissue and scattered muscle fibres forming the outer layers of the pouch are then "circumcised" a short distance beyond the site where the mucosa will finally be transected. After carefully viewing the junction of the neck of the pouch with the esophagus on all sides, a clamp is applied just distal to the line of excision; the latter may be transverse or vertical depending on the axis of the opening into the pouch. Care must be taken not to pull esophageal mucosa outwards for excision with the sac.

The sac is then amputated completely or in stages, as the mucosa is closed with continuous or interrupted sutures of silk, chromic gut or wire. The muscularis is then approximated. A two-layer closure is always used.

At this point in the procedure in recent years, a vertical myotomy is done through the cricopharyngeus and adjacent fibres above and below. Sutherland[3] has advised that the myotomy be performed before the mucosa is closed, to aid in visualization of the transverse muscle bundles in certain cases. Especially with large sacs it may be difficult to display significant transverse muscle fibres below the neck of the protrusion. The myotomy is technically similar to the procedure followed in the Heller or Ramstedt operations. The wound is then closed and a Penrose drain is retained for 24 to 48 hours. Oral liquids are started on the second day, and the intake is gradually increased to a full diet by the tenth day.

Morbidity After Excision Operations

There was no mortality after excision operations in this series. One patient developed a purulent staphylococcal wound infection (Table VI). This responded to simple drainage and antibiotics without compromise of esophageal healing.

TABLE VI.— Morbidity after Excision Operations

	No. of patients
Purulent wound infection	1
Temporary gastrostomy	1
Wound allowed to granulate	1
Temporary fistula	1

Too much mucosa was included in one excision, and the esophageal closure was felt to be insecure. A gastrostomy was performed under the same anesthetic and the patient was then tube-fed for a week. Thereafter oral feedings were begun and he was discharged two weeks later, swallowing normally.

Extensive soiling of the neck occurred during excision of one pouch. The esophagus was closed but the main wound was packed and allowed to granulate. Feeding was via a nasogastric tube for 10 days while the pack was gradually removed. Swallowing was normal a week later, and the patient was discharged.

One patient developed a pharyngocutaneous fistula on the seventh postoperative day, which drained saliva and pus. This was managed by a single dilatation of the cricopharyngeus and temporary restriction of oral feedings. The fistula closed spontaneously, and the patient was discharged three and one-half weeks after operation.

Follow-Up

Two patients, who had excision operations, have emigrated and have not been traced. One patient died of pneumonia soon after admission to hospital, before the pouch could be treated. The remaining 34 patients were followed-up for an average of 27 months each, the shortest time being three months.

(1) *Dohlman's Operation.*—The two pa-

tients managed by Dohlman's operation are well, 14 and 29 months later.

(2) *Dilatation.*—Two patients were managed by dilatation alone. One is swallowing only moderately well nine months later, but refuses further treatment. The other patient had a three-month history suggestive of hypopharyngeal diverticulum and this lesion was seen when the upper esophagus was examined radiologically. A single dilatation was performed. Dysphagia returned soon after and six months later a more complete radiologic examination showed a carcinoma of the cardia, in addition to the hypopharyngeal diverticulum. An esophagogastrectomy was done for the carcinoma and a further cricopharyngeal dilatation for the diverticulum. His subsequent swallowing history is difficult to assess.

(3) *Excision Operations.* — Following these procedures there were two recurrences. The first was discovered six months after the original operation when the patient was examined for persistent dysphagia. Esophagoscopy revealed a small pouch and a tight cricopharyngeus. A second excision operation was carried out, and he was swallowing satisfactorily five years later. The second patient with a recurrence was operated on at 48 years of age, having had increasing dysphagia for more than 10 years. Following excision of the pouch, swallowing was always "a bit tight". Radiologic examination 14 months later again showed a hypopharyngeal diverticulum but no further treatment has yet been given. Neither patient with a recurrence had a myotomy done at the time of excision.

Another patient required repeated dilatations for seven months following excision of a hypopharyngeal diverticulum; myotomy was not performed in this case. He then swallowed well for three and one-half years before dying of a perforated duodenal ulcer.

Following successful excision operations two patients developed dysphagia that was definitely due to other lesions. One of these returned 45 months after operation with a large incarcerated hiatal hernia. This was treated by operation, giving relief of dysphagia. The other patient returned with dysphagia three years after successful excision of a hypopharyngeal diverticulum. Investigation revealed a gastric carcinoma which was then resected. A third patient, mentioned above, was managed by dilatation alone, and his recurrent dysphagia may have been partly due to gastric carcinoma.

As shown in Table VII, recurrent or persistent dysphagia was seen in six patients. Three of these had been treated by excision operations when a concomitant myotomy was not performed. Three other patients had recurrent dysphagia due to pathology other than hypopharyngeal diverticulum.

TABLE VII.— RECURRENT OR PERSISTENT DYSPHAGIA

	No. of patients
Recurrent pouch	2
Cricopharyngeus stricture	1
Cancer of the stomach	2
Hiatus hernia	1

CONCLUSIONS

Review of these cases suggests the following:

The treatment of hypopharyngeal diverticulum is surgical; simple dilatation is not effective.

Many patients with this disease have lung complications.

In the investigation of these patients, attention must be given to the entire upper alimentary tract; esophagoscopy is not usually indicated.

Dohlman's operation seems effective but the indications for it and the long-term results have not yet been established.

Excision of the pouch remains the standard treatment. This should be combined with an extramucosal myotomy, which is probably more important than excising the pouch flush with the esophagus.

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RÉSUMÉ

L'auteur passe en revue les dossiers d'un groupe de 37 malades consécutifs traités pour diverticules de l'hypopharynx. On comptait 22 hommes et 15 femmes. L'âge moyen des patients était de 67 ans. Les symptômes existaient en moyenne depuis cinq ans avant l'hospitalisation. Tous les malades se plaignaient de dysphagie et plusieurs présentaient des symptômes pulmonaires considérés comme attribuables à l'aspiration périodique du contenu du diverticule. Un malade mourut d'inanition peu après son entrée à l'hôpital. Quant aux autres, ils furent traités par diverses méthodes chirurgicales, dont la dilatation, l'opération de Dohlman et l'excision. Les résultats permettent de croire que: l'excision est la méthode la plus sûre si elle est accompagnée d'une myotomie du muscle cricopharyngien; l'opération de Dohlman semble valoir la peine d'y recouvrir et la dilatation, en soi, sera probablement inefficace.

RESPONSES OF THE GASTROESOPHAGEAL JUNCTIONAL ZONE TO INCREASES IN ABDOMINAL PRESSURE*

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PREVIOUS manometric studies in normal subjects have revealed the presence of a zone of elevated pressure situated between the lower esophagus and the stomach.[1, 2] This zone extended both above and below the diaphragm and was thought to represent an intrinsic sphincter which maintained the pressure gradient between these respective organs. Fyke, Code and Schlegel[1] reported that when subjects were placed in the head-down position, thus increasing intra-abdominal pressure, the pressure within the gastroesophageal junctional zone increased and was maintained above gastric pressure. However, Nagler and Spiro[3] observed that increases in intra-abdominal pressure produced no change in the resting pressure in the portion of zone located above the diaphragm. Because of this controversy the present study was undertaken in an attempt to delineate the responses in the gastroesophageal junctional zone following increases in abdominal pressure. Specific attention was directed to accurate placement of the recording units, particularly in the segment of the zone located above "the level of the diaphragm", which for this purpose was taken as the point of respiratory reversal. There is evidence that this point does not correspond exactly with the anatomical position of the diaphragm.[2] To study the junctional zone without the influence of the diaphragm, investigations were also made on selected patients with large, fixed hiatal hernia, in whom the junctional zone was located in the thorax, and therefore well above the diaphragm.

METHODS

Intraluminal pressures within the stomach, junctional zone, and body of the esophagus were recorded by means of open-tipped polyethylene tubes (I.D. 0.066″, O.D. 0.095″). The tubes were kept patent by a continuous infusion of water at a constant rate. Respiration was recorded by means of a tubular pneumograph placed about the subject's chest. Each tube and the pneumograph were connected to strain gauges (Statham P23Db and PM5TC, respectively), the outputs of which were led to a recorder (1108 Honeywell visicorder oscillograph). The transducers were placed at approximately the same level as the

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tube openings to minimize error due to hydrostatic pressure. The system was calibrated at the beginning and end of each test. The mean pressure between end-inspiration and end-expiration was calculated for each recording site. This was expressed in centimetres of water pressure with reference to atmospheric pressure. Although other authors have reported results using intragastric pressure as the reference, we have used atmospheric pressure as the reference. The exact position for each recording site was measured in centimetres from the incisor teeth.

When the detecting units were in the abdomen, the major deflection with inspiration was positive. As the units were withdrawn from the abdomen into the thorax, the direction of pressure deflection with inspiration reversed sharply, and the major deflection became negative. The "level of the diaphragm" was taken as the point of respiratory reversal.

Part I: Studies on Normal Subjects

(a) *Resting Pressure Profile.*—The first series of investigations was conducted on 14 normal subjects whose ages ranged from 19 to 59 years with a mean age of 32 years. Pressures were recorded by means of three open-tipped tubes, each with a single lateral orifice located at five, 10 and 15 cm. from the distal tip. The size of the orifice was equal to the diameter of the tube (Fig. 1), and the mid-point of the opening was used as the reference point for the measurement with respect to the incisor teeth. During an individual study the tubes were passed through the mouth and advanced until the detecting units were situated in the stomach. The subject then assumed the supine position, the tubular pneumograph was attached, and a pneumatic cuff was placed about the abdomen. The pneumatic cuff was a standard cuff used for measuring blood pressure at the thigh and measured 6.5 × 22 in. The cuff was centred over the umbilicus and was held in position by a many-tailed abdominal binder (Scultetus binder). The subject was instructed to refrain from swallowing and to breathe in a normal manner. A continuous recording of resting pressures was then made as the units were withdrawn at 0.5 cm. intervals from the stomach into the body of the esophagus without the application of abdominal pressure (Fig. 2). (The body of the esophagus refers to that portion of the organ proximal to the junctional zone.) The units were then returned to the stomach and a second recording of the pressure profile of the junctional zone was made during the application of 50 mm. Hg pressure to the anterior abdominal wall. At each station pressures were recorded for 10 to 15 seconds. Occasionally the sequence of the recordings was reversed so that the first recording was made during the application of abdominal pressure. Pressures were calculated from the

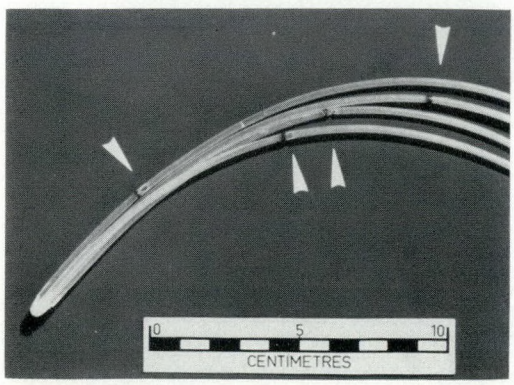

Fig. 1.—Pressure detecting units. Water-filled polyethylene tubes each with a lateral orifice (arrows) situated five, 10, 11.5 and 15 cm. from the distal tip.

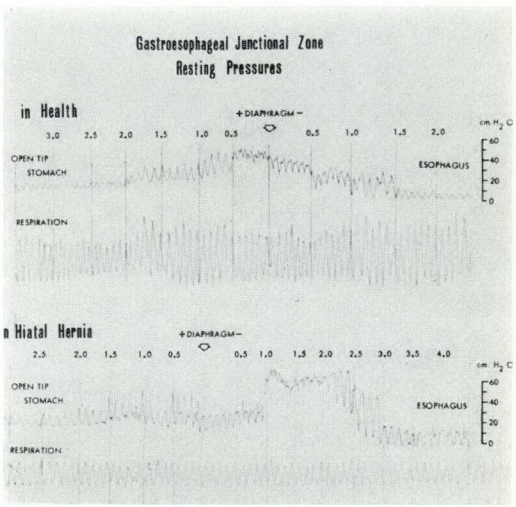

Fig. 2.—Recordings of the resting pressure profile of the gastroesophageal junction zone in health (upper) and hiatal hernia (lower).

recording of the distal detecting unit, while the two proximal units were used to monitor esophageal motor activity.

(b) *Resting Pressures From Selected Sites.*—The second portion of the study was conducted on 10 normal subjects whose mean age was 24.0 years with a range of 19 to 31. The recording units consisted of four open-tipped tubes, each tube having a single lateral orifice, located five, 10, 11.5, and 15 cm. from the distal tip (Fig. 1). During a test, an initial resting pressure profile of the junctional zone was recorded. From this recording the length, pressure characteristics, and distribution of the junctional zone in relation to the "level of the diaphragm" were determined. This special arrangement of detecting units was then positioned so that pressures could be recorded simultaneously from the stomach, segment of the zone below the "level of the diaphragm", portion of the zone above the "level of the diaphragm", and from the body of the esophagus. With these units in position, pressure recordings were then made without the application of extraneous pressure, and with the application of 50 and 100 mm. Hg pressure to the anterior abdominal wall. Recordings were made for 15 to 20 seconds following each increment in abdominal pressure.

Part 2: Studies on Subjects with Hiatal Hernia

The final portion of the investigation was conducted on nine patients with large fixed hiatal hernia as diagnosed by radiography and endoscopy. The mean age of this group was 63 years (range 35 to 73). Previous motility studies on these subjects had revealed that the maximum pressure in the gastroesophageal junctional zone was within the normal range. In addition they did not have clinical or endoscopic evidence of esophagitis and the sphincter in these subjects appeared to have normal motor function even when displaced into the thorax. Other studies in our laboratory have shown that in patients with hiatal hernia and a feeble junctional zone, the zone became incompetent during abdominal compression.[4] All of the latter patients had clinical and endoscopic evidence of esophagitis indicating abnormal function of the junctional zone; they are therefore not reported in this paper. The test methods employed were identical to those used in the first group of normal subjects (Part 1a).

The changes in pressures in the stomach, junctional zone and esophagus resulting from the application of abdominal compression were analyzed within each group of subjects by using the "t" test for paired data. The analysis of mean changes in pressure between groups was made using the "t" test for unpaired data.[5]

Results

In the first group of 14 normal subjects the mean intragastric pressure was 12.1 (±S.E. 0.82) cm. of water pressure above atmospheric pressure (Fig. 3). As the pressure-detecting unit was withdrawn from the stomach into the esophagus, a zone of elevated pressure was encountered. This zone was 3 to 5 cm. in length, with equal distribution above and below the "level of the diaphragm". The mean maximum pressure in this zone was 35.6 cm. H_2O (±S.E. 3.48). The pressure within the body of the esophagus was 1.5. This latter value is considered to approximate normal intrathoracic pressure with subjects lying supine.

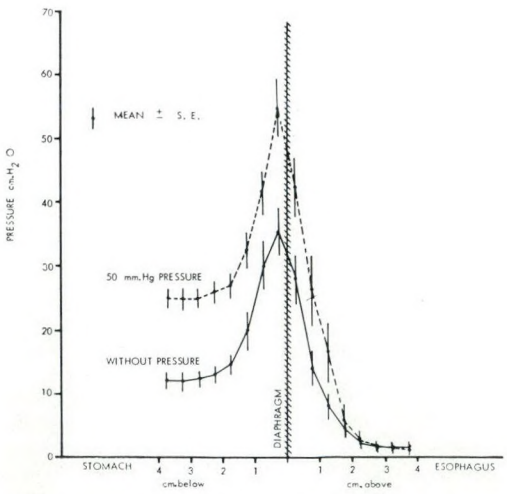

Fig. 3.—The effect of abdominal pressure on mean resting pressures in the stomach, gastroesophageal junctional zone and body of the esophagus in 14 normal subjects.

During the application of pressure to the anterior abdominal wall, the resting pressure in the stomach was 24.9 (±S.E. 1.47) cm. H_2O, a mean increase of 12.8 (Fig. 3). In each subject, intragastric pressure was elevated above the control value ($p < 0.001$). The length of the zone of elevated pressure and its relationship to the diaphragm remained unchanged in each subject. There was an increase in pressure throughout the junctional zone, the mean maximum pressure increasing to 54.8 (±S.E. 4.2) cm. H_2O. The increase in pressure at each recording site in the portion of the zone situated below the "level of the diaphragm" was significant ($p < 0.01$). The greatest increase in pressure in this segment occurred at the point of maximum pressure and was 19.2 cm. H_2O. The pressures in the two recording stations immediately above the "level of the diaphragm" were also significantly increased ($p < 0.01$). Above this point and in the body of the esophagus there was no significant change in pressure.

When the pressure-recording units were positioned in the selected recording sites similar results were obtained. In every subject each increment of abdominal pressure caused a rise in the pressure recorded from the stomach and from the portions of the junctional zone located above and below the "level of the diaphragm" ($p < 0.001$) (Fig. 4), but not in the body of the esophagus. In nine of the 10 subjects the pressure in the supradiaphragmatic segment was similar to intragastric pressure. A comparison of these pressures (Fig. 4) to those recorded in this segment under resting conditions and during abdominal compression in the previous group (Fig. 3) indicates that the orifice was located 1.0 cm. above the level of the diaphragm. Since the openings in the two tubes in the junctional zone were only 1.5 cm. apart, the pressure recorded in the infradiaphragmatic segment was near peak levels.

In the patients with hiatal hernia the pressure profile of the gastroesophageal region was quite different from that recorded in the normal subjects and confirms that reported by other authors.[5, 6] As the pressure-detecting units were retracted from the stomach into the esophagus, two zones of elevated pressure were recorded with an intervening plateau (Figs. 2 and 5). The double respiratory reversal described by other investigators was noted in many of the patients. The "level of the diaphragm" in these cases was taken as the distal point of respiratory reversal.

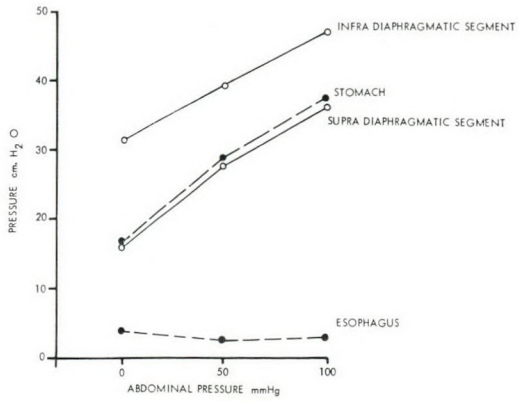

Fig. 4.—The effect of abdominal pressure on mean resting pressures recorded simultaneously from the stomach, both segments of the junctional zone and body of the esophagus in 10 normal subjects.

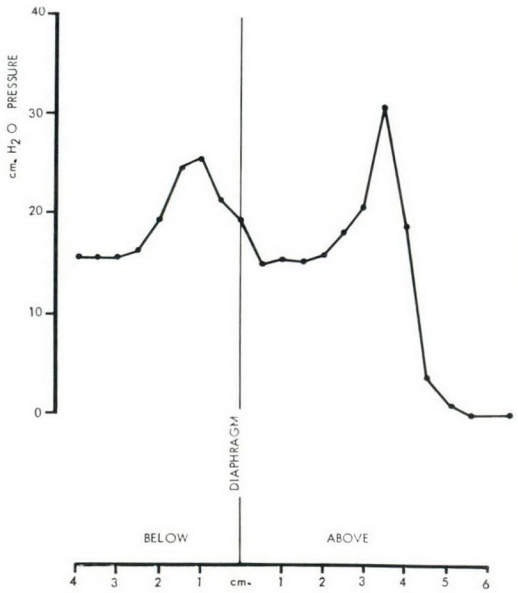

Fig. 5.—Graph of the mean resting pressure profile of the gastroesophageal region in one representative patient with hiatus hernia.

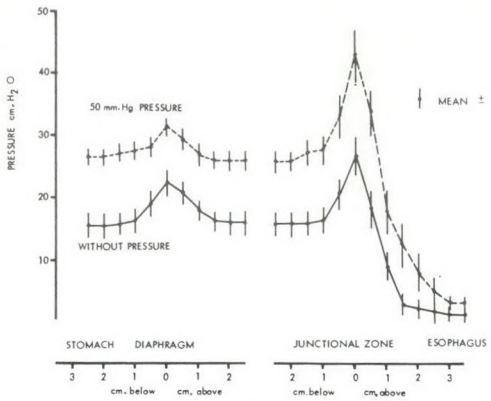

Fig. 6.—The effect of abdominal pressure on mean resting pressures in the stomach, gastroesophageal junctional zone and body of the esophagus in nine patients with hiatus hernia.

Because the length of the plateau representing the hernial sac varied between patients, these data (Fig. 6) were plotted differently from the data in Fig. 3. Pressures in the upper zone of elevated pressure were plotted such that the zero position was taker as the point of maximum pressure recorded in this zone in each patient. The same procedure was used in plotting mean pressures recorded in the lower zone of elevated pressure in these subjects. During abdominal compression the point of maximal pressure in the upper zone moved proximally in some subjects but not more than 1 cm.

On the resting study intragastric pressure was 15.5 (±S.E. 1.8) cm. H_2O, similar to that recorded in the normal subjects (Fig. 6). The maximum pressure recorded in the zone related to the "level of the diaphragm" was 25.0 (±S.E. 1.6) and the length of this zone was approximately 3.0 cm. Above this region in the segment between the two zones of increased pressure the pressure returned to the intragastric level (15.9). The upper zone of elevated pressure was approximately 4.5 cm. in length and its maximum pressure was 26.9 (±S.E. 2.84). The pressure in the body of the esophagus was 1.6 cm. H_2O.

Following the application of extraneous abdominal pressure, intragastric pressure increased by 11.0 to a mean of 26.5 (±S.E. 1.1) cm. H_2O ($p < 0.001$). In the region of the diaphragm the pressure increased to 31.5 cm. H_2O, (±S.E. 1.43), representing a significant increase of 6.5 ($p < 0.02$); however, the length of this zone was unchanged. In the plateau above the "level of the diaphragm" the pressure was the same as the intragastric level. In the zone proximal to the plateau, the maximum pressure was 43.2 (±S.E. 4.03) cm. H_2O an increase of 16.3 ($p < 0.001$). The length of this zone was increased to 5.0 cm. In the body of the esophagus the pressure was only slightly elevated above the control value.

Discussion

In the normal subjects the length, pressure characteristics and relationship of the gastroesophageal junctional zone to the "level of the diaphragm" were similar to those reported by other authors.[1, 2]

During application of pressure to the anterior wall in all of the normal subjects, an increase in pressure occurred in the stomach and in both segments of the junctional zone while no change was noted in pressure within the body of the esophagus. The finding of a significant rise in pressure in the segment of the zone situated above the "level of the diaphragm" was contrary to the results reported previously by Nagler and Spiro.[3]

The response in the junctional zone in the normal subjects may have been due to compression of an intra-abdominal portion of the esophagus in conjunction with external compression by the diaphragm. It also could have been due to contraction of an intrinsic sphincter acting independently or in concert with the diaphragm. In the 14 normal subjects there was a greater pressure increase at the point of maximum pressure in the junctional zone than in the stomach. This evidence suggested that the response in the junctional zone was not purely a reflection of the increase in intra-abdominal pressure. The additional response must have been due to muscular contraction of the junctional zone or the diaphragm, each acting independently or together. To elucidate these factors the final part of the study was carried out on patients with large fixed hiatal hernia, in whom the gastroesophageal junction was displaced into the thorax well above the

diaphragm.

In these subjects there were two zones of elevated pressure with an intervening plateau.[6, 7] The distal zone was in relation to the diaphragm and was attributed to diaphragmatic action. The pressure plateau was believed to represent the pressures as recorded from the herniated portion of the stomach. The upper zone was thought to represent the gastroesophageal junctional zone displaced into the thorax, for the following reasons. First, esophageal pressure proximal to this zone was similar to that recorded in the normal subjects. Secondly, the response to deglutition in this zone was one of relaxation followed by contraction and was identical to the response seen when the zone was in its normal position in healthy subjects. Usually no response to deglutition was seen in the region of the plateau or lower zone of elevated pressure, but when a response did occur it was always one of only slight relaxation. Finally the length and maximum pressure recorded in the upper zone of elevated pressure was not significantly different from that recorded in the junctional zone in the normal subjects.

When pressure was applied to the abdominal wall in subjects with hiatal hernia, the elevation in intragastric pressure was comparable to that seen in the normal subjects. The pressure in the region of the diaphragm also increased, but the increase was less than that seen in the stomach. In the plateau above the diaphragm the increase in pressure was equal to that recorded in the stomach. These results reveal that the diaphragm did not maintain a pressure gradient between the abdomen and the thorax. In the gastroesophageal junctional zone, the resulting rise in maximum pressure was greater in magnitude than the increase in intragastric pressure, and the length of the zone was only slightly altered. In this location the junctional zone could not have been influenced by the diaphragm or intra-abdominal pressure and any increase in pressure in this region must have been due to intrinsic contraction of a muscular sphincter. In addition, the increase in pressure seen in this zone was not significantly different from that seen in the group of normal subjects. Therefore, the major portion of the increase in pressure in the zone, following application of abdominal pressure in the normal subjects, was probably due to intrinsic muscular contraction.

The present study confirms the presence of a sphincter, intrinsic to the gastroesophageal junctional zone. This sphincter responds to increases in abdominal pressure by active contraction thus maintaining a pressure gradient between the abdomen and the thorax. The mechanism resulting in this response has not been delineated in this investigation.

Summary

An investigation was undertaken to study the response of the gastroesophageal junctional zone to increases in abdominal pressure. Studies were made on 24 normal subjects and nine patients with large fixed hiatal hernia. Pressures in the stomach, gastroesophageal junctional zone and body of the esophagus were recorded by means of water-filled, open-tipped polyethylene tubes. During each test the pressures in these sites were recorded with and without the application of pressure to the anterior abdominal wall. In the normal subjects, increases in abdominal pressure resulted in a rise in pressure in the stomach and in the segments of the junctional zone located above and below the diaphragm. The maximum increase in pressure in the zone was greater than that recorded in the stomach, indicating that this response was not merely a reflection of changes in intra-abdominal pressure. To determine if the latter was intrinsic to the junctional zone, further studies were made on patients with hiatal hernia in whom the zone was not influenced by the diaphgram. In these subjects, application of abdominal pressure also resulted in an increase in pressure in the junctional zone. As this zone was located in the thorax well above the diaphragm, the response was not a result of diaphragmatic action and could only be one of intrinsic contraction. This evidence confirms the presence of an intrinsic sphincter at the gastroesophageal junctional zone.

The authors wish to thank Dr. J. A. Hildes for his helpful criticisms during the preparation of this paper.

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Résumé

La réaction de la jonction gastro-œsophagienne à l'augmentation de la pression abdominale a été étudiée chez 24 sujets normaux et neuf malades souffrant d'une grosse hernie diaphragmatique irréductible. Les pressions dans l'estomac, la jonction gastro-œsophagienne et le corps de l'œsophage proprement dit ont été au moyen de tubes de polyéthylène remplis d'eau et à bout ouvert. Durant chaque épreuve, les pressions à ces endroits ont été enregistrées avec et sans application de pression sur la paroi abdominale antérieure. Chez les sujets normaux, les augmentations de pression abdominale se sont traduites par une augmentation de la pression dans l'estomac et dans les portions de la zône de jonction situées au-dessus et en-dessous du diaphragme. L'augmentation maximum de pression dans la zône de jonction était supérieure à celle enregistrée dans l'estomac, signe que cette réaction ne reflétait pas seulement les changements de la pression intra-abdominale. Pour établir si cette dernière s'appliquait particulièrement à la jonction, on fit d'autres essais sur des malades porteurs d'une hernie diaphragmatique, donc des malades chez lesquels la zône de jonction n'était pas influencée par le jeu du diaphragme. Chez ces sujets, l'application d'une pression abdominale s'est traduite également par une augmentation de la pression dans la jonction gastro-œsophagienne. Comme cet endroit est situé dans le thorax bien au-dessus du diaphragme, la réaction qui s'y est produite ne résultait pas de l'action diaphragmatique et ne pouvait être qu'une contraction intrinsèque. Ceci confirme la présence d'un sphincter autonome dans la jonction gastro-œsophagienne.

CIRCULATING CANCER CELLS. John D. Griffiths and Alan J. Salsbury. 164 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $8.75.

In the preface this book is said to be an impartial review of the facts relating to the subject of tumour cells in the blood. However, an important element of impartiality is lacking, in that the authors have been themselves involved in the recovery of tumour cells from the blood and are apparently committed to the view that this exercise has some merit. In the last few years serious doubts have been raised about whether the cells so confidently hailed as "tumour" in the re-awakening of interest in this field, are really malignant. The authors take in their stride the disparity between reported percentages of recovery of cells ranging from zero to almost 100%. Scant attention is paid to the fact that the majority of reports have been confined to the study of known malignancies, and the importance of control material is not recognized. Most of the workers in this admittedly highly subjective field have apparently ignored the usually accepted fundamentals of the scientific method. This is all the more surprising in view of the cautionary note signed by many of those concerned, published in the *Journal of the National Cancer Institute* (29: 1023, 1962). This reviewer is also biased, the bias arising from a two-year study of tumour cells in the peripheral blood, in which almost as many "malignant" cells were found in controls as in people with malignancy. Leaving aside the central issue of the accurate identification of these cells by the methods now available, there seems to be no evidence that their presence or absence in the peripheral blood has any prognostic import.

This would all be of little practical importance for surgeons were it not for the fact that elaborate surgical rituals have been built around what is at best, highly questionable experimental evidence. In that this book blandly perpetuates this mythology it does a disservice. It is, however, a good review of the field and has a fairly comprehensive bibliography, which makes it useful to anyone interested in the problem.

In the sections of the book dealing with vascular invasion and metastasis, unsupported sweeping statements abound, e.g. "while thrombosis may well facilitate neoplastic growth along veins, the risk of detachment of tumour cells is considerably reduced". This is a concept of thromboembolism that the reviewer finds novel and unconvincing.

USE OF THE FRACTIONATED CYSTOGRAM IN THE STAGING OF BLADDER TUMOURS

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THE planning of a satisfactory treatment for carcinoma of the bladder depends upon placing the tumour in the proper stage. In addition to the usual investigations, conventional cystograms are sometimes carried out. These frequently give little additional information. This paper will describe a new type of cystography which has been found both simple and informative.

There are several methods of "staging" carcinoma of the bladder. Fig. I illustrates the method proposed by Marshall[1] and Fig. 2 shows the Institute of Urology Classification;[2] the latter in a modified form has been incorporated into the "clinical stage classification" of the International Union Against Cancer. This last method of staging tumours is currently being utilized by many centres. Such wide use will do much to facilitate the exchange of information, and assist in the evaluation of treatment. An example of crude survival rates in carcinoma of the bladder for the various clinical stages is given in Fig. 3.

Wallace[2] has outlined a method of assessing bladder tumours which has gained wide acceptance. It includes urine culture, intravenous pyelography, cystoscopy, biopsy and careful bimanual examination under deep anesthesia. In experienced hands, careful bimanual examination will place the tumour in its proper stage in approximately 80% of cases.

Conventional cystograms are sometimes carried out, but frequently are of limited value. While double contrast examinations using barium sulphate and carbon dioxide[3, 4] demonstrate bladder tumours very satisfactorily, they give no reliable information concerning invasion of muscle. The introduction of gas into the perivesical tissues as well as intravesically[5, 6] has been carried out to determine the degree of invasion. The diagnosis of invasion is based upon thickening of the bladder wall in this latter method.

There has been considerable interest in the use of retrograde arteriography as an adjunct in the staging of bladder tumours.[7-11] There is no doubt that this method can supply additional information. However, the use of this technique is followed at times by complications which include hematoma, hemorrhage, subintimal dissection, aneurysm and thrombosis at the puncture site. These are more likely to occur in patients with extensive arteriosclerosis, not an uncommon finding in the age group in which carcinoma of the bladder occurs. In addition, retrograde arteriography has been combined with perivesical insufflation of air;[12] this, however, is a complex and difficult technique.

The following modification of the conventional cystogram is both simple and informative. Based on a technique devised by Connolly and Wallace,[13] it is useful in distinguisheding between superficial and infiltrating tumours of the bladder. This technique differs from that of Temeliescu[14] in that a constant volume of contrast material is added at each instillation until an amount equivalent to bladder capacity has been instilled. The principal advantage of this

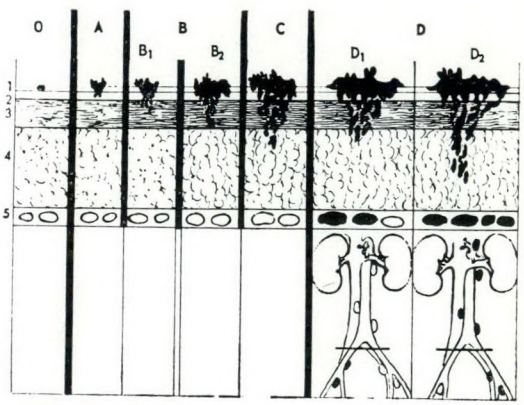

Fig. 1.—Method of staging bladder tumours proposed by Marshall.[1]

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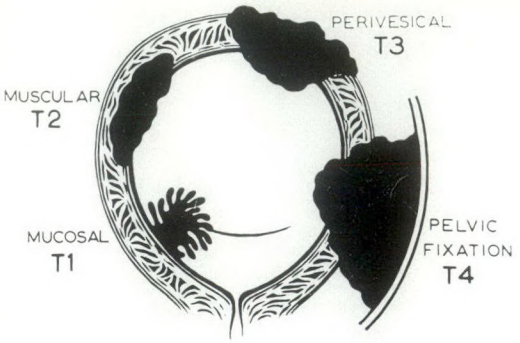


Fig. 2.—Institute of Urology classification of bladder tumours.[2]

technique is its simplicity. It can be carried out without elaborate equipment and contributes to the accurate assessment of the extent of the tumour.

Technique

A soft plastic catheter is passed and the bladder is emptied. The bladder capacity is then measured to determine the total amount of contrast material to be used.

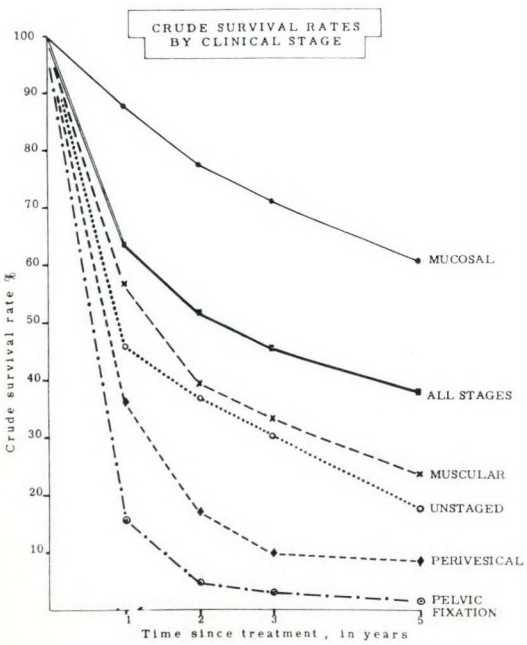


Fig. 3.—Relationship between mortality from carcinoma of the bladder and the stage of the lesion.

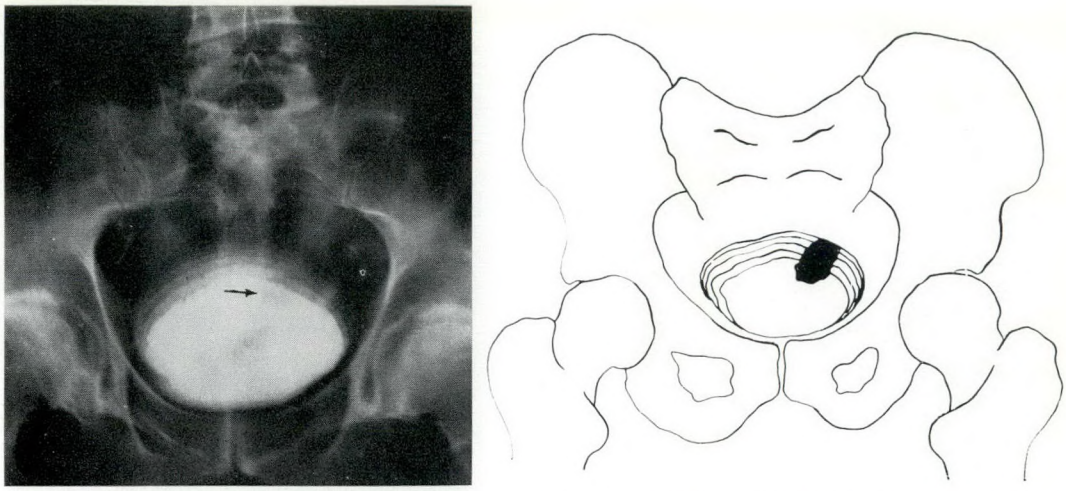
This amount is then divided into four equal increments. Methiodol (Skiodan) in 25% concentration is suitable for this type of cystogram. With the patient absolutely immobile, superimposed exposures are made on the same film following the instillations of these increments. This procedure is repeated for each of the four standard projections. First, an anteroposterior projection is taken with a 10° tilt of the x-ray tube towards the patient's feet. Following this, lateral, right oblique and left oblique views are taken. At the end of the procedure a final film of the whole bladder is exposed and the bladder is then emptied.

Case Reports

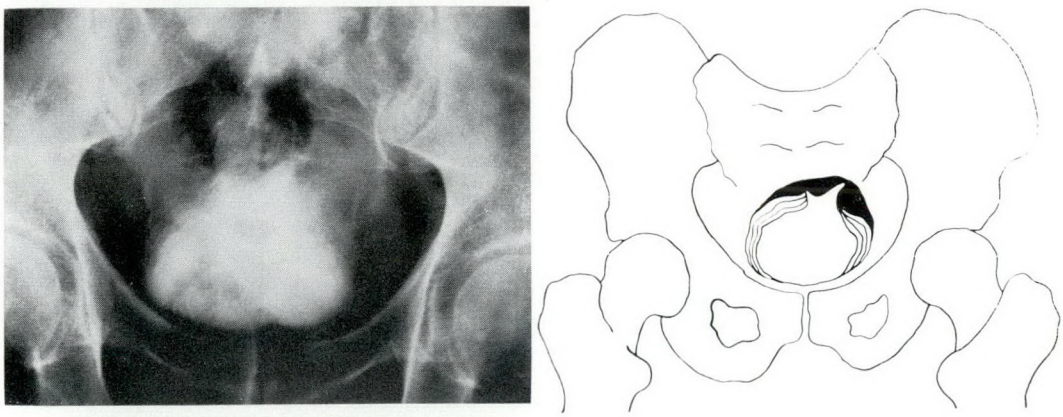
Case 1.—H.S., a 72-year-old man, presented with a three-week history of painless total hematuria. Investigations carried out included an intravenous pyelogram which showed normal upper urinary tracts and a normal cystogram. A fractionated cystogram (Figs. 4a and b) was carried out and showed a small tumour on the left side-wall of the bladder with good mobility of the bladder wall at this site. At cystoscopy a superficial tumour was noted, high on the left lateral wall of the bladder. Histologically this was a low-grade, low-stage lesion.

Case 2.—M.M., a 62-year-old man, was admitted with a diagnosis of recurrent carcinoma of the bladder. On admission there was a palpable suprapubic mass. A fractionated cystogram (Figs. 5a and b) showed marked deformity with fixation of the bladder dome. At cystoscopy the bladder was extensively involved. Bimanual examination was carried out and the lesion was found to be fixed (T4). Histologically it was a highly malignant tumour.

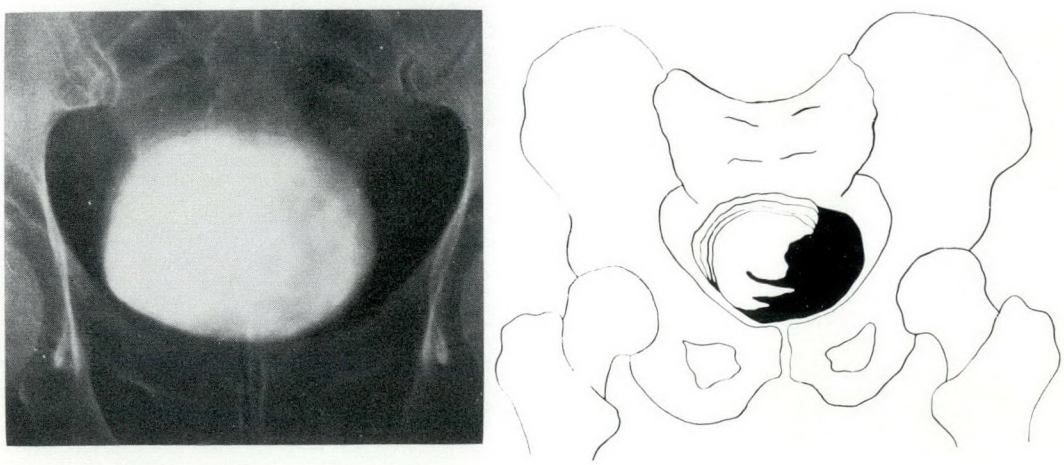
Case 3.—B., a 58-year-old man, was admitted because of painless hematuria. An intravenous pyelogram showed a filling defect in the bladder and a fractionated cystogram was carried out (Figs. 6a and b). This showed a large left-sided bladder tumour with immobility of the bladder wall at this site. At cystoscopy a large papilliferous growth was noted. On bimanual examination the tumour was palpable and mobile. Histologically the lesion was found to be invasive carcinoma of the bladder.



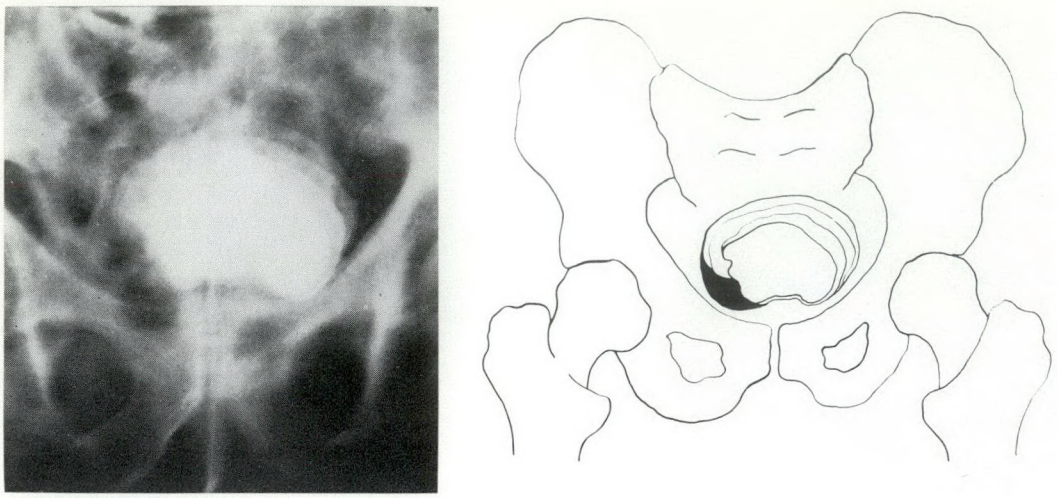
Figs. 4a and b.—H.S., fractionated cystogram showing mobility of bladder wall at site of tumour.



Figs. 5a and b.—M.M., fractionated cystogram demonstrating fixation of bladder dome by tumour.



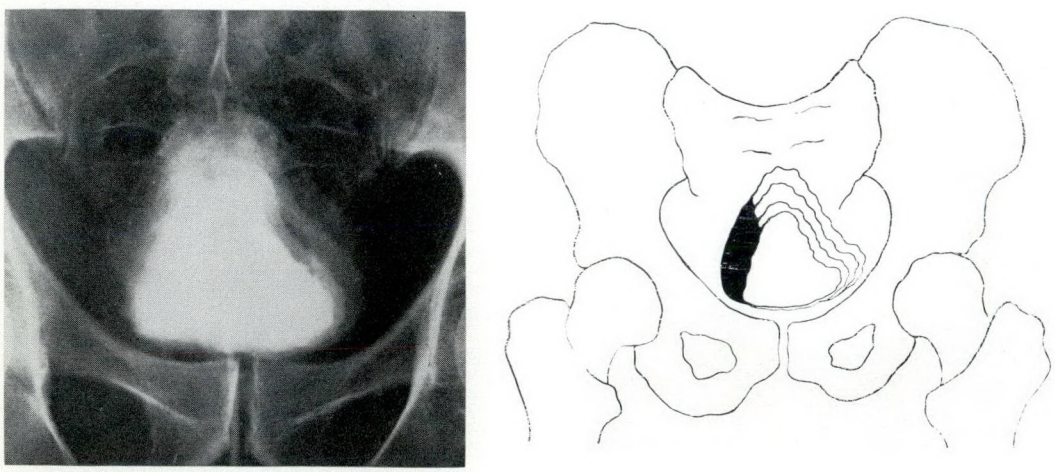
Figs. 6a and b.—B., fractionated cystogram. There is a large tumour on the left side with immobility of the wall.



Figs. 7a and b.—F.G., large tumour on the right side of the bladder with fixation demonstrated by fractionated cystogram.

CASE 4.—F.G., a 71-year-old man, was admitted with painless total hematuria. An intravenous pyelogram showed some asymmetry of the right side of the bladder. A fractionated cystogram (Figs. 7a and b) showed a large tumour of the right side of the bladder with fixation of the bladder wall. At cystoscopy there was an extensive infiltrating lesion involving the right wall of the bladder. On bimanual examination, it was fixed.

CASE 5.—R.N., a 62-year-old man, was admitted because of painless total hematuria. A conventional cystogram showed a deformity of the right side of the bladder. A fractionated cystogram (Figs. 8a and b) again demonstrated a deformity and also showed the bladder wall to be immobile. At cystoscopy a large infiltrating tumour of the right side of the bladder was noted.



Figs. 8a and b.—R.N., deformity due to a large infiltrating tumour. Fractionated cystogram showing immobile bladder wall on the right side.

Discussion

The principle underlying the technique is that once the tumour has spread into the muscle of the bladder, the wall at this site becomes relatively immobile. Thus, while the surrounding bladder wall would expand with successive instillations of the contrast material, the site at which the bladder is infiltrated by tumour remains relatively fixed.

The interpretation of the fractionated cystogram must be correlated with the clinical findings. Bladder fixation may be associated with various vesical or extravesical inflammatory conditions. A bladder which has been subjected to previous operations may be deformed. In addition, extravesical tumours may give a false positive result. Obviously the technique is of little value in the small, contracted, or hyperirritable bladder.

It is important to obtain multiple projections to prevent overlap of the tumour site by normal bladder. If normal bladder overlies the area, movement will be seen suggesting that the tumour is superficial.

Careful clinical examination is most important for the staging of bladder tumours and no radiological investigation can replace these steps. Thus the technique must be considered a supplementary diagnostic procedure only. It is felt that this technique is of value, however, in the assessment of tumours which are infiltrating bladder muscle. The method is simple. It is relatively innocuous and carries little or no morbidity.

Summary

A simple method for evaluation of non-infiltrating and infiltrating bladder tumours has been described. It is based upon the principle that infiltrating tumours cause fixation of the bladder wall. While clinical examination remains the most important method for staging bladder tumours, the fractionated cystogram can add another parameter to the assessment of the individual case.

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Résumé

Un examen clinique approfondi permet d'évaluer avec précision (dans la proportion de 80%) l'envahissement des tumeurs vésicales. En certains cas, la cystographie peut être utile. L'article décrit un type de cystogramme qui s'est révélé à la fois simple et instructif. Après instillation de quatre portions égales d'une substance contraste, on prend, sur le même film, des images superposées. Cette technique s'inspire du principe que la paroi vésicale est fixe à l'endroit des tumeurs. Les auteurs insistent sur le fait qu'un examen clinique attentif reste la méthode de choix pour l'évaluation de l'ampleur des tumeurs de la vessie. Ils estiment cependant que les cystogrammes fractionnés peuvent constituer une source de renseignements supplémentaires.

OBSERVATIONS ON STRAIGHT LEG-RAISING WITH SPECIAL REFERENCE TO NERVE ROOT ADHESIONS

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STRAIGHT leg-raising, as used in orthopedic examinations, has long been recognized as being of value in the diagnosis of disc protrusions involving one of the roots of the sciatic nerve. However, discussions of this test with both students and clinicians reveal that it means different things to different people. Its significance appears at times to become clouded by confusions with the Kernig test and Lasague's test, and the very specific implications of a positive straight leg-raising test, are sometimes inadequately appreciated in complicated clinical problems.

In the light of the literature on the subject, such confusion is not justified. The anatomical factors involved in this test have been clearly demonstrated by Inman and Saunders[4] in 1942 and by Falconer, McGeorge and Begg[3] in 1948. In 1959 Charnley[2] carried out further anatomical studies, reviewed the literature and discussed the test in detail, elucidating the significance of the test and indicating exactly how it can be used in diagnosis.

THE MECHANISM OF THE TEST (Fig. 1.)

With the patient lying in the supine position, the lower extremity of the limb to be tested is gently lifted on the palm of the hand and slowly elevated, flexing the hip while maintaining full extension of the knee. One can consider the tibia and femur with the knee joint locked in extension as a solid lever moving around a fixed point in the centre of the hip joint. The sciatic nerve lying loosely in the soft tissues, is attached variously in the distal portion of the limb in the structures to which it is distributed. The bony lever is elevated to an angle of from 30° to 40° before the slack in the peripheral arborization of the sciatic nerve is taken up. When the slack is taken up (and *not* before) traction is exerted on the nerve roots at the intervertebral foramina and above. In the normal state, the roots then commence to move. The excursion of this movement in the cadaver according to different observers varies from 4 to 8 mm. The rate of the movement is maximal at its commencement and two-thirds of the excursion is completed by the time the angle has reached 60°. The rate of movement then rapidly shades off so that very little movement of the root takes place at 70°, much like the slowing of a piston as the crank-shaft approaches dead centre.

APPLICATION OF THE TEST

The clinical significance of the test thus depends on the phase of movement in which the spasm becomes evident.

(1) If some other structure, notably a protruded portion of an intervertebral disc, is pressing on a sciatic nerve root while the test is being done the protective spasm will immediately arise when the slack is taken up in the peripheral arborization of the nerve and traction exerted on the root, i.e. at about 35°. If such a reaction does not take place at this angle then one can say with assurance that there is *no* pressure on any sciatic nerve root, *in which sensory fibres are left intact.*

(2) Pain and spasm may arise during the phase of movement when the nerve root is being dragged through the intervertebral foramen and the intraspinal portion of the root in its root sleeve is being dragged distally towards the foramen over the adjacent intervertebral disc. This reaction could be produced by the presence of an adhesion binding the nerve root to the disc or some other neighbouring structure. Thus a very dense and tight adhesion would produce its reaction very shortly after the 35° angle when the nerve root had barely started to move, whereas a looser adhesion would allow more movement, perhaps to 60° or 70°, before its tightening bands would arrest the nerve in its gliding course.

(3) After the angle of 70° has been

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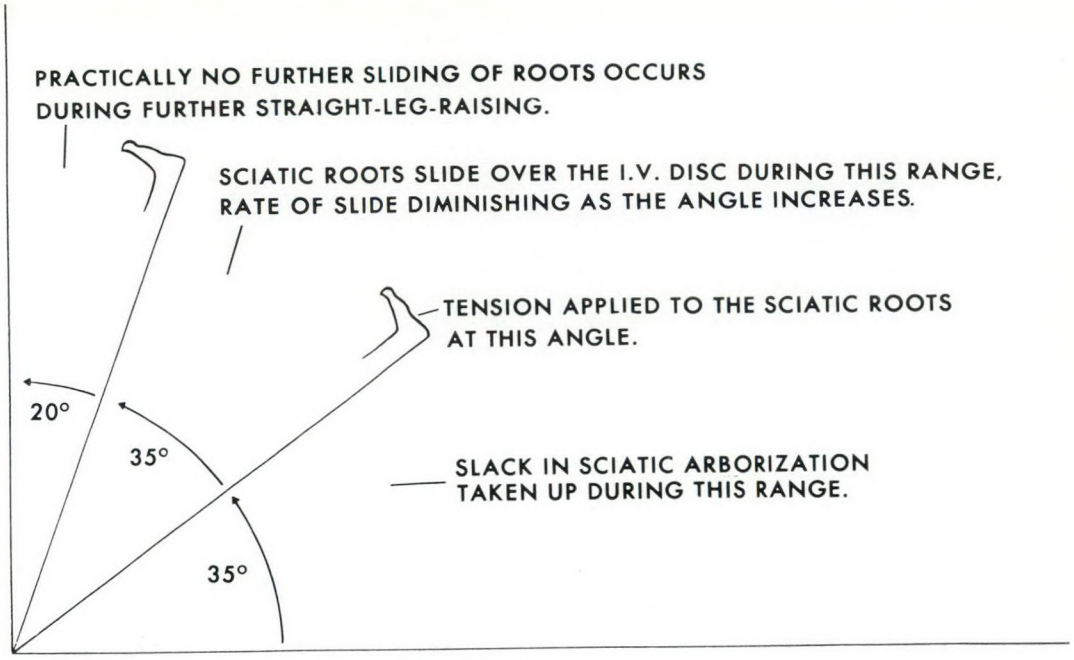

Fig. 1.—Dynamics of the straight leg-raising test.

reached, there is practically no further excursion in the sliding movement of the sciatic nerve root and therefore pain or spasm brought on in this range (i.e. from 70° to 90°) *must be from some other cause.*

Technique of the Test

The straight leg-raising test is sometimes rendered valueless by moving the limb too abruptly, too quickly, or forcing it against voluntary protective tension. These can easily produce inaccurate readings of the angle. A clear concept of the anatomical factors involved while the test is being carried out, however, should persuade any examiner of the necessity for a careful and gentle handling of the limb. The patient should be told that the limb will be lifted with the knee straight and that it will be lifted slowly, preferably without his (the patient's) assistance. He should be told that as soon as he indicates any pain the passive elevation will cease. Several trials may be necessary in this way before the examiner can assure himself that the limb is properly relaxed for the test. Spasm will, of course, be evident to the examining hand as soon as it commences and at this point it is essential to push the limb a little further in order to assess the strength of the spasm and, at the same time, feel the pelvis with the other hand. No useful information will be gained by forcing the limb up further, and the overzealous examiner may cause a serious setback in the patient's condition by this maneuver. Some patients, whose pain is not too severe, may go on up to 70° or 80° in this way by flexing the lumbar spine thus giving a grossly inaccurate reading of the angle, if the point of commencement of pelvic rotation has not been accurately observed.

Nerve Root Adhesion Simulating Disc Protrusion

Three patients have been encountered with classical symptoms and signs of disc protrusion, and consistently positive myelographic findings. No protrusion was found at operation but the nerve root was densely adherent to the disc. The nerve root in each was released but the disc was left intact. Full and lasting relief was obtained in each case.

Case Report.—In 1961, M.C., presented with right sciatic pain of four months' duration, which was of gradual onset following a

lifting strain. On examination, he had paravertebral spasm and marked limitation of forward flexion. On straight leg-raising, the limb went to 30° on the right with hamstring spasm, but to 80° on the left. The right ankle jerk was absent. The right first sacral dermatome was dull to pinprick.

He was treated with two weeks' rest at home with active flexion exercises without improvement. He also had four days' bed rest in hospital without improvement. Radiographic examination was negative but a myelogram showed a disc-level defect at lumbosacral level on the right side.

At operation at the lumbosacral level, the first sacral nerve root was found to be densely adherent to the underlying disc. On separating the adhesion a hole was evident in the posterior part of the disc, the nerve root having become adherent to the border of the hole. There was no protrusion and the opening in the disc was found to be very shallow. No disc material was removed.

In an effort to prevent reformation of tight adhesions postoperatively, the sliding movement of the root produced by the raising of the straight leg was utilized. In so doing it was soon evident that active or passive straight leg-raising was often too painful to be acceptable, or tolerated, when done in the usual way. A pulley and sling were therefore set up from a Balkan beam (Fig. 2) to allow the patient to elevate his own leg passively. He was instructed to raise it only to the point of pain, and at that elevation to loop the rope over the hook so that the angle could be maintained while he relaxed. Five minutes or more later, if his pain had eased, as was usually the case, he would pull the leg up again to the point of pain and again hold it in a fixed position. This process would go on for one-half hour or more and be repeated three or four times if easily tolerated. Within two to six days an angle of 80° to 90° was achieved painlessly.

After the patient is discharged from hospital it is usually quite feasible to continue the leg-raising actively. This is only necessary once each day, at which time the full

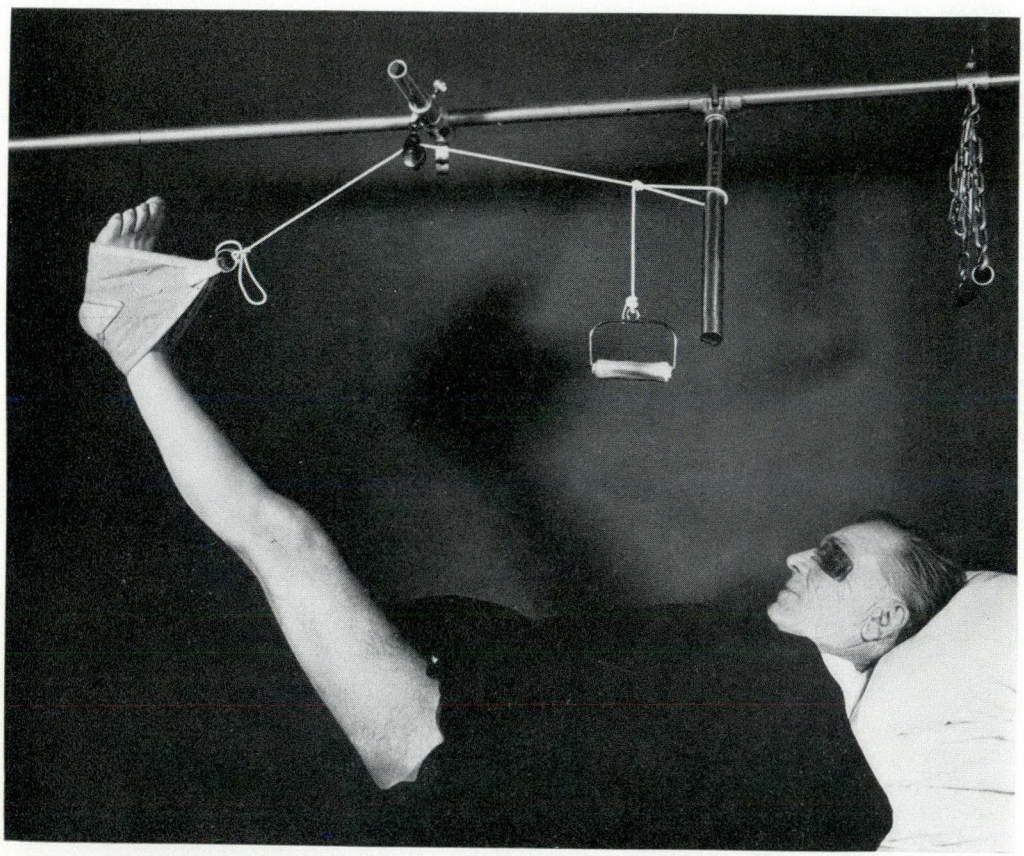

Fig. 2.—Pulley and sling arrangement for passive straight leg-raising

elevation is maintained for approximately 10 seconds, rather than repeated elevations which may be very irritating and defeat the purpose. The patient is advised to continue this daily movement permanently. On follow-up, this patient has gone two years without recurrence of his symptoms.

Two other similar patients, with shorter follow-ups, have been seen, each of whom had a classical history, and symptoms and signs of disc protrusion including straight leg-raising of 35° with hamstring spasm. Each one had a firm adhesion of the nerve root to the underlying disc at operation with no evidence of disc protrusion. Each had full recovery following operation at which the adhesion was released without interference with the disc.

Both these patients were instructed to carry out a single straight-leg elevation once daily for an indefinite period. Each has been symptom-free on follow-up over a 12-month period.

None of these three patients had any history of previous spinal surgery. In each case, the preoperative diagnosis was intervertebral disc protrusion and, in retrospect, there appears to be no way in which a diagnosis of this type of adhesion can be made before operation.

Postoperative Nerve Root Adhesions

Following operations for disc protrusion, patients occasionally complain of mild recurrent sciatic pain. This usually remains mild and recurrent but occasionally becomes progressively more severe and resistant to treatment. It is sometimes associated with lancinating pains in the same or other areas and may be complicated by electric shock-like sensations, and scalding or burning pain which may initially be related to activity but later become constant as the symptoms worsen.

Excluding a new disease process, the diagnosis in this situation lies between: (1) increasing disc degeneration: (2) recurrent disc protrusion; and (3) nerve root adhesions at the old operative site. The first two lesions can be recognized following the application of the usual methods. The diagnosis of nerve root adhesions may be greatly assisted by the process of exclusion, if the first two can be ruled out. The diagnosis of nerve root adhesions is consistent with the finding of a straight leg-raising test which produces pain and spasm at any point between 35° and 70°.

If, on exploration, nerve root adhesions are found and released, their recurrence may be prevented by repeated but gentle stretching by straight leg-raising. It is most likely that, rather than preventing the reformation of adhesions, this movement simply keeps the adhesion in a sufficiently loose state so that it does not produce any painful symptoms.

Case Report.—This man was seen on January 11, 1965, complaining of bilateral sciatic pain, which was worse on the right. He also had low back pain. A history was obtained of L4-5 disc protrusion on the right side treated successfully by operation in January, 1962. He was troubled by relatively mild attacks of sciatic pain thereafter, successfully treated by rest. In July 1964, this pain gradually worsened and became constant, aggravated by any movement. Examination showed marked limitation of movement of the spine with associated paravertebral spasm. Straight leg-raising was tolerated to 45° on the right, and 55° on the left. Motor power and sensation were normal. His reflexes were also normal. A myelogram was performed and showed an irregular hour-glass type of defect at the L4-5 level.

Laminectomy was carried out at L4-5 level using a bilateral approach. The structures on the left side were normal but on the right, sheets of white fibrous tissue were encountered in the extradural space densely binding the dura to the inner wall of the spinal canal and to the nerve root. The dura and the L4-5 nerve root were separated from each other and from the inner wall of the spinal canal. A small amount of the fibrous tissue was removed for this purpose. The intervertebral disc was flat and firm, and was not interfered with. Postoperatively the patient used a passive straight leg-raising sling and achieved 90° elevation within three days. There has been no recurrence of pain on eight months' follow-up.

Two further patients with complaints of similar type have been operated upon. In each, the diagnosis of disc protrusion was made because straight leg-raising was possible only to 35° and myelogram indicated a defect at the appropriate level. Opera-

tion, however, revealed only adhesions similar to those described above, and the disc was flat and firm. Postoperatively, straight leg-raising in these patients was very painful but, when done slowly and passively by pulley and sling, the limb was brought readily to 90° within 48 hours.

Conclusion

It is known that nerve root adhesion may develop following operative removal of an intervertebral disc. Using the postoperative leg-raising routine, described in this communication, prophylactically would prevent the development of such adhesions.

Three cases have been presented to demonstrate that nerve root adhesions may simulate intervertebral disc protrusion, and that recurrence of the adhesion after separation can be prevented. Three other cases indicate that recurrence of postoperative root adhesions after operative separation may be similarly prevented.

The use of straight leg elevation in diagnosis and treatment is discussed. The rationale of an exercise to prevent the formation of sciatic nerve root adhesions is also described. The hope is that thereby postoperative courses of exercise can be more efficiently applied and unnecessary (and possibly irritating) exercises eliminated.

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Résumé

Pour faire le diagnostic de compression nerveuse par hernie d'un disque intervertébral, l'élévation de la jambe est un test utile et reconnu depuis longtemps. Le test d'élévation de la jambe met en œuvre un mécanisme très bien décrit et basé sur des notions anatomiques précises. La jambe, lorsque élevée à un angle de 30°, met le nerf sciatique sous tension. Dans l'anatomie normale, si l'on continue d'élever le membre, les racines du nerf sciatique commencent alors à se mobiliser. Lorsque rendu à un angle de 60°, la mobilité du nerf et de ses branches est complète et un mouvement dans un angle plus élevé ne provoque aucun mouvement des racines nerveuses. En clinique, ce test est utile comme moyen de diagnostic différentiel dans trois catégories de lésions.

1. Une hernie discale, comprimant une racine nerveuse, provoquera un spasme protecteur réactionnel entre 30° et 35° d'angle d'élévation.

2. Les adhérences qui fixent un disque ou les structures du voisinage à la racine nerveuse; le spasme réactionnel se produit entre 35° et 60°.

3. La cause doit être cherchée ailleurs que dans le disque, si on obtient un spasme musculaire secondaire après un angle de 70° d'élévation.

L'auteur relate ensuite trois cas dont le diagnostic d'hernie discale était basé sur des signes et des symptômes classiques. A l'opération il n'existait pas de hernie, mais plutôt des adhérences fixant la racine nerveuse sciatique. Dans les suites postopératoires, l'auteur a utilisé le test d'élévation de la jambe comme moyen prophylactique pour prévenir les adhérences consécutives à l'intervention. Il a inventé un appareillage permettant au malade de faire lui-même l'élévation passive de son membre. Ainsi progressivement le malade finit facilement et sans douleur par élever son membre jusqu'à un angle de 90°. En conclusion, l'auteur signale que cet exercice devrait être utilisé dans les suites post-opératoires après une opération pour disque intervertébral. L'élévation du membre étendu, servirait donc à la fois comme moyen de diagnostic et comme moyen de traitement.

LES RESULTATS LOINTAINS DE LA CHIRURGIE POUR L'ULCERE DE L'ESTOMAC ET DU DUODENUM*

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LA maladie ulcéreuse intéresse le monde médical, depuis Hippocrate qui en fit une description remarquable.

A travers l'histoire médicale, divers auteurs en ont fait des descriptions sommaires, jusqu'à ce que Abercrombie, en Angleterre (1828), et Cruveilhier, en France (1829), apportent une étude clinique adéquate de cette entité pathologique.

Lorsque Billroth, dans les 1880, eut démontré ses techniques de résection gastrique, les indications opératoires des ulcères de l'estomac et du duodénum furent mieux étudiées et standardisées. Aussi, dans le même temps les physiologistes avaient attiré l'attention sur la nature des sécrétions gastriques, plus particulièrement la forte teneur en acide chlorhydrique, à qui on attribuait déjà, de même qu'à la pepsine, un rôle important dans la génèse des ulcères.

C'est ainsi qu'après la première guerre mondiale, la gastro-entérostomie fut préconisée comme l'opération de choix dans le traitement chirurgical des ulcères gastro-duodénaux; en raison de sa facilité technique, de son taux de mortalité peu élevé et aussi parce que l'on envisageait le passage rapide de la sécrétion acide de l'estomac vers le milieu alcalin du jéjunum comme la solution physiologique la plus rationnelle.

Cependant, comme on le sait, des complications survinrent et surtout la récidive de l'ulcère, généralement au voisinage de l'anastomose, soit sur le versant gastrique, soit sur le versant jéjunal: ulcus peptique ou ulcus marginal.

Les chirurgiens, en face d'une indication chirurgicale, optèrent alors pour la gastrectomie subtotale aux deux tiers (2/3) ou aux trois quarts (¾), qui emporte l'antre et une bonne partie de la zone sécrétante de l'estomac.[4, 55] Les résultats furent immédiatement jugés très bons, puisque le nombre des récidives d'ulcères peptiques ou marginaux fut considérablement diminué; néanmoins, certaines complications vinrent assombrir le tableau telles que les troubles de la nutrition, les états anémiques, le syndrome du "dumping", etc.[6, 9, 14, 16, 31, 32, 38, 42]

Les travaux de Pavlov du début du siècle sur les diverses phases de la sécrétion gastrique furent repris par plusieurs écoles et amenèrent finalement Dragstedt[11] à scruter expérimentalement ce problème. Il préconisa la section des deux pneumogastriques afin d'éliminer la phase céphalique de la sécrétion gastrique, à qui il attribua, dès ce moment, une part importante dans la pathogénie de l'ulcère du duodénum.

Il s'ensuivit cependant des troubles d'évacuation dûs à la disparition de la motilité gastrique. Il devint évident qu'il fallait ajouter à la vagotomie bilatérale une voie d'évacuation gastrique. L'adjonction de la gastro-entérostomie ou de la pyloroplastie rendit à la vagotomie sa valeur.

Au cours des années 1940-1960, période sur laquelle porte ce relevé clinique des opérations gastriques pour ulcères à l'Hôtel-Dieu de Québec, les opérations les plus en faveur furent la gastrectomie subtotale et la double vagotomie avec gastro-entérostomie.

Il faut cependant noter que durant la dernière décennie d'autres interventions[11, 41, 43, 48, 50, 53, 54] furent préconisées et gagnèrent graduellement la faveur des chirurgiens, puisqu'elles respectent mieux la physiopathologie de l'ulcère de l'estomac et de l'ulcère du duodénum: la vagotomie avec pyloroplastie, la gastrectomie partielle,[13] l'antrectomie combinée, à la vagotomie double ou à la vagotomie sélective.[21] Cette dernière permet d'éviter les effects nocifs de la vagotomie complète,[44] sur divers viscères abdominaux comme le pancréas et la vésicule biliaire.

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Matériel clinique de l'Hôtel-Dieu de Québec entre les années 1940-1960

Durant ces 20 années de chirurgie gastrique pour ulcus de l'estomac et du duodénum, 845 malades furent opérés; 542 furent retracés, dont 186 étaient porteurs d'ulcère de l'estomac, 333 porteurs d'ulcère du duodénum et 24 souffrant d'ulcère double et du duodénum et de l'estomac (Tableau I).

TABLEAU I.

Malades	*Opérés*	*Retracés*	*Sexe M.*	*Sexe F.*
Ulcères de l'estomac...	300	186	151	35
Ulcères du duodénum...	516	333	298	35
Ulcères doubles	29	24	21	3
Total.....	845	543		

La chirurgie des ulcères de l'estomac

Le Tableau II montre que sur 300 opérations pour ulcères gastriques, la technique suivie a été la gastrectomie subtotale (66 à 75%) dans 92% des cas, la majorité étant du type Billroth, 2e manière: soit des Polya simples durant les années 1940 à 1948, soit des Polya-Hoffmeister transmésocoliques postérieurs durant les années subséquentes.

Quelques malades furent traités par vagotomie et autres techniques pour des raisons ancillaires, comme l'âge, soit peu avancé, soit très avancé, comme le mauvais état général du malade, ou encore un très mauvais équilibre psychique, ou en présence de maladies chroniques concomittantes qui détournèrent le chirurgien du traitement classique habituel.

TABLEAU II.—La chirurgie des ulcères de l'estomac

Opérations: 300	*M.*	*F.*	*Total*
Excisions simples.....	4	1	5
Excisions et gastro-entérostomie.....	1		1
Gastrectomies subtotales (66 - 75%)			
92% Billroth I.....	13	7	20
92% Billroth I et vagotomies....	2		2
92% Billroth II.....	212	39	251
92% Billroth II et vagotomies....	4		4
Gastrectomies totales.....	3	1	4
Vagotomies et gastro-entérostomies...	7	2	9
et pyloroplasties.....	3	1	4

La chirurgie des ulcères du duodénum pendant cette période a porté sur 516 malades. On constate ici l'influence qu'a jouée Dragstedt dans l'évolution de la chirurgie de l'ulcère du duodénum. En effet, 68% des malades eurent des résections gastriques et 28% furent traités par la vagotomie seule ou associée à des opérations favorisant l'évacuation gastrique. Tableau III permet en même temps de constater le faible pourcentage de femmes ayant subi un traitement chirurgical pour ulcus duodénal.

TABLEAU III.—La chirurgie des ulcères du duodénum

Operations: 516	*M.*	*F.*	*Total*
Excisions simples.....	2	1	3
Gastro-entérostomie.....	1		1
Gastrectomies subtotales (66-75%)			
68% Billroth I.....	16	5	21
68% Billroth I et vagotomie.....	1		1
68% Billroth II.....	301	30	331
68% Billroth II et vagotomies....	2		2
Gastrectomies totales.....	1		1
Gastrectomies (50%) antrectomies.....	1	1	2
Vagotomies.....	5	1	6
28% et gastro-entérostomies....	127	13	140
28% et pyloroplasties....	4	3	7
28% et gastro-entérostomie et pyloroplastie.....	1		1

Les résultats lointains

Afin de bien apprécier et d'étudier les résultats obtenus par ces interventions chirurgicales pour ulcus, nous avons convenu de les répartir en trois catégories (Tableau IV):

TABLEAU IV.—Les critères d'appréciation des résultats lointains de la chirurgie pour ulcères de l'estomac et du duodénum

1—Résultats excellents—
Etat de santé normal, vie normale.

2—Résultats bons—
Présence d'au moins un symptôme et/ou un signe important mais poursuite d'une vie normale.

3—Résultats médiocres—
Les symptômes et/ou les signes présents sont importants et rendent toute vie normale impossible.

Des 543 malades retracés qui avaient subi une intervention pour ulcus gastro-duodénal, 66 malades ont été classés comme ayant eu des résultats médiocres, c'est-à-dire mauvais.

Si l'on étudie ces 66 cas de plus près, l'on peut mettre en évidence les symptômes dont ils se plaignent et les signes cliniques les plus fréquents. La Fig. 1 montre que la douleur est le symptôme dont se plaint le plus grand nombre de malades, soit dans 74% des 66 cas.

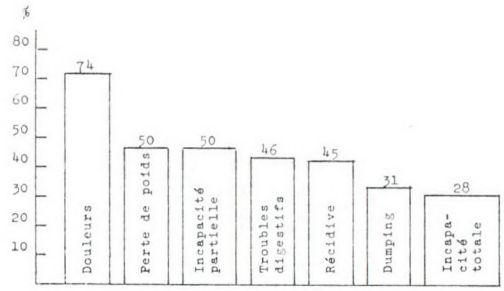

Fig. 1.—Résultats médiocres: 66 cas. Symptômes et signes les plus fréquents.

On peut penser que cette douleur, de type ulcéreux la plupart du temps, soit due, dans la majorité des cas, à une hyperchlorydrie persistante, ou à une récidive dans les suites post-opératoires plus ou moins tardives. D'ailleurs dans une bonne proportion des cas, soit 45%, une récidive réelle, radiologiquement visible, a été constatée.

La perte de poids vient en second, après le symptôme de douleur. Elle paraît être en rapport avec une nutrition déficiente, soit par récidive (45% des cas) soit par troubles digestifs (46% des cas).

Il faut aussi signaler le syndrome de "dumping" que nous avons rencontré dans 31% des cas. Il s'agit de cas présentant un dumping classique, se manifestant soit par une asthénie post-prandiale avec bouffées de chaleur, et hypotension artérielle, soit par des épisodes de sensation de plénitude épigastrique et même des épisodes de diarrhée.

Il est évident que ces troubles entraînent chez nos 66 cas de résultats médiocres, une incapacité plus ou moins importante dans la conduite normale de leur vie. En effet, nous avons eu 50% des cas d'incapacité partielle et 28% des cas d'incapacité totale.

Si l'on essaie de répartir nos échecs selon le site des ulcus et selon le type d'opérations pratiquées, nous pouvons possiblement arriver à une meilleure compréhension des causes des mauvais résultats obtenus, de façon à pouvoir tirer des conclusions utiles pour l'avenir.

Ainsi, les gastrectomies subtotales Billroth, 2e manière pour ulcères gastriques, ont donné 7% de mauvais résultats. Les chiffres pour les autres types d'opérations n'ont aucune valeur statistique significative puisqu'ils totalisent un nombre de cas insuffisant (Tableau V).

Il s'agit en effet de 12 cas ayant eu soit un Polya simple (sept cas) soit un Polya-Hoffmeister (cinq cas). Ces patients ont manifesté les troubles suivants: Dans le groupe d'ulcus de l'estomac, ayant été traités par une gastrectomie subtotale de 66 à 75%, il y eut sept cas classés médiocres et jugés ainsi pour les raisons suivantes: Six des patients ont continué de souffrir de leur estomac; trois ont eu une perte de poids très importante; trois se sont plaints de troubles digestifs marqués; et deux ont offert le tableau classique du dumping. A remarquer qu'il n'y eut pas de récidive d'ulcus parmi ces cas.

Du groupe d'opérés ayant eu un Polya-Hoffmeister, cinq ont été jugés comme étant de mauvais résultats: les cinq ont continué à se plaindre de douleurs; quatre n'ont pu refaire leur poids initial; deux ont présenté un syndrome de dumping, et remarquons qu'il y eut deux cas de récidive radiologiquement diagnostiqués.

Ces deux types d'opération ont donc don-

TABLEAU V.—Les ulcères d'estomac

Résultats éloignés: Excellents..... 56%
Bons..... 39%
Médiocres..... 8% - 19 cas

	Total	*Résultats médiocres*	%
Gastrectomies subtotales:			
Billroth I.....	14	3	21%
Billroth II.....	162	12	7%
Gastrectomies totales.	4	1	25%
Vagotomies:			
et gastro-entérostomies....	7	2	30%
et pyloroplasties...	4	1	25%

né comme troubles morbides, des tableaux à peu près identiques.

Si d'un autre côté, nous étudions maintenant les résultats médiocres dans les cas d'ulcères du duodénum, nous constatons que nous pouvons statistiquement apprécier deux catégories d'opérés: ceux qui ont eu une gastrectomie subtotale, type Billroth II; et ceux à qui on a fait une vagotomie bilatérale avec gastro-entérostomie.

TABLEAU VI.—Les ulcères du duodénum

Résultats éloignés: Excellents..... 56%
Bons..... 31%
Médiocres..... 12% - 13 cas

	Total	*Résultats médiocres*	%
Excisions.....	3	1	33%
Gastrectomies subtotales (66 - 75%)			
Billroth I.....	14	5	35%
Billroth II.....	226	23	10%
Vagotomies et gastro-entérostomies....	89	7	7%
et pyloroplasties...	6	2	33%

Respectivement ces deux catégories montrent pour la gastrectomie subtotale, Billroth II, une proportion de 10% de mauvais résultats et pour la vagotomie avec gastro-entérostomie, 7% des résultats médiocres (Tableau VI). Il va sans dire que ces deux chiffres, 10% et 7%, ne sont pas très éloignés l'un de l'autre et confirment les travaux de Dragstedt qui préconisait dès 1943, cette intervention.[11] Il n'en demeure pas moins que cette proportion soit élevée, prouvant ainsi qu'un bon nombre de malades restent handicappés après cette chirurgie.

Quelles sont les constatations faites chez ces malades — 23 gastrectomisés et sept vagotomisés?

Des 23 cas de Billroth II classés comme résultats médiocres, 17 ont continué de souffrir; 12 avaient des troubles digestifs; 13 n'ont pas regagné leur poids initials. Il y eut neuf cas de "dumping" et 10 récidives.

Par ailleurs, des sept cas de mauvais résultats après vagotomie, cinq ont continué de se plaindre de douleurs, cinq ont fait une récidive, deux ont offert le tableau d'un dumping.

Ces constatations montrent donc que les mauvais résultats sont du même ordre à la suite des deux types d'opérations. Cependant, la pathologie *cible* de l'hyperchlorhydrie peut fort bien être causée par des processus physiopathologiques différents, dépendant des lieux d'origine de la stimulation de la sécrétion acide.

Etudes physiologiques des diverses opérations

Il convient maintenant de revoir les bases physiologiques qui justifient ou justifiaient pendant la période de 1940 à 1960, les interventions chirurgicales pratiquées; et aussi d'apprécier pourquoi les opérations conseillées plus récemment sont plus rationnelles et devraient donner de meilleurs résultats lointains (Tableau VIII).

A—La vagotomie avec gastro-entérostomie annule complètement la phase céphalique de la sécrétion gastrique, mais ne contrôle pas la phase antrale. Bien plus, cette dernière peut être exagérée,[45] soit par l'alcalinisation continue, par médica-

TABLEAU VII.—66 cas médiocres — troubles

Type d'opération	*Douleurs*	*Dumping*	*Perte de poids 10 lbs**	*Récidive*	*Troubles digestifs**	*Capacité de travail*	
						Partie	*Nulle*
Vagotomie.....	2	—	1	2			1
Vagotomie + gastroentérostomie	7	3	5	6	4	3	2
Vagotomie-pyloroplastie.....	4	1	—	2	4	3	1
Vagotomie + gastrectomie partielle.....	1	—	—	—	1	1	—
Excision ulcère.....	2	—	1	1	1	1	—
Billroth I.....	4	3	5	6	2	6	—
Billroth II.....	28	14	21	13	17	18	15
Gastrectomie totale.....	1	—	—	—	—	1	—
Total.....	49	21	33	30	29	33	19

*Troubles digestifs: Diarrhée, vomissements.

TABLEAU VIII.—La vagotomie avec gastro-entérostomie

Ulcère	Phase céphalique: nil
	Phase antrale { Rétention gastrique / Alcalinisation
	Phase intestinale
Dumping	Transit rapide
	Distention jéjunale

tion de l'antre, soit par la stagnation alimentaire passagère dans l'antre. Par ailleurs, les troubles de l'anse jéjunale restent possibles, comme le syndrome de l'anse afférente, le dumping avec distention de l'anse jéjunale et indirectement l'exagération de la sécrétion antrale par stimulation augmentée d'origine entérique, encore dite "phase intestinale".

B—La vagotomie avec pyloroplastie[49, 58] peut donner exactement les mêmes complications, pour les mêmes raisons, sauf que le passage des aliments à travers le duodénum peut stabiliser la sécrétion antrale en raison de son rôle inhibiteur probable.[27, 39] Cependant, un transit rapide, même avec dumping, peut survenir et stimuler l'antre par la "phase dite intestinale" (Tableau IX).[12, 15, 47]

TABLEAU IX.
La vagotomie avec pyloroplastie

Phase céphalique: nil	
Phase antrale	{ Rétention gastrique / Alcalinisation
Transit pratiquement normal parfois rapide.	

C—La gastrectomie (66-75%) Billroth — 1ère manière, est théoriquement une bonne opération qui n'enlève pas cependant la phase cépalique; quoiqu'elle emporte l'antre et une bonne partie de la masse des cellules sécrétantes de la muqueuse gastrique (Tableau X).

TABLEAU X.—La gastrectomie (66 - 75%) et Billroth I

Phase psychique: Hcl
Insuffisance du réservoir gastrique
Transit rapide
Distention jéjunale
Phase intestinale

Les troubles surviennent par insuffisance du réservoir gastrique et persistance de la phase céphalique, par l'existence dans certains cas d'un transit rapide provoquant une distention jéjunale, un syndrome de dumping, et possiblement une stimulation intestinale (phase intestinale) au niveau de la masse sécrétante conservée.

D—La gastrectomie (66-75%) et Billroth 2e manière (Tableau XI): Cette intervention qui pendant des années a été l'intervention classique pour ulcus, laisse, comme nous venons de le voir, des séquelles désagréables et pathologiques dans 7% à 10% des cas; cette intervention a donc des défauts marqués. L'étude physiologique permet de constater les faits ou états suivants:

TABLEAU XI.—Gastrectomie (66 - 75%) et Billroth II

Phase psychique: Hcl
Insuffisance du réservoir
Distention jéjunale
Reflux duodénal (Phase intestinale)
Diète irrationnelle

(a) La persistance de la phase psychique de la sécrétion gastrique, quoique l'opération élimine une bonne partie de la zone sécrétante de la muqueuse gastrique.

(b) Le réservoir gastrique est très réduit, de sorte que le transit gastro-jéjunal devient très rapide et provoque dans plusieurs cas, une distention jéjunale avec syndrome de dumping,[59] et provoque parfois un reflux duodénal avec un syndrome de l'anse afférente.[51]

(c) Persiste aussi la possibilité d'une exagération de la phase intestinale[29] de la stimulation des cellules bordantes à la ligne. La gastrectomie, Billroth II, a donc, malgré ses preuves des résultats excellants et satisfaisants de 90% et plus, ses faiblesses; et il est opportun d'étudier d'autres modalités d'interventions basées sur l'anatomie et la physiologie de l'estomac et du duodénum, dans le but d'améliorer, s'il se peut, les résultats lointains de cette chirurgie. C'est ainsi que l'intervention la plus rationnelle sera donc celle qui d'une part, élimine les phases céphalique et antrale de la sécrétion gastrique et emporte une partie assez importante de la masse cellulaire sécrétante et, d'autre part, conserve un réservoir gastrique suffisant en permettant un transit pratiquement physiologique y compris le brassage duodénal, qui peut alors jouer son rôle régulateur sur la sécrétion chlorhydrique.

TABLEAU XII.—Vagotomie avec antrectomie —Billroth I

Phase psychique: nil
Réservoir suffisant
Phase antrale: nil
Transit modérément rapide
Phase intestinale +

Il s'agit donc d'une gastrectomie partielle — d'environ 50% — ou antrectomie combinée à une vagotomie bilatérale complète ou sélective,[1, 40] avec anastomose gastro-duodénale, type Billroth I. Avec cette intervention, le transit est modérément rapide, le dumping très rare, la phase intestinale de stimulation gastrique non exagérée (Tableau XII).

Cette intervention est pratiquée actuellement dans notre milieu chez un grand nombre de malades depuis les dernières quatre à cinq années et le recul du temps nous renseignera sur la valeur chiffrée, comptée, qu'elle peut donner.

Les courbes suivantes indiquent d'ailleurs les tendances dans l'utilisation des divers types d'opération au cours des 25 années, allant de 1940 à 1965 (Fig. 2).

Conclusion

Ce travail a mis en évidence les résultats mauvais, dans des proportions variant de 7% à 10%, qu'on a obtenus au cours des années 1940 à 1960, avec les opérations classiques: la gastrectomie subtotale (66% à 75%) d'une part, et la vagotomie avec gastroentérostomie d'autre part.

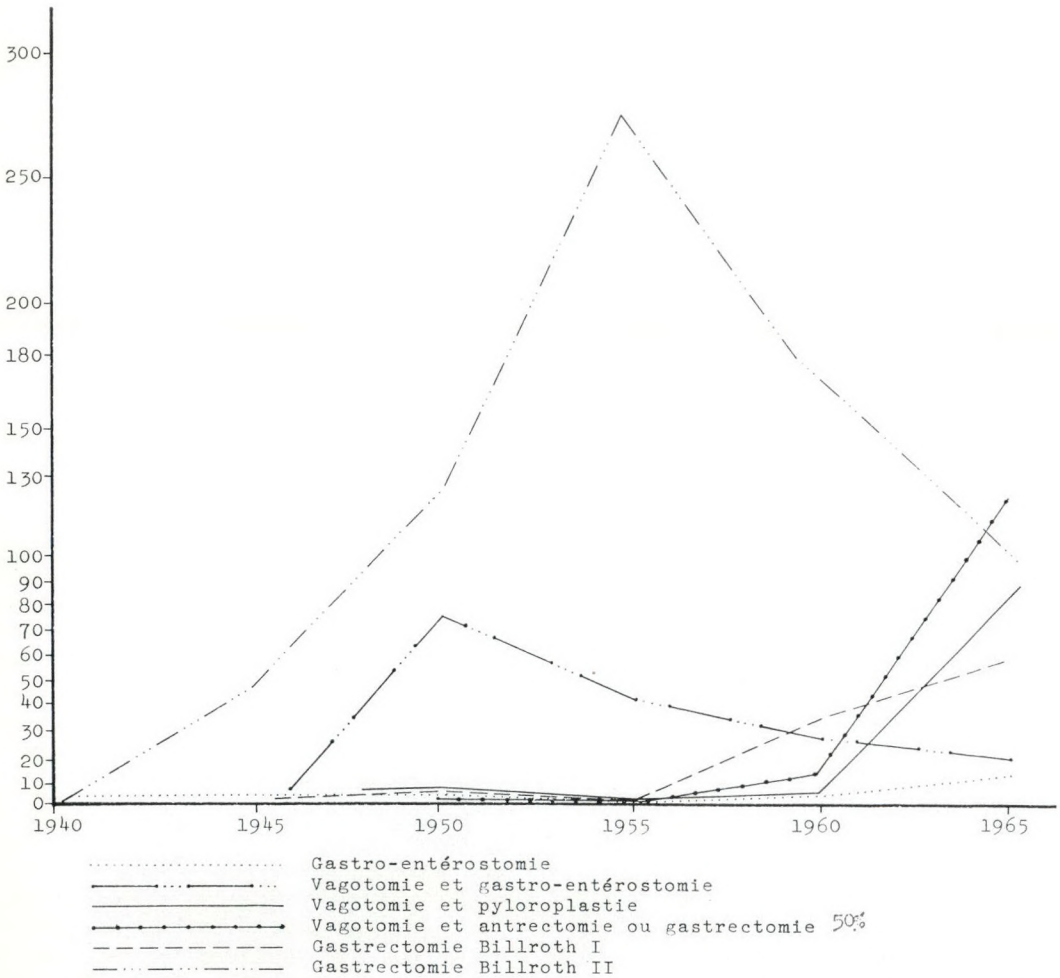

Fig. 2.—Les tendances dans l'utilisation des divers types d'opération au cours des 25 années, allant de 1940 à 1965.

L'étude de la physiopathologie des complications en confrontation avec les styles d'opérations, nous font croire que les modalités d'interventions maintenant préconisées permettront d'améliorer de façon remarquable les résultats à longue échéance et réhabiliteront un plus grand nombre de malades à une vie utile et heureuse.

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PRATIQUE CANCEROLOGIQUE. Marcel Dargent. 232 pp. Illust. L'Expansion Scientifique Française, Paris, 1965. 90 F. $18.00 (approx.).

On pratique de moins en moins, dans nos écoles de médecine nord-américaines, l'art tout européen de la leçon magistrale. Cliniques, symposiums, discussions à la table ronde, démonstrations appuyées d'illustrations audiovisuelles ont remplacé le cours du professeur attaché à son lutrin, dispensant à la fois la profondeur de sa science et l'élégance de son style. Si la génération d'ajourd'hui y a sans doute gagné, celle d'hier éprouve encore la nostalgie de pédagogie ancienne.

C'est à ce titre que les leçons de cancérologie publiées par l'Ecole de Lyon plairont au groupe rassis, sophistiqué et blasé des médecins canadiens. Conçu à l'intention des étudiants du niveau sous-gradué, l'ouvrage ne vise pas à l'originalité, à la rigueur scientifique ou à la révélation sensationnelle. Le style lui-même en est celui de la leçon parlée bien plus que de l'article de revue ou du chapitre de manuel. On n'y trouve pas de bibliographie formelle. Les statistiques et les protocoles expérimentaux sont réduits au strict minimum. Toutefois, l'illustration est abondante et d'excellente qualité, la présentation agréable et l'impression générale plus que satisfaisante.

Quand au fond, les aspects généraux du cancer, aux points de vue clinique, experimental, anatomopathologique et thérapeutique, y trouvent tous leur compte. Tout en simplifiant, on a soigneusement évité l'erreur qui aurait consisté à tomber dans la vulgarisation. Si certaines considérations philosophiques un peu vagues atteignent à la banalité du lieu commun, il en reste une vue d'ensemble à laquelle on ne s'arrête peut-être pas assez souvent. S'il faut trouver à redire de cet ouvrage fort louable, on pourrait souligner le peu de cas qu'on y fait des tumeurs non-épithéliales, en particulier des lymphomes et des leucémies. Ainsi est-il à peine question de ces types tumoraux dans le chapitre sur les antimitotiques. On pourrait aussi mettre en doute certaines affirmations de détails, dont la plupart, par contre, relèvent de différences valables d'opinion.

A tout prendre, il s'agit d'un ouvrage qui peut rendre grandement service à tous ceux qu'intéressent l'étude, l'enseignement ou la pratique de la cancérologie.

SUPERFICIAL ARTERIES OF THE CUBITAL FOSSA WITH REFERENCE TO ACCIDENTAL INTRA-ARTERIAL INJECTIONS

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Anatomical variations have differing degrees of importance to the practising physician and, in this respect, some regions of the body are cited less frequently in the medical literature than others. The occurrence of a complication sometimes recalls a well-known anatomical fact more or less forgotten in daily practice. Intravenous injections in the elbow region are frequent minor surgical procedures during the performance of which the presence of an aberrant artery may change an intravenous into an intra-arterial injection. The possible disastrous consequences to the patient of such an error are so much more regrettable when the injection is performed during the investigation or treatment of a minor ailment. The complications range from marked temporary arterial spasm to complete obstruction and gangrene. Textbooks of anatomy do not insist on this ever-present danger and some well-known student textbooks do not even mention the significant anatomical variations. An underestimation of the frequency of aberrant superficial arteries in this area is, without doubt, a contributing factor in accidents following injection.

Tiedmann[1] in 1831 appears to have been the first to describe these arterial anomalies systematically. His bibliography contains 38 references on the subject dating back to Laurentius (1600). Quain[2] in 1844, Henle[3] in 1868, De Vriese[4] in 1902 and Müller[5] in 1903 also made important contributions to the subject. Poynter[6] summarized all of the reported series of anomalies up to 1920, adding 250 specimens of his own, providing in all, a review of several thousand cases. Dubreuil-Chambardel[7] in 1926 made a comprehensive study of the question and Adachi[8] in 1928 reported observations based on 1198 upper limbs. His series showed a significantly lower incidence of superficial ulnar arteries in the Japanese. Hazlett[9] in 1949 studied the occurrence of superficial ulnar arteries alone in 94 cadavers and in 271 living individuals. Finally in 1953, McCormack, Cauldwell and Anson[10] added a study of the frequency of arterial anomalies based on 750 upper extremities.

Normal Anatomy

In the normal arrangement (Fig. 1) a rich vascular network surrounds the elbow joint. Ulnar and radial collateral arteries provide secondary channels in addition to the brachial bifurcation and probably can supply blood to the distal portion of the upper limb in some cases of arterial occlusion. The bifurcation of the brachial artery into radial and ulnar branches occurs normally below the articular line. It is significant that arteries become superficial at the elbow when their level of individual origin or the brachial bifurcation itself take place above the articular line. Another vessel and its origin worthy of consideration is the artery to the median nerve. This vessel arises from the anterior interosseous artery. The latter is a branch of the common interosseous which in turn most frequently arises from the ulnar artery. When the

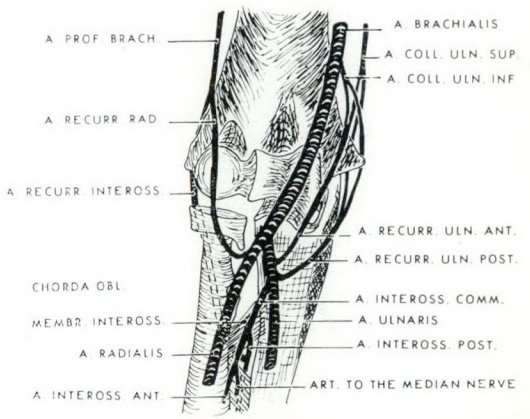

Fig. 1.—In the normal arrangement of the vessels around the elbow, the brachial bifurcation is located below the articular line. The artery to the median nerve is a branch of the anterior interosseous which in turn arises from the common interosseous artery. The normal origin of this larger arterial trunk is from the ulnar artery.

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brachial bifurcation or the origin of the ulnar artery occurs at a high level, the common interosseous artery is apt to take its origin from the radial (Fig. 2) or the brachial (Fig. 3) rather than from the ulnar artery. An arrangement of this type explains why the musculature innervated by the median nerve has been spared in some instances of thrombosis of a superficial ulnar artery caused by an accidental injection.

Abnormal Anatomy

An analysis of the literature on this subject[2, 8-10] indicates that, on the average, 18% of the population have superficial arteries at the elbow. The condition is bilateral in one-fifth of the group. One may find only one artery in a superficial location at the elbow, but all of the three important ones, the brachial, the radial and the ulnar may be superficial (Fig. 3). When superficial, the radial artery usually runs under cover of the bicipital aponeurosis (lacertus fibrosus) (Figs. 3-5) and reaches a superficial position in the forearm where it may remain subfascial all the way down to the wrist without the protection normally afforded by the brachio-radialis muscle. McCormack, Cauldwell and Anson[10] report that the radial artery is the most common of the superficial arteries of the elbow region, being found in approximately 15% of normal individuals.

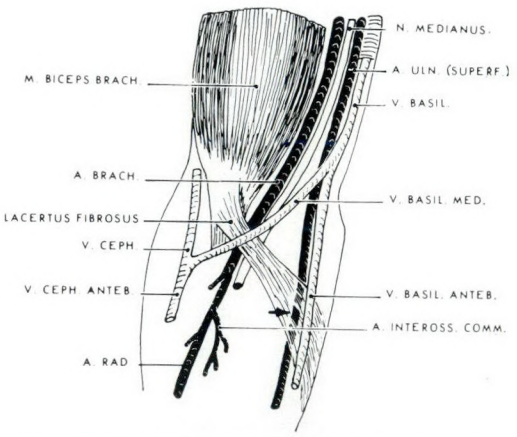

Fig. 2.—The high origin of the ulnar artery had rendered it subfascial in the elbow region until, by piercing the bicipital aponeurosis, it became subcutaneous for a short distance (arrow). By piercing the aponeurosis again it returned to a premuscular course. The ulnar artery had no common interosseous branch, this artery arising from the radial. Right elbow redrawn from Hazlett.[8]

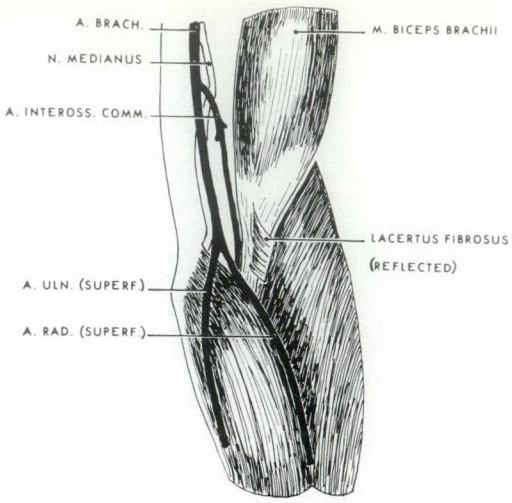

Fig. 3.—The brachial bifurcation was proximal to the articular line and closer to the skin. Both radial and ulnar arteries were superficial (subfascial). The common interosseous did not have an ulnar origin, being a direct branch of the brachial. Left elbow redrawn from Hazlett.[9]

The ulnar artery is found in an aberrant location less frequently than the radial. According to Hazlett,[9] it is in a superficial position in about 3% of individuals. This artery may be superficial for a short distance only. Hazlett found a case (Fig. 2, arrow) where it ran a brief subcutaneous course through the bicipital aponeurosis and was subfascial and therefore premuscular for the rest of its course in the forearm. Fig. 4 illustrates a dissection from our laboratory where the ulnar artery was subcutaneous well above the elbow region and became subfascial by entering the lacertus fibrosus. It remained premuscular, and thus subfascial, in the forearm for its entire length except at mid-forearm level where it was crossed superficially by a tendinous part of an inverted palmaris longus muscle. This palmaris longus had a muscular belly in the lower part of the forearm from which it fanned out into the palmar aponeurosis, as would normally be expected. It had an additional slip of insertion to the hypothenar eminence.

In less than 1% of cases,[10] a superficial accessory brachial artery, in addition to and parallel with a normal one, is present. Superficial ulnar and radial arteries are

apt to appear as "satellite arteries" of veins of the cubital fossa (Fig. 6) and one should be suspicious when parallel vessels are seen in this region. Arterial pulsations are not a reliable or absolute criterion for identification of an artery since slight compression or a disadvantageous position may reduce or obliterate pulsations in these arteries. Abduction of the arm to a right angle at the shoulder is a position which may interfere with detection of the pulse.

Accidental Injections

Accidents during injection involve the experienced practitioner as well as the beginner. Stone and Donnelly[25] reviewed reports by Lundy,[28] Cohen[13] and Dundee[29] whose respective figures are one in 8000, one in 56,000, and one in 3500. Vidal[12] reported an incidence of one per 120 injections of hydroxidione. Such figures are

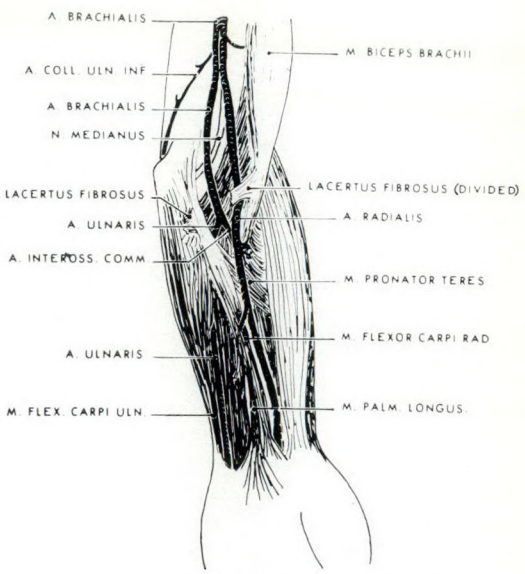

Fig. 5.—The brachial bifurcation occurred above the elbow. The radial artery became superficial after running deep to the bicipital aponeurosis. Left elbow from a personal dissection.

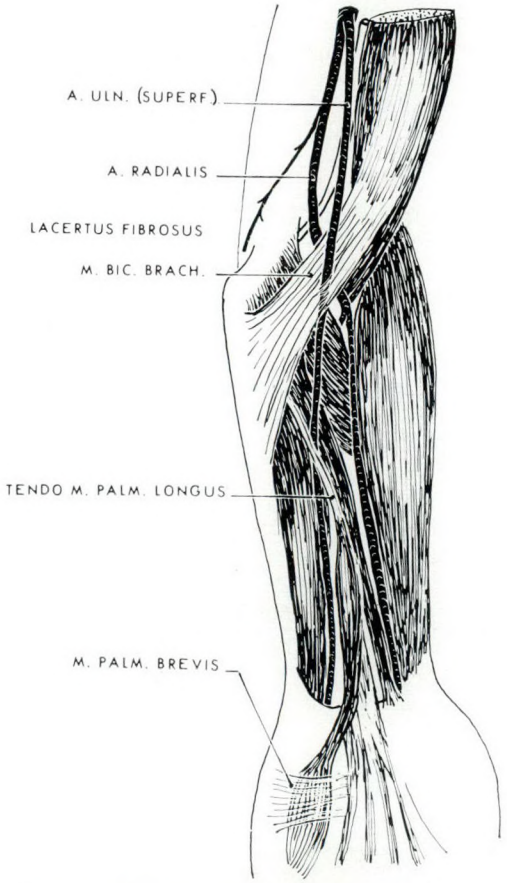

Fig. 4.—The bifurcation of the brachial artery occurred at an exceptionally high level in the arm. The radial artery was subfascial from the elbow down to the wrist. The ulnar artery was subcutaneous at arm level. It entered the bicipital aponeurosis (lacertus fibrosus) and remained premuscular except for a short distance in the midforearm where it was crossed by the abnormally located tendon of the palmaris longus muscle (from a dissection made by the author).

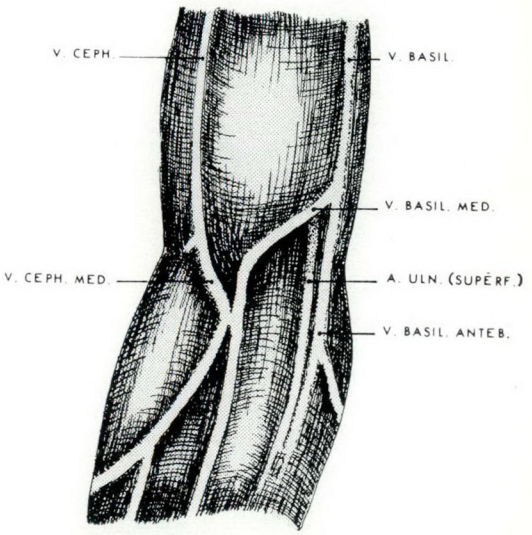

Fig. 6.—A superficial ulnar artery very often is a "satellite vessel" of a regular antecubital vein. A radial superficial artery could have the same appearance (from Hazlett[9] and Cohen[13]).

widely diverse and indicate the problems involved in determining the incidence of such accidents.

The drugs which have led to arterial obstruction after their injection included arsenides, ether, alcohol, opiates, thiopentone, quinine, antibiotics and tranquillizers. Contrast media and even steroids, employed in some countries for short-term anesthesia were also implicated.

The injection apparently need not be intra-arterial in all cases of thrombosis. An irritant in the adventitia of the vessel may cause prolonged spasm which, in many cases, does not permit smaller collateral vessels to supply the deprived area adequately. Although the presence of superficial arteries increase the risk of accidental intra-arterial injections, errors may occur even with normally located arteries. By the level of demarcation of a gangrenous forearm or hand, one can mark with accuracy not only the artery accidentally punctured but even the level of the injection in or around this artery. Exceptions occur when a higher level of obstruction of the brachial artery is caused by an upward extension of the thrombosis due to a markedly lowered arterial pressure, vasospasm or compression at the shoulder region. This creates a well-delineated but higher and more extensive level of gangrene.

This review concerns reported cases of arterial obstruction in the available literature since 1948.[11-24] Reports of local necrosis, venous thrombosis, sensory losses and local mucular destruction or fibrosis following injections without proof of arterial involvement were discarded. Cases of arterial obstruction with resulting gangrene were selected as an indirect method of determining the limitations of the collateral circulation around the elbow joint. My interest was in the collateral circulation and the possibility of the blood bypassing the main arterial trunks of the elbow in order to sustain the distal portion of the upper limb. Most of the publications about accidental injections pertain to the upper limb, because the elbow is the choice site for the intravenous administration of fluids. Cases where treatment was instituted in time to prevent gangrene were discarded. No reports were found in which arteriograms were performed following these recoveries.

The purpose of the present study was to determine whether collateral channels at the elbow could substitute adequately for the main arteries in cases of complete and sudden obstruction. From these published reports the inadequacy of this collateral circulation was demonstrated by the appearance of gangrene of the extremity following accidental injections. In some instances arteriography[18] or surgery for thrombectomy and decompression[19] were performed, thus identifying the exact level of obstruction. Fifty-five cases of gangrene and amputation suitable for this study have been reported since 1948. In 21, precise anatomical descriptions were available; in five of these superficial arteries were involved.

Brachial Artery Involvement

The most extensive amputations (Fig. 7a) were performed in the two reported cases where the thrombosis reached to within two inches of the axilla. There is justification here for thinking that the circulation through the inferior ulnar collateral, superior ulnar collateral and the profunda brachii arteries were affected by the obstructive process. The bifurcation of the thrombosed brachial artery in these cases was found one inch above the elbow with both radial and ulnar arteries being superficial. The amputations were done at the

Fig. 7a to 7e.—Résumé of the cases where the brachial artery was involved.

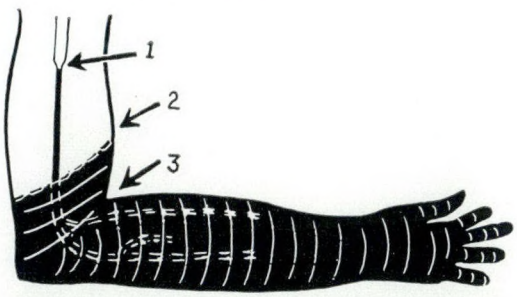

Fig. 7a.—Case 6 of Cohen[13] and the case of Ogilvie, Penfold and Clendon[17] following the use of thiopentone in the former, and of a muscle relaxant (Myanesin) in the latter. (Arrow 1 points to the level of thrombosis just below the axilla. Arrow 2 indicates the level of the amputations. Arrow 3 is the injection site.)

level of the lower third of the arm. Gangrene occurred and amputation was performed at the level of the upper forearm in four cases when thrombosis took place about two inches above a normally located brachial bifurcation (Fig. 7b). It was possible to conserve the upper forearm in two patients when thrombosis was arrested at the brachial bifurcation (Fig. 7c) by preventive measures. These amputations were done at the mid-forearm level.

In three cases of low-forearm amputations (Fig. 7d) thrombosis involved the radial and ulnar arteries, the brachial bifurcation being either spared or only partially thrombosed. Amputations at wrist level were performed in two patients in whom, presumably, the thrombosis was limited to a low level in both the radial and the ulnar arteries (Fig. 7e). The brachial

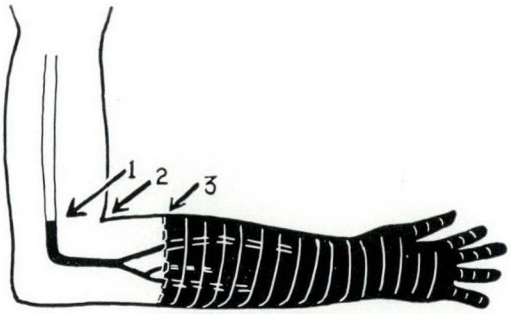

Fig. 7b.—Sequence of events in Cases 1, 3 and 5 of Cohen[13] and in the case of Olivier, Rettori and Languepin.[18] In this last accident, alcohol 33% had been used. (Arrow 1 indicates the level of thrombosis, arrow 2, the injection site and arrow 3, the level of amputations at the upper third of forearm.)

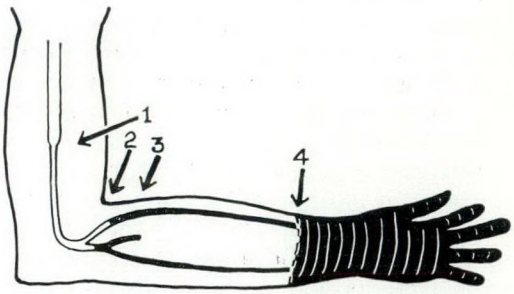

Fig. 7d.—The case of King and Hawtof[16] following an injection of ether, and Case 1, of Opinsky, Serbin and Rosenfeld.[19] (Arrow 1 shows the site of narrowing of the artery which was pulseless, whitish but not thrombosed at the operation. Arrow 2 indicates the site of the injection and arrow 3, the level of thrombosis on both the radial and ulnar arteries). The brachial bifurcation was either spared or only partially thrombosed. (Arrow 4 shows the level of the amputations.) Similar events occurred in a case reported by Vidal[12] following an injection of a steroid. In the case of King and Hawtof,[16] the brachial bifurcation was high and the ulnar artery was superficial. Early surgical decompression was successful in preventing further ascent of the thrombosis.

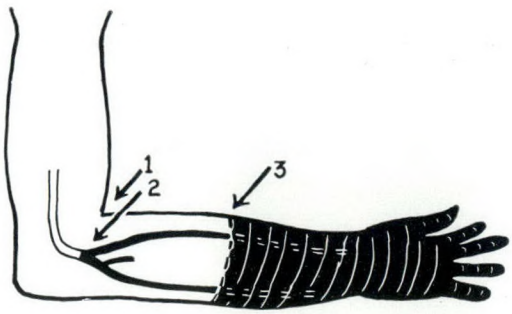

Fig. 7c.—The case of McIntosh and Heyworth as reported by Cohen[13] (Case 11) and that of Balice.[11] (Arrow 1 is the site of the injection, arrow 2, the thrombosis just above the brachial bifurcation, and arrow 3, the mid-forearm amputations.) In the case of McIntosh and Heyworth, the patient was a five-year old boy whose obstructive symptoms increased suddenly eight days after an accidental injection of thiopentone. This deterioration occurred when the thrombosis progressed from below, upward to the brachial bifurcation. Chlorpromazine was the drug involved in Balice's reported case.

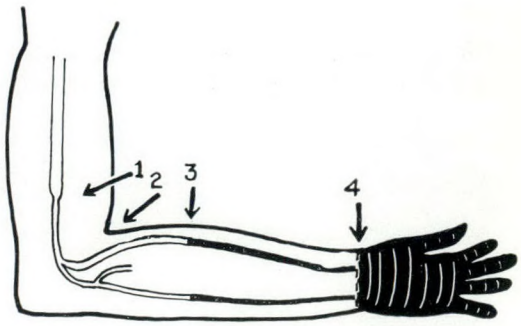

Fig. 7e.—Case 2 of Weese and Hentschel[23] and the case of Grondin *et al.*[15] (Arrow 1 gives the level at which the brachial artery became small, whitish and pulseless.) In Case 2 of Weese and Hentschel[23] the thrombosis had spared the brachial bifurcation; the drug involved was Basinarkon. In the case of Grondin *et al.*[15] the level of thrombosis is not stated; the drug injected was promazine. (Arrow 2 indicates that the site of injection was at the elbow in both cases, arrow 3 indicates the thrombosis of both radial and ulnar arteries in Case 2 of Weese and Hentschel[23] and arrow 4, the transcarpal amputation in both examples.)

artery in each of the five patients was spastic, whitish and pulseless at the time of the operation.

Ulnar Artery Involvement

Complications are not as extensive following obstruction of either the ulnar or the radial artery alone. When the ulnar artery is the only important vessel involved, necrosis is limited to the ulnar side of the forearm and hand, and to the paralysis of muscles innervated by the median nerve. Fig. 8a is an illustration of very severe ulnar thrombosis. Since the ulnar artery in this case was not superficial it gave off its common interosseous branch below the level of thrombosis. Therefore the anterior interosseous and the artery to the median nerve were occluded. Tissue loss was extensive in the forearm and in the hand. In another case (Fig. 8b) of similar ulnar thrombosis, the loss was not as extensive. The third example (Fig. 8c) is different in that it occurred in a subject in whom the superficial ulnar artery arose one inch above the elbow. The flexor musculature was spared because the common interosseous artery did not originate from the ulnar. Fig. 8d represents the very limited loss in two additional cases where the occlusion involved only a short segment of the ulnar artery.

Radial Artery Involvement

The complications following thrombosis of this artery are limited to the radial side

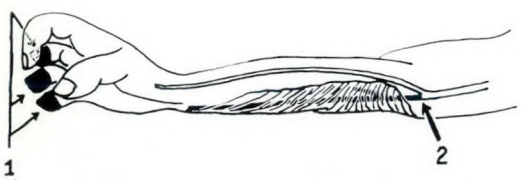

Fig. 8a to 8d.—Résumé of the case where the ulnar artery was thrombosed. The injection site was at the elbow.

Fig. 8a.—Case 1 of Weese and Hentchel,[23] Arrow 1 indicates the level of amputation of the 3rd, 4th and 5th digits which was necessary following thrombosis of the ulnar artery from the level of Arrow 2. The fibrous retraction of the paralyzed flexor group of muscles caused a fixed flexion of the fingers. The extensors of the wrist overcame the contracture of the flexors. There is a reduction in size and an extensive scarring of the forearm.

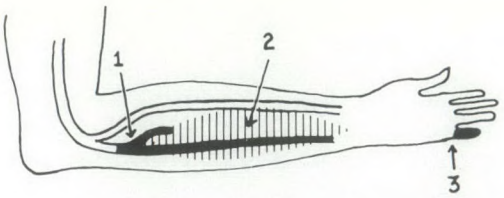

Fig. 8b.—This is the case described by Vourc'h.[22] (Arrow 1 points at the high ulnar thrombosis and arrow 2, (hatched area), to the paralyzed flexors in the forearm.) This complication together with the normal course of the ulnar artery makes it certain that the common interosseous artery was thrombosed and the anterior inter-osseous artery was involved. (Arrow 3 indicates the level of amputation of the fifth finger.) The drug injected was Estil.

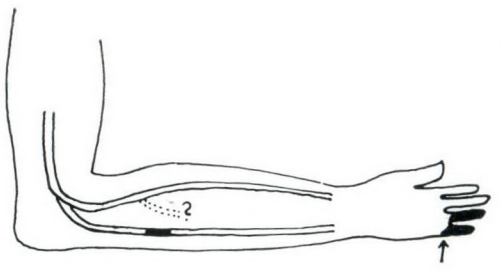

Fig. 8c.—Case 12 of Cohen[13] (Van der Post), The arrow points at the level of amputation of the 4th and 5th finger. The ulnar artery was superficial and arose a "full inch above the elbow". The thrombus extracted from the vessel "was about ¾ inch long and ⅛ inch thick". The interrogation mark suggests a probable origin of the common interosseous from the radial artery since the anterior interosseous was not involved. Here again, the injection site was at the elbow.

Fig. 8d.—Case 2 of Opinsky, Serbin and Rosenfeld,[19] and one stage in the case of Wolfe and Burckhardt.[24] The arrow points to the small amputation done at the 4th finger at the former where a two-inch-long clot was removed from the ulnar artery. No mention was made of a possible superficial position of this artery or of the level of the thrombosis, although the flexor musculature was spared. In the latter case, subsequent amputations to the level of the metacarpal were necessitated by progression of the gangrene.

of the limb and none are as extensive as in the more severe ulnar involvements. Both the radial and the ulnar arteries were superficial in the two cases represented by Fig. 9a. The common interosseous artery was not involved. Gangrene of the thumb and the index finger in two other cases (Fig. 9b) followed thrombosis of a normally located radial artery.

Prevention of Accidents and Complications

If injections must be made in the antecubital fossa, this region should be carefully examined beforehand, particularly if the solution to be injected is an irritant. With such drugs one should keep in mind that vasospasm and thrombosis occur even if the artery is not punctured. Indeed the literature contains several examples where para-arterial spillage of a drug has caused distal gangrene in both the upper or the lower limbs.[26, 27] In many cases superficial arteries can be recognized by their pulsations. Persons who administer intravenous injections should be aware that in certain circumstances the pulse of such arteries may be reduced or absent. Superficial arteries may bear a striking resemblance to veins. At the beginning of an intra-arterial injection, blood entering the syringe may not have a scarlet colour or a pulsating character. An intense burning pain in the forearm or in the hand provides an early clue to such accidents in the conscious patient and the injection should be terminated at once. The immediate addition of local anesthetic at the site of injection has successfully prevented complications in many cases, the symptoms receding in a matter of minutes. In unconscious or anesthetized individuals, the patient's agitation at the time of the injection or a delay in producing relaxation should arouse suspicion. The disappearance of the pulse at the elbow or the wrist and the blanching and coolness of the limb followed some hours later by intense redness were the common sequence of events reported in patients who eventually required amputation. These signs suggest an arterial accident and treatment should be instituted.

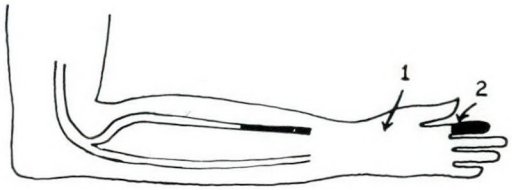

Fig. 9a and 9b.—Résumé of the cases where the radial artery was thrombosed.

Fig. 9a.—Case 4 of Cohen.[13] Arrow 1 points at the intrinsic muscles of the hand and of the thenar eminence, temporarily involved by the thrombosis. Arrow 2 indicates the level of amputation of the index finger. The injection was into the cubital fossa. Gordh's[14] case had similar involvements and the same amputation. The radial artery was superficial in both Cohen's and Gordh's case. The common interosseous was not involved.

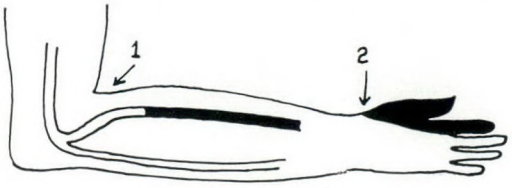

Fig. 9b.—This figure illustrates Case 9 of Cohen[13] and Case 21 of Sénèque and Hugenard.[21] Although in the former,[13] the injection site was at the elbow (Arrow 1) the amputation was limited to the thumb, the index finger and part of the radial side of the hand. In the latter case,[21] the injection (Arrow 2) was intended for the cephalic vein of the thumb in the anatomical snuffbox. The level of amputation was the same in spite of the great difference in the site of injection. The drug injected in the latter case was a mixure of Diparcol-Dolosal (piridosal-diéthazine).

In many of the more extensive cases of gangrene, the accident went unnoticed for hours, thus sealing the fate of the limb. Some of the patients were symptomless after a few hours and were unfortunately allowed to leave the hospital after two or three days, only to return for amputation later.[13] Decompression, if it is carried out promptly, results in a good prognosis even if the limb has become edematous and reddened. King and Hawtof[16] had the rewarding experience of having a pulseless, narrow and white brachial artery regain a normal size, colour and pulse when they relieved the pressure by dividing the antebrachial fascia and leaving it open for some days. Besides supportive therapy, anticoagulant and vasodilator drugs have been used together with brachial or stellate sympathetic blocks. Thrombectomy accelerated the gangrene in one instance, the patient's

condition deteriorating faster after the operation. In the future, improvement of methods of removal of thrombosis and the insertion of small arterial grafts may improve the prognosis of cases of this type.

Summary

Intra-arterial injections are an ever-present danger when intravenous fluids are administered *via* the antecubital fossa. Superficial arteries exist in 18% of individuals and add to this risk. According to some authors, thrombosis of an artery may progress to a higher level than the site of the accidental injection when the patient is in a Trendelenburgh position, if undue compression is exerted at shoulder level, or if the arm is at right angles to the body. Such a high level of thrombosis may necessitate an amputation above the level of the elbow joint. If occlusion of the brachial artery occurs just above the elbow joint, the demarcation of the gangrene and the site of amputation are usually at the upper third of the forearm. When the brachial artery is thrombosed at its bifurcation, early and intensive treatment may save the upper half of the forearm. Occlusion involving the radial and ulnar arteries together may spare the upper two-thirds of the forearm. Even if the brachial artery is narrowed and spastic, the forearm may be saved and the amputation done at the wrist. When a normal ulnar artery is the vessel occluded, widespread complications in the forearm and the hand can be expected although the gangrene is limited to the ulnar aspect of the limb. If the occluded ulnar artery is in a superficial position, chances are that the flexor musculature of the forearm will be unharmed. A thrombosed radial artery often results in amputation of the thumb and the index finger. Aberrant arteries are involved in a considerable percentage of accidental injections at the elbow (about 24%). One may be misled by an overconfidence concerning the ability of collateral vessels to supply the distal part of the limb in sudden occlusion of the main arterial trunks. In this respect, such channels have very marked limitations.

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Résumé

On trouve au pli du coude, chez environ 18% des individus, des artères anormalement superficielles dont les représentantes sont l'artère radiale (15%), l'artère cubitale, l'artère humérale elle-même et, très rarement, une artère humérale superficielle accessoire (3%). La présence de telles artères en position superficielle augmente le risque, toujours présent d'ailleurs, de voir une injection prévue comme intra-veineuse, devenir inopinément intra-artérielle. Des conséquences regrettables et graves telles que la gangrène et l'amputation n'ont pu être évitées dans le passé malgré un traitement précoce et intense. Ainsi l'occlusion de l'artère humérale peut exiger une amputation au niveau de l'avant-bras ou même du bras. Si l'artère radiale ou l'artère cubitale est thrombosée, les lésions sont limitées respectivement externes ou interne de l'avant-bras ou de la main. Les fléchisseurs du poignet sont paralysés s'il y a thrombose du tronc des artères interosseuses puisque l'artère du nerf médian est une branche de l'interosseuse antérieure. Cinquante-cinq cas d'amputations survenus depuis 1948 ont été investigués. Vingt et un d'entre eux ont une description anatomique suffisamment précise pour que l'on puisse déduire à quel niveau se fait l'amputation si on connaît le niveau de l'occlusion artérielle ou inversement à quel niveau se situe l'occlusion artérielle lorsqu'apparaît la ligne de démarcation gangréneuse. Cinq de ces 21 cas d'injections accidentelles survinrent dans des artères anormalement superficielles. L'injection intra- ou para-artérielle accidentelle de substances irritantes provoque donc souvent une obstruction artérielle avec des séquelles importantes et le risque de telles injections est augmenté par la présence d'artères anormalement superficielles.

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CASE REPORTS

MALADIE KYSTIQUE CONGENITALE DU POUMON

Présentation de deux cas traités par lobectomie pulmonaire avec survie*

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La maladie kystique congénitale du poumon, bien que rare, ne doit pas être considérée comme une curiosité pathologique. En 1638, Fontanus a été le premier à décrire la maladie qui avait été découverte chez un nourrisson de trois mois. En 1687, Bartholinus rapporta à son tour le cas d'un enfant de quatre ans qui présentait des dilatations kystiques à tout le lobe pulmonaire supérieur gauche. Au siècle dernier, plusieurs auteurs ont publié des cas typiques de la maladie. Mentionnons, parmi eux, Meyer (1859); Biermer (1860); Virchow (1862); Barlow (1880); Grawitz (1880); Klebs (1889); Couvelaire (1904); Löhlein (1908); Hueter (1914).

En 1925, Koontz[2] fut le premier à rapporter dans la littérature médicale américaine le cas d'un nouveau-né mort à l'âge de 12 jours de kystes congénitaux de deux poumons. Il fit une revue complète de la littérature médicale à ce sujet, releva tous les cas qui avaient été publiés avant 1925 (moins de 100 cas) et discuta des différentes théories pathogéniques émises pour expliquer la maladie.

Depuis quelques années, le nombre de cas publiés a augmenté considérablement. En 1937, Schenck a revisé un total de 381 cas. Dans la même année, Wood a ajouté 48 cas, découverts au "Mayo Clinic". Wiese, en 1942, a trouvé 400 cas publiés. Par ailleurs, à la suite des revues faites par Dickson (1946) et Adams (1946) et de nombreux autres rapports faits par Ch'in et Tang (1949), Goodyear et Shillitoe (1959),[4] Herrmann (1959), Levine (1962),[8] le nombre total de cas dépasse maintenant 600; ce qui prouve que la maladie ne peut plus être considérée comme une curiosité médicale.

Plusieurs théories ont été émises pour tenter d'expliquer la pathogénie de la maladie. Certains, comme Stoerk,[14] pensaient qu'elle était due soit à une bronchectasie fœtale secondaire à un processus inflammatoire avec sténose et fermeture des bronchioles chez le fœtus et comme conséquence dilatations kystiques bronchiques, soit à un développement excessif des tissus environnant l'arbre bronchique, principalement le tissu conjonctif, avec persistance des caractères fœtaux.

D'autres (Sandoz, Balzer et Grand'homme) ont pensé qu'il s'agissait de syphilis congénitale provoquant un arrêt de développement des alvéoles à partir des terminaisons de l'arbre bronchique chez l'embryon.

D'autres encore (Orth, Heller, Franke) ont décrit la maladie comme une bronchectasie atélectatique où les bronches se dilateraient pour compenser un défaut de développement des alvéoles.

Löhlein et Couvelaire ont prétendu qu'il s'agissait d'une lésion néoplasique.

Virchow et Klebs ont soutenu, de leur côté, que ces kystes pulmonaires congénitaux étaient des dilatations lymphatiques semblables à celles qu'on observe dans d'autres organes tels que le rein.

Aujourd'hui, l'origine embryologique de ces kystes semble bien établie. L'arbre bronchique de même que la trachée prennent naissance du mésenchyme indifférencié au niveau des arcs branchiaux primitifs. Au cours de la vie fœtale, ce tissu descend dans le thorax et se divise en deux pour donner les éperons pulmonaires droit et gauche qui se subdivisent à leur tour en trois lobules du côté droit et en deux lobules du côté gauche. Aux dépens de ces lobules se développent des branches multiples dont les terminaisons se canalisent et

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se dilatent pour former des cavités.

Au sixième mois de la vie fœtale, les alvéoles pulmonaires sont donc déjà formées.

Dans la maladie kystique congénitale des poumons, il survient un arrêt de développement dans le processus de canalisation des embranchements lobulaires distaux. Au delà de ces zones, la formation des cavités se continue, ce qui laisse des segments canalisés isolés, tapissés d'une membrane muqueuse bronchique normale. Les glandes muqueuses sécrétant et le liquide ne pouvant être évacué ni absorbé aussi rapidement qu'il est secrété, des kystes liquidiens se forment. Après la naissance, le poumon se dilatant, ces kystes liquidiens se rupturent dans les bronches et leur contenu est remplacé par de l'air.

Ces cavités kystiques se dilatent ensuite rapidement parce que l'orifice qui assure la communication avec une bronche est ordinairement situé tangentiellement à la circonférence du kyste, formant ainsi un système valvulaire en "bec de flute" qui permet l'entrée de l'air à l'inspiration mais empêche sa sortie à l'expiration.

Du point de vue pathologique et clinique, la maladie peut se présenter sous deux formes principales:

1. Un gros kyste solitaire peut occuper un ou plusieurs lobes, déplaçant le cœur et le médiastin du côté sain et comprimant le parenchyme pulmonaire environnant. Ce kyste, ordinairement tapissé d'un épithélium cilié cylindrique ou cubique, est formé d'une paroi bronchique normale avec des muscles lisses, des anneaux cartilagineux et des glandes muqueuses. On peut aussi trouver des signes d'inflammation aiguë ou chronique dans la paroi du kyste. Parfois, une trop grande distension de la cavité peut causer une atrophie de la tunique propre, des glandes séro-muqueuses et du cartilage qui forment sa paroi. L'épithélium peut être aplati ou même inexistant. Ce sont de telles images histologiques qui ont pu faire croire à certains auteurs que ces kystes étaient des dilatations lymphatiques et non bronchiques.

Le kyste communique toujours avec une bronche, mais il est souvent très difficile de mettre cette communication en évidence.

Cette forme pathologique se rencontre surtout chez le nourrisson et le jeune enfant et donne des symptômes de cyanose avec dyspnée et des signes physiques d'un pneumothorax de tension. Sur le cliché pulmonaire, le kyste solitaire donne une image opaque bien délimitée au niveau d'une plage pulmonaire avec déplacement du cœur et du médiastin du côté sain.

2. Le parenchyme pulmonaire est remplacé par de multiples kystes qui peuvent être très petits et disséminés à travers tout un poumon; ou larges, unis ou multiloculaires occupant un ou plusieurs lobes.

Ces kystes ont une structure histologique semblable à celle de la forme solitaire avec une paroi bronchique ou alvéolaire normale et un épithélium cylindrique ou cubique. Cet épithélium peut être aplati par compression ou proliférer d'une façon démesurée dans la cavité pour donner parfois une apparence adénomateuse à la lésion. C'est pourquoi, certains auteurs ont décrit la maladie sous le vocable de malformation adénomatoïde du poumon. L'aspect macroscopique, à la tranche de section, ressemble à une éponge. D'où le terme de "Honeycomb lung" ou encore de "Poumon aréolaire" ou "Wabenlunge" des auteurs allemands.[14]

Le tableau clinique est parfois celui de la détresse respiratoire avec cyanose et dyspnée chez le nouveau-né. Parfois, la maladie est asymptomatique jusqu'à l'âge adulte et ne se manifeste qu'à la suite d'une complication telle que l'hémorragie ou l'infection.

Le traitement consiste dans la résection du lobe atteint. Nous avons eu à traiter deux cas typiques à l'Hôpital Sainte-Justine.

Histoire de Cas

Cas 1.—Jean-Yves M. a été admis à l'Hôpital Sainte-Justine le 2 avril, 1959 à l'âge de 10 jours pour cyanose et dyspnée depuis la naissance. A l'examen, on nota une submatité à la partie antérieure de l'hémithorax droit (Fig. 1).

Le cliché pulmonaire du jour de l'admission, montra une zone d'opacité occupant les 2/3 inférieur et antérieur de la plage pulmonaire droite, repoussant le cœur et le médiastin vers la gauche. Un œsophagogramme fait deux jours plus tard, montra un œsophage perméa-

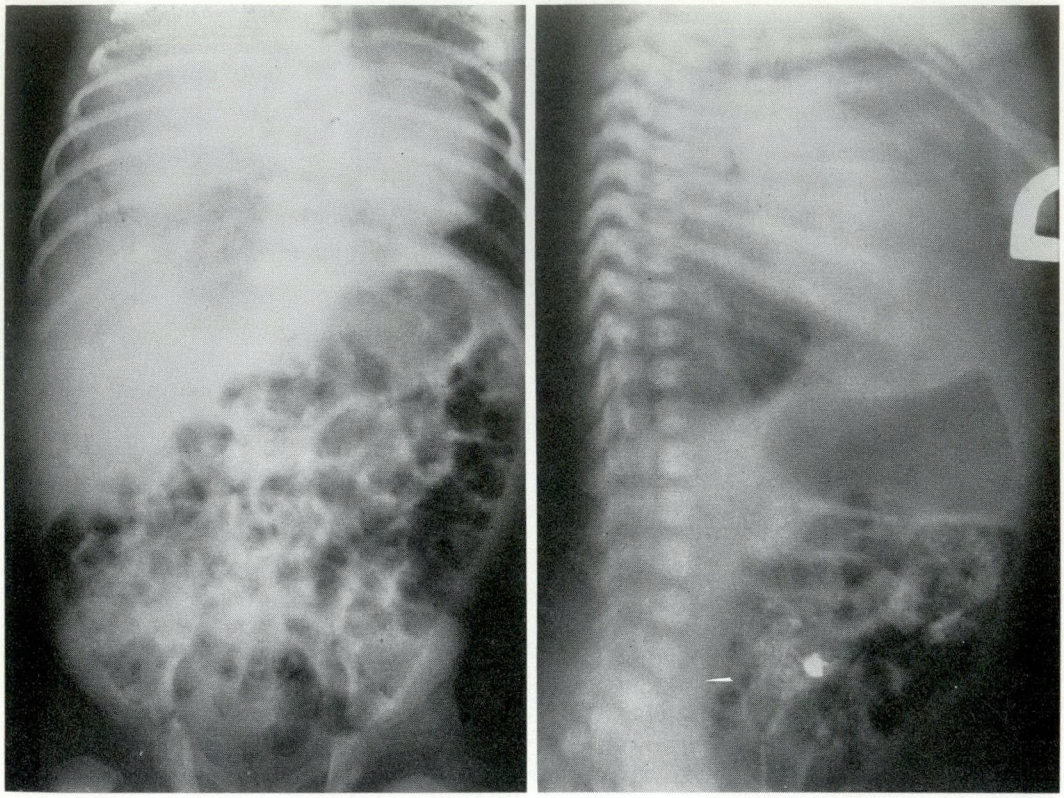

Fig. 1.—Poumons (face et profil)—pré-opératoire. Présence d'une masse homogène vaguement arrondie remplissant la partie antérieure et basse de l'hémithorax droit. Cette masse comprime le poumon droit et déplace le cœur et le médiastin vers la gauche.

ble légèrement repoussé à gauche par une masse paramédiastinale droite (Fig. 2).

Une thoracotomie droite fut pratiquée le 7 avril, cinq jours après l'admission; on trouva une tumeur énorme de consistance fibro-kystique occupant tout le lobe moyen. La résection de ce lobe fut pratiquée selon la technique habituelle. La cavité pleurale fut drainée et la paroi thoracique refermée. L'évolution post-opératoire fut sans incident, et le patient quitta l'hôpital le 14 avril, 1959 (Fig. 3).

Rapport histopathologique.—Lobe moyen du poumon droit pesant 95 g. et ayant un volume de 95 cm.^{3} Le tissu pulmonaire semble rénitent à la pression. A la section de la pièce, il s'écoule du liquide trouble mais très peu visqueux. La tranche de section donne l'impression d'une éponge et on y voit plusiers petits kystes à surface reluisante et sillonnée de petits vaisseaux (Fig. 4).

L'examen microscopique montre qu'il s'agit de dilatations kystiques bronchiques multiples.

Conclusion.—Poumon aréolaire.

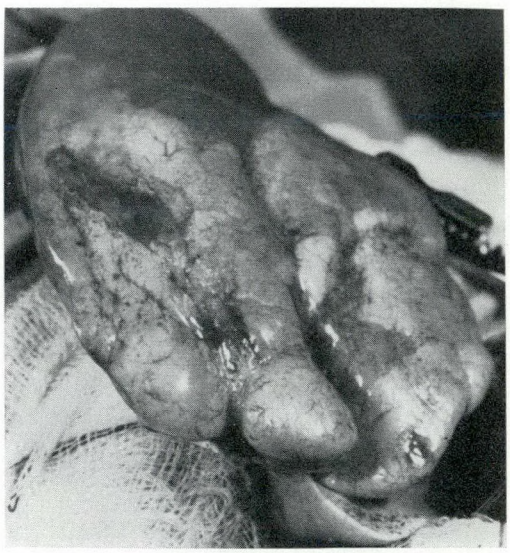

Fig. 2.—J.-Y. M.—Photographie prise pendant l'opération, montrant le lobe pulmonaire inférieur droit augmenté de volume et contenant des kystes.

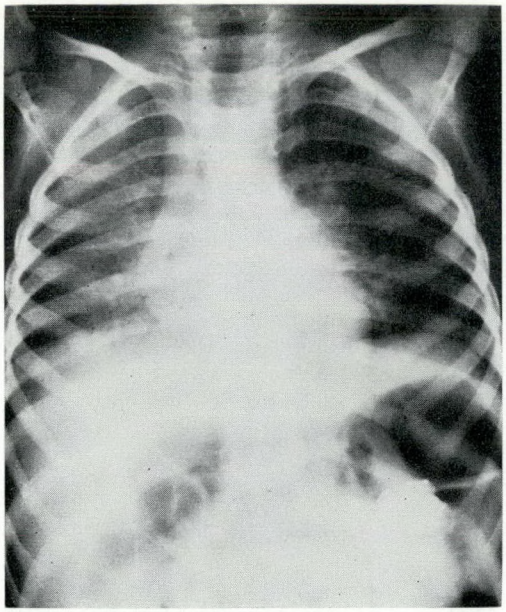

Fig. 3.—J.-Y. M.—Poumons (face)—post-opératoire. Image cardio-pulmonaire dans les limites normales.

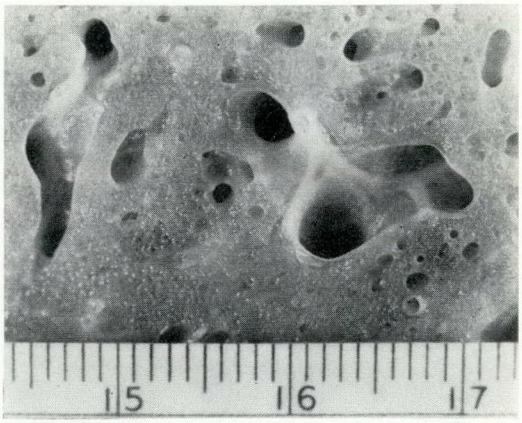

Fig. 4.—J.-Y. M.—Tranche de section du spécimen (lobe pulmonaire inférieur droit) montrant les cavités kystiques bronchiques.

Cas 2.—Sylvain B. a été admis à l'Hôpital Sainte-Justine le 4 juillet, 1961, à l'âge d'un jour et demi pour dyspnée marquée avec tirage sus-et sous-sternal.

A l'examen, on nota une submatité avec diminution du murmure vésiculaire au niveau de toute la plage pulmonaire gauche (Fig. 5).

Le cliché pulmonaire du 4 juillet, 1961, montra une masse opaque oblongue occupant la majeure partie de l'hémithorax gauche, le cœur et le médiastin étant repoussés vers la droite.

Une thoracotomie gauche fut pratiquée le 6 juillet, 1961. On trouva un lobe supérieur gauche énorme, bosselé, de consistance variable mais contenant plusieurs kystes. Le lobe inférieur gauche parut normal macroscopiquement. Une lobectomie supérieure gauche fut pratiquée selon la technique habituelle. La cavité pleurale fut drainée et la paroi thoracique refermée (Fig. 6).

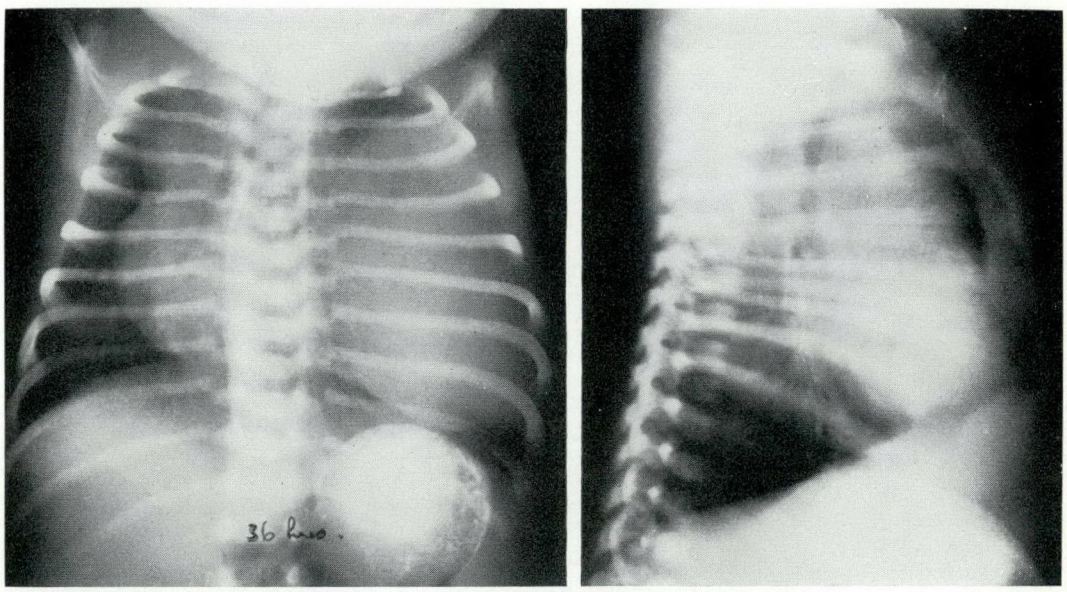

Fig. 5.—S.B.—Poumons (face et profil)—pré-opératoire. Presence d'une énorme masse ovalaire de densité homogène, occupant presque toute la cavité thoracique gauche et déplaçant le cœur et le médiastin vers la droite.

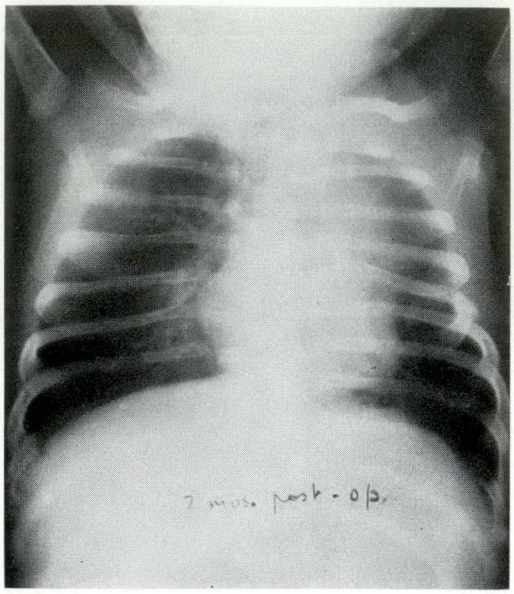

Fig. 6—S.B.—Poumons (face)—deux mois post-opératoire. Disparition de la masse précédemment décrite. Le cœur et le médiastin sont médians. L'aération du lobe supérieur gauche est très légèrement diminuée.

L'évolution post-opératoire fut sans incident. Un cliché pulmonaire fait le 20 juillet, 1961, montra des plages pulmonaires claires avec du parenchyme bien aéré jusqu'au sommet gauche (Fig. 7). Le maiade quitta l'hôpital le 22 juillet, 1961.

Rapport histopathologique.—Lobe pulmonaire supérieur gauche en totalité. A la palpation, on nota une zone indurée qui, à la tranche de section correspondait à du parenchyme fort dense, blanchâtre. On nota, par ailleurs, la présence d'une cavité énorme.

A l'examen microscopique, la zone indurée apparut comme du parenchyme pulmonaire histologiquement normal mais atélectatique par compression. Le kyste central parut tapissé par de l'épithélium bronchique et tout autour du kyste, on nota une adénomatose diffuse consistant en des cavités à revêtement épithélial bronchique (Fig. 8).

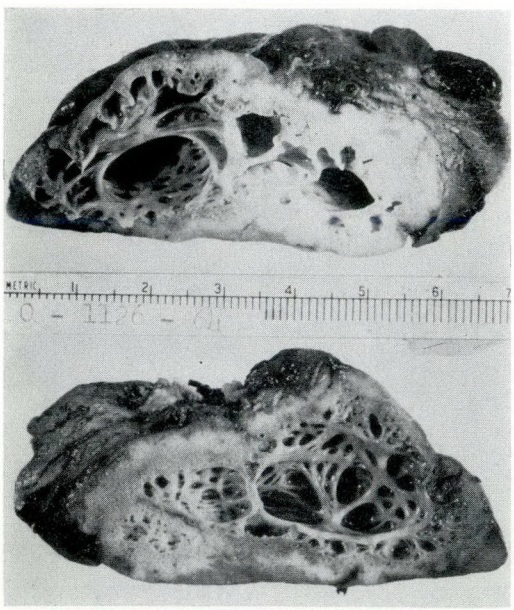

Fig. 7.—S.B.—Tranches de section du spécimen (lobe pulmonaire supérieur gauche) montrant l'image typique du poumon aréolaire consistant en des dilatations kystiques bronchiques.

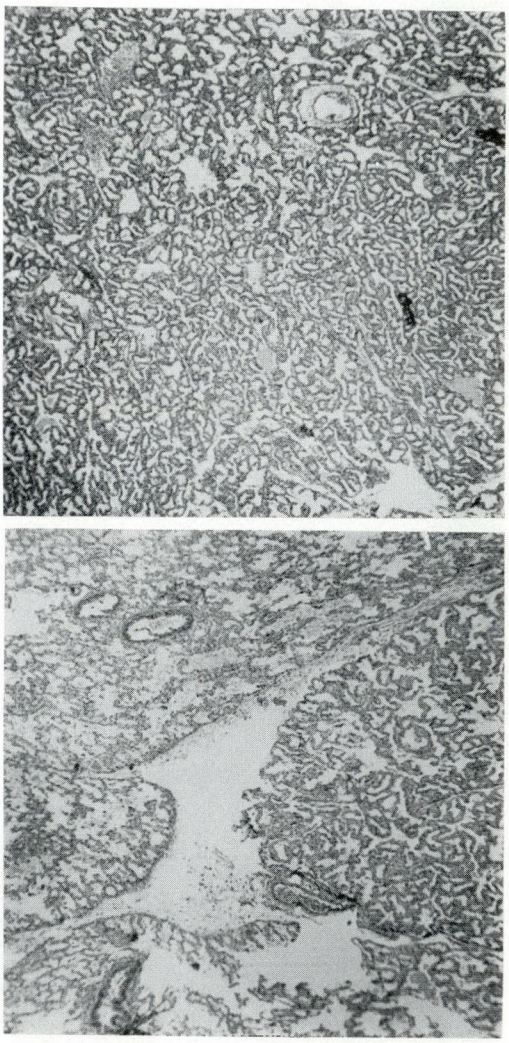

Fig. 8.—S.B.—Coupes histologiques montrant les images de dilatations kystiques bronchiques entourées de tissu pulmonaire sain.

Conclusion.—Etat aréolaire du lobe pulmonaire supérieur gauche avec présence de kystes bronchiques congénitaux.

Conclusion

Les deux cas que nous avons opérés présentaient des lésions typiques de kystes bronchiques multiples congénitaux occupant un lobe pulmonaire. Les images macroscopiques (Figs. 3 et 6) étaient caractéristiques du "Poumon aréolaire" ou "Wabenlunge".

Cependant, sur certaines coupes (Fig. 9), l'image histologique pouvait faire penser à des dilatations lymphatiques anormales comme Virchow et Klebs l'ont déjà affirmé, alors qu'en réalité, il s'agit plutôt de kystes bronchiques.

Par ailleurs, la résection du lobe pulmonaire atteint a amené la guérison dans les deux cas; et nous pouvons ajouter que ce sont les deux plus jeunes patients opérés avec succès pour kystes congénitaux du poumon qui aient jamais été présentés dans la littérature médicale.

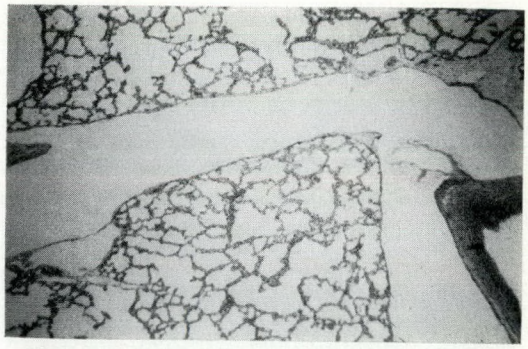

Fig. 9.—J.-Y. M.—Coupe histologique montrant des dilatations kystiques bronchiolaires.

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Summary

Congenital cystic disease of the lung cannot be considered as a pathological curiosity although it is an uncommon condition.

Since the first case reported by Fontanus in 1638, numerous articles dealing with that condition have been published. Up to now, more than 600 cases have been recorded in the literature.

Several theories have been put forward in order to explain the pathogenesis of the disease. However, the great majority of writers have come to the conclusion that the condition is a developmental defect.

From a pathological point of view, the disease may be divided in two groups: (a) a large solitary cyst compressing the surrounding lung parenchyma; and (b) multiple cysts which may be multi- or uniloculated.

The disease may produce symptoms of respiratory distress or may be asymptomatic.

A chest radiograph usually shows the picture of a parenchymal opacity with displacement of the mediastinum and the heart on the contralateral side. The treatment of choice is pulmonary lobectomy.

Two typical cases in young infants have been presented: (One of 10 days old and the other 1½ day old.) Both have undergone a pulmonary resection.

Those two cases are believed to be the youngest patients successfully operated on for that condition.

HERNIATION OF THE HEART—A HAZARD OF THORACIC SURGERY: REPORT OF TWO FATAL CASES

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My attention was first drawn to the possibility of herniation of the myocardium in 1960, by a report by Munchow *et al.*[1] which described a young woman who developed cardiac arrest after she had been stabbed in the chest with an ice pick. Cardiac tamponade had preceded the cardiac arrest. Emergency thoracotomy was carried out, the pericardium was opened and, as the heart was massaged, cardiac activity returned. A 2 mm.-perforation of the right ventricle was repaired and the pericardium was left partially open. At the conclusion of the procedure, the patient was alert and her vital signs were stable. About 62 hours after the first episode of cardiac arrest the patient again developed asystole. Emergency thoracotomy was performed. The pericardium was edematous and thickened. The left ventricle had herniated through the opening left for drainage following the previous resuscitation. The heart was in asystole. Complete reopening of the pericardium, rhythmical compression of the heart and intracardiac injection of calcium chloride and epinephrine restored cardiac action. The pericardium was left completely open. The patient recovered.

Two fatal cases of this unusual complication were recently encountered in a general surgical practice.

Case 1.—A 54-year-old man, an asbestos mill worker for 38 years, had had chronic bronchitis for many years. He had experienced progressive dyspnea over the last few years. Throughout the previous year his cough had become more hacking and recently his sputum had been blood streaked. His fingers were clubbed. His chest was full of rales.

Pulmonary function tests showed a decrease of 30% in his volumetric and mechanical reserves. When bronchodilators were administered, his maximal breathing capacity increased from 75% to 89%. His sedimentation rate was 41 mm./hr. (Westergren). Appropriate laboratory tests and bronchoscopy were negative.

The patient was accepted for thoracotomy. The right chest was entered. A large mass was found in the right lower lobe and a small one in the middle lobe. A radical pneumonectomy was performed with intrapericardial ligation of the vessels. The pericardium was left open.

The pathologist reported that the lesions were epidermoid bronchopulmonary carcinomas. Asbestosis was present in all three lobes. Postoperatively, the drainage tube was connected to active suction by mistake. As soon as the patient was turned on his back a superior vena-caval syndrome developed, with tachycardia and a fall in blood pressure. The anesthetist on duty disconnected the suction as soon as the error was discovered. When the patient did not respond, the right thoracotomy wound was opened and the heart was found to have herniated into the right chest. Massage was instituted and the heart was repositioned inside the pericardial sac.

Immediate and complete recovery followed. The chest was closed. Two hours later all was normal and the patient was conscious.

Two days later the patient developed severe dyspnea and died. A clinical diagnosis of acute right heart failure was made. Postmortem examination revealed that all sutures and ligatures were intact. The changes in the heart resembled those seen in chronic cor pulmonale, but herniation had not occurred again. Metastases from the bronchogenic carcinoma to the left kidney was present.

Although this potentially fatal type of myocardial herniation was corrected immediately the episode seems to have been too much for this already seriously handicapped patient.

Case 2.—A 59-year-old man who was completely asymptomatic had a chest radiograph at work. Twenty-four years previously his left arm had been amputated because of a fibrosarcoma. He smoked, but not heavily. Bronchoscopy was negative. The cytological examination of the sputum revealed suspicious cells, Class III. A radical pneumonectomy was done, with glandular dissection of the mediastinum. The pericardium was left open.

Four hours postoperatively the patient suddenly became cyanotic and lost consciousness, His respiratory rate dropped to 10 per minute

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and his blood pressure dropped to zero. External cardiac massage was instituted and the patient was intubated. The right thoracotomy wound was opened and the heart was found to be herniated into the right chest. It was repositioned and massaged. Ventricular fibrillation developed, the heart was defibrillated, but in spite of the usual measures its vigour gradually diminished until all heart action ceased. Pathological examination of the lung revealed the presence of an anaplastic carcinoma.

Discussion

The two cases reported in this communication gave rise to considerable soul-searching, particularly in view of the fact that the patients died despite early and vigorous resuscitative measures.

Because the heart is firmly enclosed in the pericardial sac, herniation is impossible unless the pericardium has been opened. As the heart herniates from the pericardial sac, a twisting of the low pressure areas (i.e. the superior and inferior vena-cavas) occurs. Cardiac output rapidly falls with peripheral stagnation.

If the superior vena cava is twisted sufficiently, a superior vena-caval syndrome is produced. Disturbances of rhythm develop quickly. For practical purposes, the clinical problem presented by these patients is similar to cardiac arrest.

As a result of this experience, measures have been taken to prevent recurrence of herniation of the heart. This complication has not recurred because the pericardium has been partially closed with a plastic sheet (Fig. 1). The particular material used was Teflon mesh, which is pliable and quite satisfactory for this purpose. Routinely now, the pericardium is closed with this mesh whenever there is any possibility of herniation.

Summary

Two cases of herniation of the heart following radical pneumonectomy are reported.

In both instances the pericardium had been left open following intrapericardial ligation of the pulmonary vessels.

In spite of prompt and aggressive resuscitative measures both cases terminated fatally.

This complication can be prevented if Teflon mesh is used for pericardial closure.

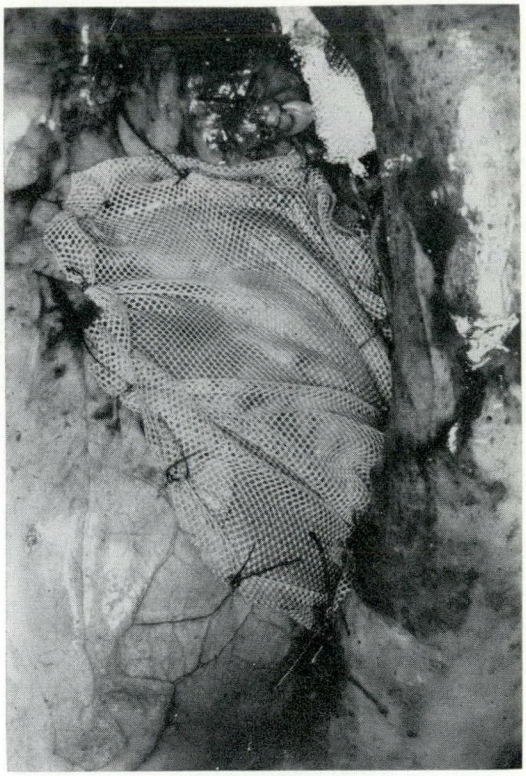

Fig. 1.—Partial closure of the pericardium with Teflon mesh.

Reference

1. Munchow, O. B. G. *et al.*: Cardiac arrest due to ventricular herniation, *J. A. M. A.*, **173:** 1350, 1960.

Résumé

Deux cas de torsion du cœur après pneumonectomie radicale pour cancer pulmonaire sont rapportés.

Dans chacun des cas un évidement médiastinal avait été fait, le péricarde avait été ouvert et les vaisseaux sectionnés à leur origine. L'ouverture ainsi laissée dans le péricarde permit que le cœur glisse en dehors du sac péricardique et devienne hernié.

A ce moment les zones de tention basse telles les deux veines caves se tordent et arrêtent le retour veineux au cœur droit. Les troubles du rythme ne tardent pas à suivre. Le tableau clinique est celui d'un arrêt cardiaque.

Maintenant nous prévenons cette luxation du cœur grace à une prothèse sous forme de tricot de Teflon. Dans tous les cas ou nous l'avons employée cette prothèse semble satisfaisante.

BILATERAL SUBCUTANEOUS RUPTURE OF THE QUADRICEPS TENDON: REPORT OF A CASE WITH DELAYED REPAIR

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SIMULTANEOUS bilateral rupture of the quadriceps tendon is an exceedingly uncommon condition. Only 10 such cases were reported in the English medical literature before 1957.[1] The majority of these were treated soon after diagnosis. The present case is exceptional in that a period of nearly five months elapsed between the time of the injury and the time of definitive repair.

CASE REPORT

T.H.J., a tall well-built 49-year-old man, injured himself in an unusual manner while mounting a horse. After he placed his left foot in the stirrup, the saddle slipped as he was about to mount and he fell to the ground, landing with both knees flexed. His body fell backwards over his legs which were doubled up beneath him. He was unable to rise or walk following the accident. He was taken to his home where he remained for three weeks. He was later transferred to a local hospital for further investigation and treatment. Radiographs of both knees revealed no evidence of fracture and the patient was started on intensive physiotherapy treatment. Gradually he began to walk with difficulty with the aid of two canes and by a circumduction motion of the hips. Over the next five months, the patient was unable to carry out his duties which involved walking and active use of the knees. He also reported that he had fallen three times due to instability in his lower limbs. He was unable to climb stairs alone.

I examined the patient on October 1, 1962, and noted that he was unable to elevate the extended limb on either side. He was also unable to extend the knee from a flexed position. A gap was noted just above the patella bilaterally (Fig. 1). Neurological examination was entirely negative. A diagnosis of bilateral rupture of the quadriceps tendon was made.

Radiographs of both knees at this time revealed marked calcification in his suprapatellar regions, a picture resembling myositis ossificans.

The right leg was operated upon on October 5, 1962, the left leg on October 15, 1962. Doubly curved incisions were used (Fig. 2) to skirt the patella. The findings at operation were almost identical in the two limbs. The quadriceps tendon was completely avulsed from the superior border of the patella. There was some attachment of the vastus medialis muscle on the medial side but all tendinous fibres were completely disrupted. There was abundant calcification in the distal portion of the quadriceps muscle. Considerable fibrosis and scarring were present, binding the quadriceps muscle to the femur. All scar tissue was excised. The quadriceps muscle was freed from the femur and the end was freshened. The patella was mobilized and, when sufficient mobility had been obtained, it was demonstrated that the lower end of the tendon and the upper border of the patella could be approximated. A strip of fascia was taken from the tensor fascia lata with a fascial stripper and a fascial weave was used to approxi-

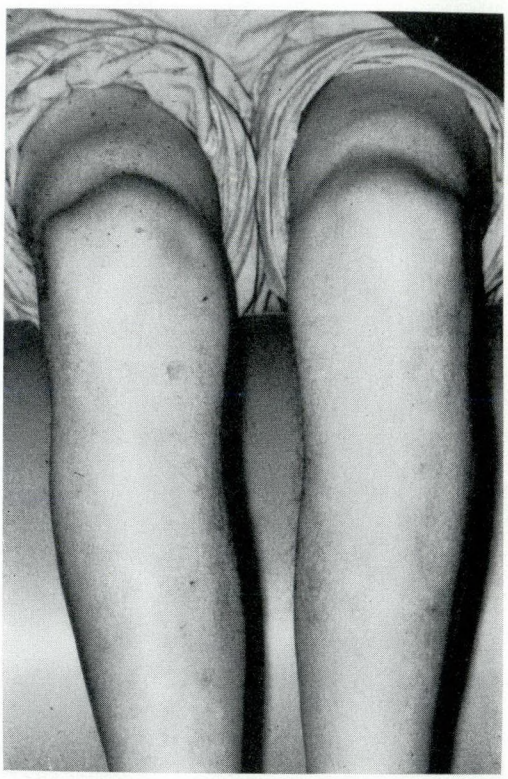

Fig. 1.—Preoperative appearance of legs, nearly six months after injury. Gaps above patella are clearly shown.

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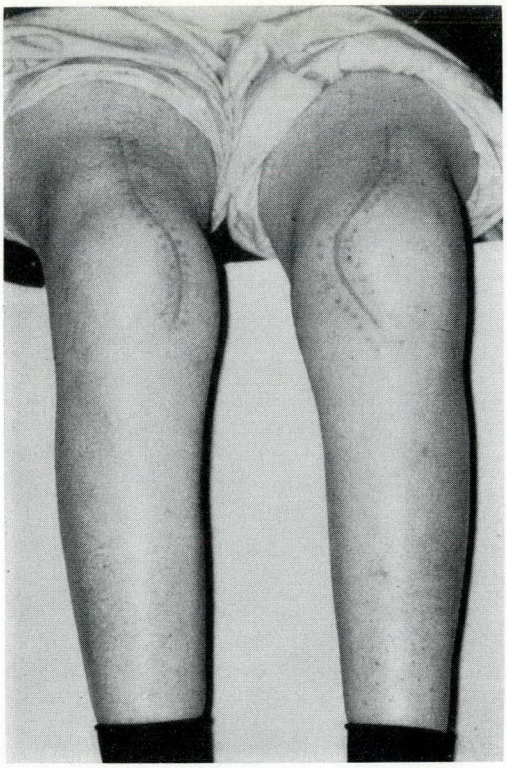

Fig. 2.—Postoperative appearance of limbs.

mate the quadriceps tendon to the patella. There was a reasonably good cuff of tissue just above the patella to secure the fascia and it was not necessary to tunnel the fascia through bone. This double layer of fascial weave was reinforced with a number of mattress sutures of chromic catgut. A Bunnell type of pull-out wire suture was not used to take the tension off the suture line. The wound was closed in layers and the limb was immobilized in a long-leg, plaster-of-Paris cast with the knee in full extension. An identical repair was performed on the opposite leg, 10 days later. Following the second procedure, however, the patient developed deep venous thrombosis in one leg, complicated by pulmonary embolism and infarction. The patient was placed on anticoagulants and his condition gradually improved.

Each limb was immobilized in a plaster for a period of six weeks. The wounds healed by primary intention. He was then started on intensive physiotherapy and walking exercises. He was soon able to walk with the aid of crutches and at the time of discharge was only using one crutch. Postoperative views of his limbs taken 18 months after operation are shown in Figs. 3-5. The patient had normal flexion of both knees but there was still about 15° short of full extension. He returned to his regular duties three months after operation.

Discussion

Bilateral simultaneous rupture of the quadriceps tendon is an uncommon lesion. Delay in repair of the duration discribed in this case is also exceedingly rare. The mechanism of injury is believed to be sudden contraction of the quadriceps muscles against resistance, or a hyperflexion injury as in the present case.[2] Most quadriceps rupture occur as a result of a fall down stairs in which both knees are doubled beneath the body. Pre-existent tendonitis or systemic disease have been incriminated to account for the subcutaneous type of rupture. DePaoli and Acerbi[3] list diabetes, syphilis, nephritis and arteriosclerosis as possible etiological factors. This condition

Fig. 3.—Degree of flexion of right knee, 18 months later.

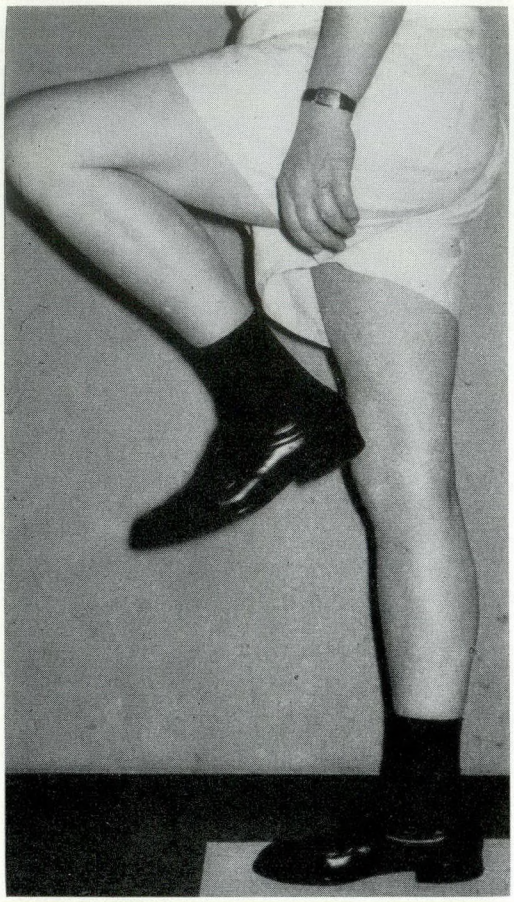

Fig. 4.—Range of flexion of left knee, 18 months after operation.

occurring in conjunction with chronic nephritis was described by Wilson[4] in 1957. Some authors feel that these patients have a degree of tendon or muscle degeneration because most cases occur in individuals over the age of 50 years.[3]

The diagnosis should provide no difficulty. A history of a fall or a hyperflexion injury, followed by complete inability to extend the flexed knee or an inability to raise the extended limb suggests the diagnosis. The palpation of a gap above the patella (although this may be obscured by hematoma) should confirm the diagnosis. Radiographs soon after the injury reveal no bony injury. Later, calcification in the lower end of the quadriceps tendon, suggestive of myositis ossificans, may be seen.

Treatment should be by immediate surgical repair. Some authors recommend that the patella be excised in all cases of quadriceps rupture.[3] Others introduce sutures through drill holes placed in the upper border of the patella. Hinkamp and Pellicore[1] recommend the use of a pull-out wire of the Bunnell type to remove tension from the suture line. Many types of suture material, mostly non-absorbable, including wire, silk and fascia, have been recommended. All authors feel that immobilization should be continued for five to six weeks.

The results of treatment are good following immediate repair but are uncertain following late surgical repair. Full flexion usually returns, although there may always be some lack of full extension. The functional results are generally satisfactory.

Summary

A case of bilateral simultaneous rupture of the quadriceps tendon with delayed repair is described. The unusual nature of the injury is discussed and the types of repair are reviewed. In the present case, fascia lata was used to reconstruct the tendon.

The author wishes to thank Mr. A. Smialowski for the preparation of the photographs.

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4. Wilson, J. N.: Bilateral rupture of rectus femoris tendons in chronic nephritis, *Brit. Med. J.*, **1:** 1402, 1957.

Résumé

La rupture bilatérale des tendons quadriceps, est une condition très rare qui jusqu'en 1957 ne comptait que 10 cas dans la littérature anglaise. L'auteur signale un cas personnel dont le délai entre le traumatisme et la réparation se chiffre à cinq mois. Il s'agit d'un homme âgé de 49 ans qui lors d'une chute de cheval, tomba assis sur ses membres inférieurs et ruptura ses deux tendons quadriceps. Le patient ne pouvait marcher, ne pouvait descendre des escaliers et tombait fréquemment. Ce n'est qu'après un délai de cinq mois que le malade consulta l'auteur. A l'examen,

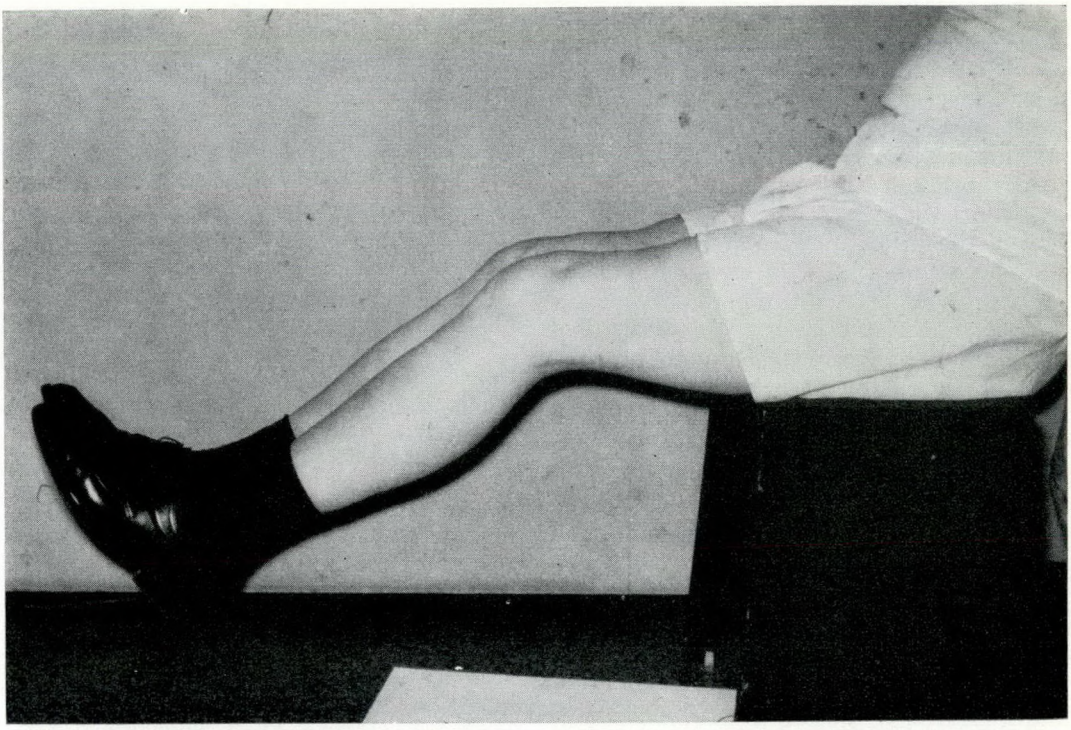

Fig. 5.—Note lack of full extension in both knees, 18 months later.

l'extension des jambes était impossible. On sentait une faille susrotulienne des deux côtés. La radiographie montrait des calcifications simulant le myosite ossifiante. A l'opération il existait un arrachement complet du tendon du quadriceps sur les rotules. La réparation se fit à l'aide de fascia après avoir fait l'excision de tout le tissu calcifié et adhérentiel. Dans les suites post-opératoires un plâtre en extension complète fut installé pendant six semaines. La récupération fonctionnelle dans les trois mois qui ont suivi l'opération a été complète, il ne persistait qu'une limitation partielle de l'extension des deux jambes. En discussion, l'auteur signale la rareté de cette lésion. Il en explique le mécanisme de production. Le diagnostic est habituellement facile, le traitment consistant à la réparation chirurgicale immédiate. Les résultats fonctionnels sont excellents.

PRINCIPLES OF BONE X-RAY DIAGNOSIS. 2nd ed. George Simon. 191 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1965. $15.25.

The second edition of this book, based on the original concept of classifying radiological changes according to their appearances and not according to the responsible disease, has been improved a number of ways. This has been achieved principally by the addition of 52 illustrations and by some revision in the text on the clinical aspects of bone diseases.

This approach to the classification of X-ray appearances presents certain problems, however, since in common conditions reference to several different sections of the text is often required. This difficulty is particularly evident in generalized disorders of the skeletal system.

There are some inaccuracies. Under "Congenital Dislocation of the Hip," the Van Rosen technique is accurately described in the text but in the accompanying Fig. 37, the leg is not abducted to the essential 45° and the reference line is drawn along the axis of the neck of the femur instead of the axis of the shafft of the femur. Fig. 54, a case of "dysplasia epiphysalis hemimelica" is listed as possible chondrodysplasia. For a number of illustrations a possible diagnosis is given, which seems inappropriate and misleading to this reviewer in a book emphasizing principles of diagnosis.

The illustrations are, in general, of good quality and there is a brief but useful bibliography at the end of the book.

In spite of some imperfections this is a good textbook, particularly adapted to the use of students and residents in radiology and orthopedics, and would be useful in any radiological library.

SECRETAN'S DISEASE

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THE clinical entity characterized by solidifying dorsal edema of the hand, complicating relatively minor trauma was first described by Henri Secretan.[1] The English translation of the title paper is "Hard edema and traumatic hyperplasia of the dorsum of the metacarpus". Secretan reported 11 cases in 1901. This syndrome has also been called peritendinous fibrosis of the dorsum of the hand. Secretan emphasized that an essential feature of this condition is the history of relatively minor trauma resulting in a a disproportionate dorsal edema which is persistent, hard and begins in the distal half of the metacarpals. He also mentioned the disabling limitation of finger flexion with the conspicious exclusion of any disturbance of thumb movements. Secretan differentiated two types of edema in these cases. The commonest he called a "benign" type which resolved in two or three months; and the second type he described as "hyperplastic", in which sclerotic fibrous tissue embeds the extensors of the fingers and partially immobilizes them. Although he describes no histological changes, Secretan reported the operative findings in one case as uniform fibrous tissue, thick, grey-white and hard, which embedded the extensor tendons of the fingers on their volar aspect. He proposed that in these cases after relatively mild trauma, the contusion is complicated by the production of a diffuse exudate which infiltrates the lax cellular tissue of the tendons (paratenon). Although the edema usually resolves, the exudate may organize and immobilize the extensor tendons of the fingers culminating in the hyperplastic variant.

Van Denmark, Koucky and Fischer[2] reported two cases of peritendinous fibrosis of the dorsum of the hand in 1948. The excised tissue consisted of dense collagenous tissue with intracellular and extracellular iron pigments identified with Prussian-blue stain. Boyce, in the revised edition of Bunnell's "Surgery of the Hand",[3] includes Secretan's disease with the post-traumatic reflex dystrophies. He suggests that the edema component predominates in Secretan's disease and this feature distinguishes it from causalgia, Sudeck's atrophy and the vascular reflex dystrophies.

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CASE REPORT

In September 1963, J.W., a 41-year-old white man, sustained a "blunt" closed injury to the left hand while at work on a railway section gang. A tender swelling developed gradually during the next few days on the dorsum of the left hand. Radiographs one week after the injury were negative for fractures. Despite physiotherapy and analgesics the tender swelling persisted and an operation was advised. When the patient was admitted to hospital for surgery on January 21, 1964, examination revealed a fixed, tender, solid swelling over the distal two-thirds of the dorsum of the left hand. There was limitation of all movements of the metacarpophalangeal and interphalangeal joints of the fingers. The thumb movements were completely free. His hemoglobin was 15.3 g. and the erythrocyte sedimentation rate (ESR) was 19 mm./hr. A radiograph of the left hand was negative for bone pathology.

On January 22, the dorsum of the left hand was explored under general anesthesia and tourniquet. A diffuse "pancake-like" mass of yellow sclerotic tissue with a maximum central thickness of 3 cm. was identified, infiltrating the extensor tendons. In the vertical plane the mass extended from the metacarpal bases to the proximal interphalangeal joints. Horizontally the mass was confined to the medial four digits. Frozen-section microscopy showed the tissue to be a benign granuloma. The pathological tissue was excised without any attempt to dissect away the diffuse infiltration of the extensor mechanism.

The specimen was grey-white, interspersed with yellow streaks and was firm and rubbery in consistency. The specimen measures 6 x 5 x 3 cm. Microscopy (Fig. 1) showed dense, hyalinized collagenous tissue in a background of adipose tissue. There were focal areas of calcification and non-specific inflammation. The diagnosis was that of sclerosing lipogranulomatosis. Prussian-blue staining was positive for minimal amounts of iron pigments. The immediate postoperative course was satisfactory

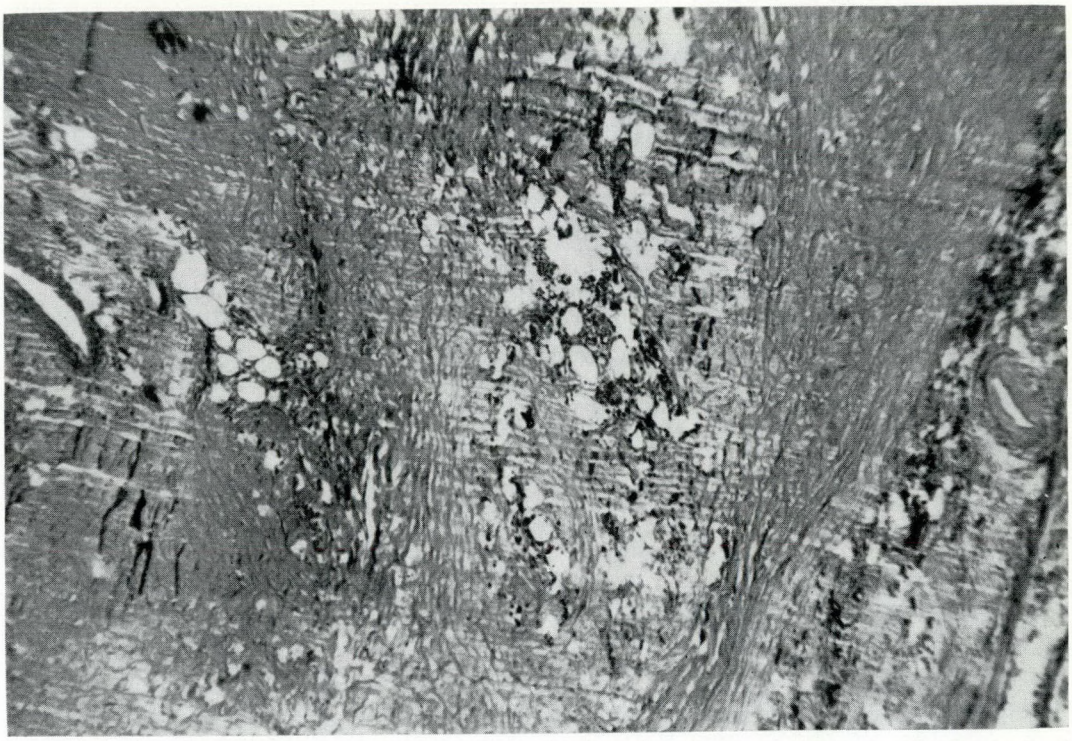

Fig. 1.—Microscopic section showing dense, hyalinized collagenous tissue in a background of adipose tissue.

and the function of all the finger joints improved. When seen one year after operation, this man had no recurrence of the tumour and the immediate postoperative improvement was maintained.

Discussion

The case presented here seems to illustrate the essential features of the hyperplastic variant of Secretan's disease. The history of a solidifying dorsal edema following a relatively minor hand injury was prominent in this case. The subsequent operative findings were those of a sclerotic type of tissue embedding the extensor mechanism with the associated restricted finger movements, but characteristic absence of thumb involvement. Sudeck's atrophy was excluded by the absence of characteristic spotty osteoporosis in the radiograph of the hand. Although the swelling was tender, it was not the excruciating pain characteristic of causalgia. Van Denmark, Koucky and Fischer[2] suggested that hemorrhage played a vital role in the pathogenesis where subsequent hemosiderin formation excited the formation of infiltrating fibrous tissue. These authors go on to explain that the hemorrhages occur in the subaponeurotic layer and therefore may not be visible externally after relatively mild trauma. The excised tissue in the case presented was composed of infiltrating fibrous tissue, with a positive Prussian-blue stain, but the main feature on microscopy was a sclerosing lipogranulomatous process.

Conservative management, including voluntary exercises without passive manipulations, is recommended. If an exploratory operation for pseudotumour is carried out, as in the case reported, a conservative dissection and excision of pathological tissue is advocated.

Summary

A case is reported of solidifying dorsal edema of the hand complicating relatively minor trauma. This clinical entity, first described by Henri Secretan in 1901, is best classified with the post-traumatic reflex dystrophies, in which the edema compon-

ent predominates. Conservative treatment is advocated; however, an exploratory operation may be necessary in the case of a pseudotumour (as in the present instance) but a conservative dissection is suggested. The underlying pathology in the case described herein was a sclerosing lipogranuloma.

The author wishes to thank the staff of the Department of Pathology, St. Paul's Hospital, Vancouver, B.C., for the histology and photograph.

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Résumé

L'auteur rapporte un cas d'œdème dur de la face dorsale de la main, complication d'un traumatisme bénin. Cette entité clinique, décrite pour la première fois par Henri Secrétan en 1901, peut être classée dans la catégorie des dystrophies réflexes post-traumatiques, dans lesquelles prédomine la composante œdème. On conseille un traitement conservateur, mais il peut être nécessaire (comme dans le présent cas) de procéder à une exploration chirurgicale dans le cas d'une pseudo-tumeur. On propose alors une dissection conservatrice. Dans le cas qui nous occupe, la pathologie profonde était un lipogranulome sclérosant.

BIOPHYSICAL MECHANISMS IN VASCULAR HOMEOSTASIS AND INTRAVASCULAR THROMBOSIS. Edited by Philip N. Sawyer. 379 pp. Illust. Appleton-Century-Crofts, Inc., New York, 1965. $8.95.

This volume is a detailed report of a conference involving members of many specialties, including vascular surgery, hematology, biophysics and engineering, concerning "the relationship between interfacial phenomena, thrombosis and hemostasis". Fifty scientists contributed to the various sessions on: fundamental electrokinetic phenomena; relation of basic electrochemical phenomena to the vascular tree; rheology of blood flow; bioelectric phenomena, surface effects, blood clotting and thrombosis; and electrochemistry, surface physics and insight into construction of a suitable vascular interface. Panel discussions are included, in addition to the individual papers presented at each session, and both historical and special references are listed. It should be of special value to scientists interested in blood and the vascular system and research related to the cardiovascular system.

TRAITE DE THERAPEUTIQUE CHIRURGICALE. Edited by J. Senèque. Tome I. Chirurgie orthopédique et traumatologie des membres-rachis-bassin. R. Judet and J. Judet et al. 733 pp. Illust. Masson & Cie, Paris, 1964. 156 NF. $31.20 (approx.).

Ce premier tome du nouveau traité de thérapeutique chirurgicale est consacré à la chirurgie orthopédique et traumatologie.

L'analyse, en quelque cents mots, de ce volume risque de ne pas rendre justice aux auteurs. Le seul résumé de l'introduction de six pages nous fait envisager immédiatement toute l'importance apportée par les auteurs au choix judicieux qu'il faut accorder au traitement orthopédique et au traitement opératoire pour le traitement d'un certain nombre de lésions.

La connaissance de l'évolution de certaines malformations osseuses pose souvent à l'orthopédiste des problèmes embarrassants — telles les sub-luxations de la hanche. Certaines maladies; la tuberculose ostéo-articulaire, les séquelles de polio, l'ostéomyélite diminuent; par contre, la traumatologie augmente.

L'expérience considérable des Judet en ce domaine ressort dans le traitement des fractures ouvertes, dans celui de leurs complications: pseudarthrose, infection, blessures nerveuses et embolies. Chaque chapitre du volume est appuyé sur la pathologie du tissu osseux et il est l'expression de l'expérience personnelle "appuyé d'une solide information". Toute la traumatologie du rachis, des membres supérieurs ou inférieurs, du bassin de même que les problèmes orthopédiques rencontrés à ces mêmes niveaux est étudiée de façon systématique après que les généralités et la pathologie générale ont été exposés dans les premières cent pages.

Comme le dit P. Lance "les indications de l'une et de l'autre méthode orthopédique ou opératoire sont balancées avec soin, compte tenu de l'âge du sujet, du temps d'évolution, du tempérament et des habitudes du chirurgien".

C'est un volume que tout chirurgien orthopédiste doit avoir et qu'il trouvera grand profit à consulter.

THE EFFECT OF HYPERBARIC OXYGENATION ON MYOCARDIAL INFARCTION IN DOGS

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SINCE the recent revival of hyperbaric oxygenation (OHP) as a therapeutic adjunct by Churchill-Davidson, Sanger and Thomlinson,[1] by Boerema,[2] and by Illingworth *et al.*,[3] a major interest has been in the effect of such treatment on myocardial ischemia and infarction. A number of experimental studies indicate that OHP therapy causes a decrease in ventricular fibrillation, in infarct size, or is otherwise beneficial but several workers report no benefit.[4-12] OHP has been used, on a trial basis, for human myocardial infarctions with equivocal results to date.[13] This study, which commenced in 1962, investigated the effect of OHP on the extent and incidence of myocardial infarction, and on the prevalence of ventricular fibrillation in 130 dogs subjected to coronary artery ligation.

METHODS

The plan in general was to tie a coronary artery as similarly as possible in two groups of dogs (*vide infra*), exposing one group to OHP at 2 or 3 atmospheres absolute (ata), the other to air at 1 ata, for various times up to 24 hours. In addition, the oxygen was humidified in some experiments. Pulse, respiration, blood pressure, animal and pressure chamber temperature, arterial blood pO_2 (paO_2), pCO_2 ($paCO_2$), pH and hematocrit were recorded. Electrocardiograms were performed throughout. Gross and microscopic pathological studies were carried out. The dog was used because, although there is some disagreement over the coronary anastomoses in dogs (the consensus favours the existence of abundant large anastomoses[14-18]), there is quite marked disagreement concerning the pig's anastomoses.[19, 20] Ligation at about the mid-point of the anterior descending branch of the left coronary artery was chosen to produce a definitely cyanotic, ischemic zone approximately the distal half of the anterior left ventricle, so that ventricular fibrillation would not occur too frequently or promptly, while infarction frequency would be high in untreated dogs. Since survival for several hours is too short for accurate evaluation of infarction without special stains, even though irreversible ischemic damage occurs 30 to 60 minutes after coronary ligation,[21-26] it was desirable to have a number of at least four to six-hour survivors. When mid-point ligation did not produce the desired area of cyanosis and ischemia a further tie was placed higher up or suitable side branches were tied. It was realized that the cyanotic epicardial area did not accurately represent the true amount of myocardial ischemia because coronary size, distribution, collaterals and coronary flow are so variable. Circumflex ligations were performed later in the study. Arterial blood pO_2, pCO_2 and pH studies were carried out using the Instrumentation Laboratory Inc. pH and Blood Gas Analyzer, Model 113. Samples taken from animals at 2 or 3 ata into room atmosphere gave readings with a high paO_2 of 2050; with the analyzer in the tank, top readings for paO_2 were 2200 to 2400.[27, 28] A direct reading microhematocrit centrifuge was used to check the blood and provide information relative to the occurrence of hemorrhage, shock, and the hemoconcentration associated with the congestive effects of oxygen at increased pressures.[29-31]

PROCEDURES AND OBSERVATIONS

For clarity, the groups studied, the main variations in procedure, in each group, and the relevant observations in each will be described. Observations on lung and brain changes and on blood studies will be given separately.

Group I

There were 30 dogs in this preliminary series. Under pentobarbital anesthesia and ventilation with a pneophore positive-pressure demand valve with 100% oxy-

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gen, arterial cannulas were inserted for further anesthesia, heparinization, blood pressure readings and blood sampling. Through a left thorocotomy, the heart was "hammocked" and the anterior descending branch of the left coronary artery was tied near the mid-point to produce a definitely cyanotic, ischemic zone approximating the distal half of the anterior left ventricle. Immediately after ligation the dog, still ventilated through a pneophore valve with 100% O_2 and with chest held open, was placed right side down in the pressure chamber which was brought by ambient air pressure to 3 ata within five to 10 minutes. Animals were then observed up to 24 hours and those still living were then sacrificed. Tank pressure was varied in order to observe the changes that occurred between 1 to 3 ata. Gross and microscopic pathological studies were carried out subsequently.

The cyanotic, ischemic, and usually non-contractile zone produced by coronary ligation became temporarily pink at about the time 3 ata was reached, usually within five to 10 minutes of tank closure in 20 of 27 dogs in which this was noted. The blood in the ligated artery was definitely blue beyond the ligature in six dogs and in each instance OHP converted this blood to a pink colour for a time. Left ventricular veins became red (arterialized) in eight of 20 dogs and the right atrial blood was frequently pink. In 14 dogs, soon after 3 ata was reached, tank pressure was slowly brought to 1 ata and myocardial cyanosis returned, but gave way to pinkness again when 3 ata was re-established. The pinkness of the ischemic zone changed within an hour or so to a dry, leathery, mahogany-coloured, non-contractile area, smaller in size than the original zone of ischemia. On microscopic section this area was infarcted. Electrocardiograms showed a typical infarction pattern. While under OHP, 3 ata, four of the 30 dogs died of ventricular fibrillation, three early, one late.

Two other animals being prepared for this group, fibrillated before pressurization was complete and are listed under Group IVB.

Group II

Thirty-one dogs were treated as in Group I except that after similar coronary ligation the chest was closed, so that 20 to 40 minutes elapsed before the animal was fully exposed to OHP, 2 or 3 ata, up to 24 hours. This time lapse was much less than the earliest time a human might be exposed to OHP after the onset of a "coronary attack". This short interval gave these dogs a chance to recover from the myocardial ischemia before damage leading to necrosis was complete, since it is known that irreversible ischemic damage occurs 30 to 60 minutes after coronary ligation.[21-26] These dogs were placed on the right or left side and respiration was unassisted following chest closure. Endotracheal tube, tracheotomy plus short wide tube, or natural airway was used.

Of 24 dogs exposed to OHP, 3 ata, four died of ventricular fibrillation, 11 died in standstill, usually in 10 to 18 hours, nine lived to sacrifice at 22 to 24 hours. All showed extensive infarction and the typical electrocardiographic changes of infarction.

Of seven dogs exposed to OHP, 2 ata, two died of ventricular fibrillation, four in standstill, and one lived 24 hours. All but one had extensive infarction. All showed typical electrocardiographic changes of infarction.

Six other dogs, being prepared for this group, died of early ventricular fibrillation while still on 100% O_2, 1 ata, by pneophore valve, and are listed under Group IVB.

Group III

Acid fuchsin staining of microscopic sections of myocardium was done to detect the time of earliest infarction, and the extent of infarction, in 11 additional dogs, prepared as in Group II, and exposed to OPH, one-half at 2 ata, one-half at 3 ata, for either two, four or six hours before sacrifice.

All of these animals showed, with the acid fuchsin stain, extensive heart-wall necrosis, mostly transmural, and varying from focal to complete necrosis even at two hours.

In the two-hour sections, hematoxylin and eosin stain showed edema, congestion, early hemorrhage and some loss of cross-striations, while in most of the four-hour

sections these changes and more definite loss of cross-striation, variations in staining and increasing polymorphonuclear infiltration were seen, and at six-hours, muscle fragmentation was added. Electrocardiograms confirmed infarction in all animals; none fibrillated.

Controls–Group IVA

This group consisted of dogs with left anterior descending coronary ligation exposed to air, 1 ata. In order to check myocardial and lung changes and fibrillation incidence, 12 control dogs had anterior descending coronary ligation as above and the chest was closed while they were being ventilated with room air. They were then allowed to recover from anesthesia and live in room air until sacrifice, six at 24 hours, six at 48 hours.

All of these animals recovered well from anesthesia and moved about freely. Electrocardiograms showed infarction. At sacrifice extensive infarctions were found in all, comparable to the OHP animals. No animal developed ventricular fibrillation.

Controls–Group IVB

Group IVB was comprised of dogs with left anterior descending coronary ligation exposed to 100% O_2, 1 ata. Two dogs of 32 being prepared for Group I, and six dogs of 37 being prepared for Group II fibrillated within two to 15 minutes after anterior descending ligation, while ventilated with 100% O_2, 1 ata. No significant difference in breed, age, weight, physical condition, coronary size and distribution, or estimated collateral circulation accounted for these early fibrillations.

In Group III, of 11 dogs prepared as in Group II, none fibrillated.

In Group VI, 11 of 24 dogs undergoing anterior descending ligation were ventilated with 100% O_2, 1 ata, before OHP exposure. Of these 11 dogs, one died 10 minutes after anterior descending ligation. No plausible determining factor could be found.

With Drs. F. G. Pearson and B. J. Ginsberg, studies were performed on 10 dogs (mentioned under changes related to oxygen toxicity) to determine what lung changes occurred in animals lying continuously anesthetized with pentobarbital in room air for 24 hours. Two dogs had thorocotomy only and eight had thorocotomy plus mid-point anterior descending coronary ligation. Of these eight dogs, two fibrillated at 10 and 15 minutes respectively during chest closure, while still being ventilated with 100% O_2. A third dog died in cardiac standstill in about 30 minutes. Again no factor was identified which explained the development of early fibrillation.

Group V

The circumflex artery was tied within 2 to 4 mm. of origin in 14 dogs. Six were control dogs, ventilated with and exposed only to room air. Eight OHP dogs were ventilated with 100% O_2, 1 ata, a ligature looped around the artery, and when the dog was under OHP for at least 15 minutes, the artery was closed by pulling the ligature loop ends. Three dogs were exposed to 2 ata, five to 3 ata. The pattern, and absolute and relative size of the coronary vessels were noted.

Ventricular fibrillation occurred more frequently in the OHP dogs. Six of eight dogs exposed to OHP died of early ventricular fibrillation, two in standstill at 25 minutes and 4½ hours respectively. Three of six "air-controls" fibrillated early, one died in standstill at 3½ hours, two lived to sacrifice at 22 and 24 hours. These animals all showed typical electrocardiographic signs of infarction and the four that lived long enough, showed large posterior infarcts.[14, 23]

The animals in this group were selected in that they weighed 12 to 14 lbs. They developed ventricular fibrillation readily, a finding which is opposed to Chardack's views.[6]

The origin of the coronary vessels, their absolute and relative sizes, distribution and estimated anastomoses, and the origin of the septal vessel, did not appear to play any part in the results produced by coronary ligation.

Group VI

Tetrazolium staining of 5 mm.-thick slices of ventricular myocardium[12, 32, 33]

was carried out immediately after sacrifice in 20 matching dogs prepared as in Group II above, one of each pair being exposed for three hours to OHP, 3 ata, the other to room air.

Myocardial slices from three dogs were examined to test the efficacy of the stains. These animals had similar ligations and were exposed only to air, 1 ata, for 30 minutes, four hours and 24 hours and showed large areas of unstained myocardium, denoting infarction; but slices from the 30-minute dog showed many scattered spots and irregular islands of purple stain in the "unstained" area; in the four-hour dog, the basal slide of the slice nearest the tie, was almost completely stained, denoting no infarction, while the apical side was unstained, as were both sides of the other slices; and the slices from the 24-hour dog again showed many scattered spots and irregular islands of purple stain in the large "unstained" zones of myocardium. One other dog, on 100% O_2 by pneophore valve, fibrillated 10 minutes after coronary ligation, but was pressurized for 30 minutes at 3 ata while fibrillating and one side of one slice showed a definite unstained zone near the endocardium.

The tetrazolium-stained slices from the OHP dogs compared to air controls, showed as large or larger areas of infarction, and confirmed the opinion that infarcts are usually heterogeneous.[14, 34] Even when large areas of unstained myocardium were found there were usually many scattered spots and irregular islands of purple stain throughout the "unstained" zone (Fig. 1). Moreover the basal and apical side of the same slice often differed greatly (Fig. 2). Usually it would have been impossible to cut out and weigh the "unstained" tissue, and any estimate of the amount of infarction would have been very inaccurate (Fig. 3).[35]

Pulmonary Hepatization from Oxygen Toxicity

The gross lung changes were the most startling find in this study. The majority of dogs exposed for over six hours to OHP, 2 or 3 ata, showed varying degrees of blue-black consolidation or hepatization of both lungs; two four-hour and two six-hour dogs were similarly affected. There was great variation in the extent of this reaction with respect to OHP exposure time.[29, 36-38]

Controls for lung changes were 19 dogs, anesthetized continuously with pentobarbital, and lying in room air, on right or left side for 24 hours, some with endotracheal tube, some with tracheotomy plus short, wide tube and some with natural airway. Nine of these dogs had had no thorocotomy or coronary ligation; while of 10 dogs studied with Dr. F. G. Pearson and Dr. B. J. Ginsberg, two had had thorocotomy only and eight, thorocotomy plus mid-anterior descending coronary tie. In addition the 12 dogs of Group IVA, active in room air 24 to 48 hours after coronary ligation, served as further lung controls. None showed lung hepatization on gross examination; at most, they had areas of superficial edema and congestion.

Micropathology of both lower lobes of 51 OHP and control dogs usually showed, in OHP dogs, severe congestion, atelectasis and edema, and in controls, minimal to moderate, with occasionally severe changes. Our consultants in pathology suggested that the severe micropathology in OHP dogs might be due to the effects of myocardial failure following myocardial infarction, rather than to oxygen toxicity. We have this point under continuing study.

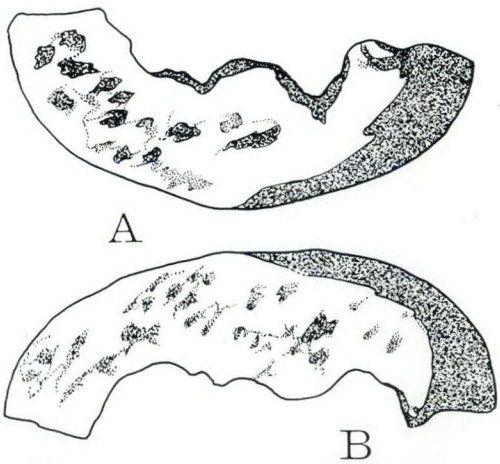

Fig. 1.—Tetrazolium-stained transverse slice, 5 mm. thick, from the mid-point of a typical myocardial infarct to show that the "unstained" (infarcted) area has spots and irregular islands of purple stain throughout; a non-homogeneous infarct. It would be difficult to estimate the size of this infarct correctly. A = basal side; B = apical side.

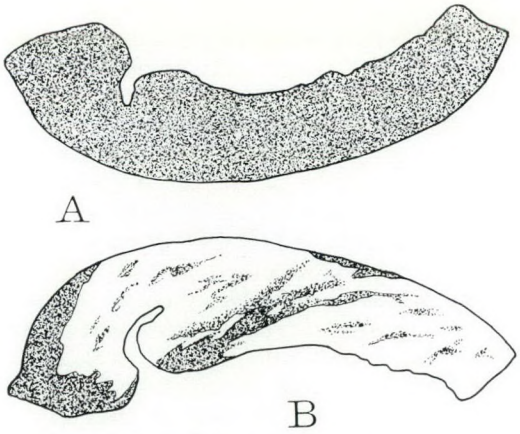

Fig. 2.—Tetrazolium-stained transverse slice from the mid-point of a myocardial infarct to show the marked difference between basal and apical side of a single slice 5 mm. thick. A = basal side, all purple (no infarction); B = apical side, purple one end only, but spots and irregular islands of purple stain throughout the large "unstained" (infarcted) zone.

The gross and microscopic lung changes both in OHP and air-control dogs were usually more marked on the dependent side. The type of airway,[27] positive pressure ventilation,[39-41] pentobarbital, heparinization,[40, 42, 43] or humidity,[44] did not seem to alter the lung changes in either OHP or air-control dogs.

The brains of OHP dogs were freqeuntly scarlet, with marked congestion, at autopsy, while air controls were normal. Dr. N. B. Rewcastle of the Department of Neuropathology, University of Toronto, has found no microscopic lesions, except moderate to severe congestion in the OHP dogs. Cerebral "air" embolism was found in two OHP dogs de-pressurized inadvertently and suddenly.[45]

Arterial Blood pO_2, pCO_2 and pH

The usual paO_2 range for 50 dogs with multiple readings was 1200 to 1800 at 2 or 3 ata. There was no correlation between paO_2 values and the severity of lung pathology, long-surviving dogs with severe hepatization having a high paO_2 as often as a low paO_2, except for animals near death when paO_2 fell rapidy. Nine controls-in-air had paO^2.[65-75]

No relation was noted between the $paCO_2$ and pH readings and the myocardial events or the lung changes, except that with impending death pH fell as low as 6.825 and $paCO_2$ was off the scale — over 100 — on several occasions. Nineteen dogs with marked pulmonary hepatization had a $paCO_2$ ranging 22 to 68, but usually in the 30 to 60 range, with corresponding pH readings of 7.52 to 7.19, usually in the 7.46 to 7.2 range. A number of dogs had a pH of 7.1 to 7.2 with a $paCO_2$ in the 40 range, and probably represented organic acidosis, though lactate and pyruvate estimations were not available for proof.[46]

The Hematocrit

The hematocrit rose during most OHP experiments from a range 25 to 35 up to 45 to 55, corresponding to the congestive changes seen by Karsner[29] and the increased hematocrit noted by Jamieson and van den Brenk.[31]

Discussion

This study was undertaken with considerable bias in favour of OHP as an adjunct to the treatment of coronary ischemia and infarction although it was realized that hyperbaric therapy would require expensive facilities, much space and time, demand specially selected and trained person-

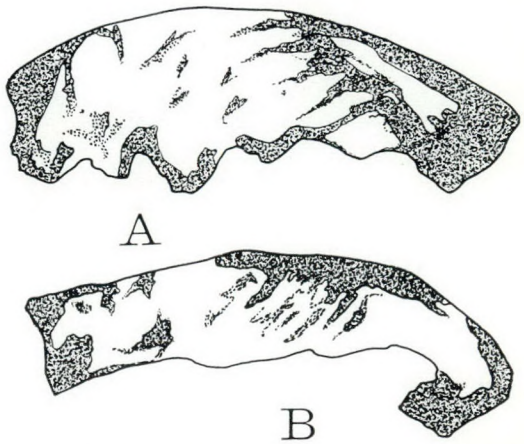

Fig. 3.—Usual appearance of tetrazolium-stained slices; apical side of a 5 mm. thick transverse slice from the mid-point of a myocardial infarct from A = OHP dog; B = matching air control dog. It would be difficult to estimate the size of these infarcts correctly.

nel, and present definite hazards.[47] Furthermore few "heart-attack" patients might reach a hyperbaric facility in time for benefit if an ischemic zone was for the most part irreversibly damaged in 30 to 60 minutes.[21-26] Fortunately coronary ischemic disease is so variable that sparing of myocardium might still be possible, especially in border zones. In this study animals were exposed to OHP immediately after coronary ligation, or within 20 to 40 minutes. OHP did not lessen either ventricular fibrillation or infarction.

The usefulness of oxygen at increased pressure in myocardial ischemic disease has been challenged. Russek, Regan and Naegele[48] reported that in exercising angina patients, given 100% O_2 by mask, the RS-T and T changes were made worse and pain not prevented. Zao[49] pointed out that changes in oxygen gradient may cause fibrillation. Sobol *et al.*,[50] and Eckenhoff, Hafkenschiel and Landmesser,[51] found that 100% O_2 decreased coronary backflow, while West and Guzman[52] demonstrated coronary constriction.

Other possible adverse effects of oxygen at increased pressures, besides the lung and brain toxicity changes, have been noted. Dautrebande and Haldane[53] noted bradycardia, Whitehorn and Bean[54] a prolonged P-R interval and A V block, while vasoconstriction was noted in the retinal vessels by Cusick, Benson and Boothby[55] and Saltzman[27] and many have described vasoconstriction in cerebral circulation.[56-58] Others[59-62] have noted reduced cardiac output and McDowall[63] has suggested that this, plus vasoconstriction, may negate benefits of increased oxygen.

Although oxygen may be used when delivered to tissues at increased pressures, and in physical solution, as evidenced by the experiments of Bunzl *et al.*[64] and many others,[65-69] and although, in OHP, physically dissolved oxygen exceeds overall body metabolic needs, venous blood being 100% saturated, still oxygen storage is quite limited, and organ function fails promptly in total absence of circulation. The myocardium particularly uses constantly large amounts of oxygen, with a large A-V gradient, and in this study OHP did not supply enough oxygen to diminish either ventricular fibrillation or infarction.[27, 70, 71]

That OHP may produce the severe lung damage noted above is well known,[29, 36, 37, 72, 73] and in humans oxygen over 60% becomes increasingly dangerous with time.[74, 75] Only two human deaths from "oxygen poisoning" of the lung are known to the writer, one from OHP reported by Fuson *et al.*,[76] and Spencer[77] reported a death from 100% oxygen.

The incidence of ventricular fibrillation varies tremendously after anterior-descending,[78, 79] or circumflex ligation.[6, 80, 81] None of the usual explanations for this are satisfactory.[6, 82-84] As shown in Table I, the inci-

TABLE I.—Variation of Fibrillation Rates

Rates in dogs exposed to *air*, 1 ata, only

Anterior descending tie, mid-point	*No. of dogs*	*No. of fibrillations*	*%*
Group IV A	12	0	0
Group VI	13	0	0

Rates in dogs exposed to 100% O_2, 1 ata

Anterior descending tie, mid-point	*No. of dogs*	*No. of fibrillations*	*%*
Group I	32	2	6
Group II	37	6	17
Group III	11	0	0
Group VI	11	1	9
Special group controls lung	8	2	25

TABLE I.—Variation of Fibrillation Rates

Rates in dogs exposed to *air*, 1 ata, only

Circumflex tie	*No. of dogs*	*No. of fibrillations*	*%*
Group V	6	3	50

Rates in dogs exposed to *O H P*, 2 or 3 ata

Anterior descending tie, mid-point	*No. of dogs*	*No. of fibrillations*	*%*
Group I	30	4	13
Group II	31	6	20
Group III	11	0	0
Group VI	10	0	0
Circumflex tie			
Group V	8	6	75

dence varied considerably in this study between various groups, but was not related to dog breed, age, sex, weight, coronary size, distribution or estimated collaterals, and is probably too variable to be a good criterion of therapy. In this study fibrillation was more frequent in OHP animals.

Infarct size after similar coronary ties has always varied;[26, 85] so much so that its value has been questioned as a criterion of therapy designed to minimize the consequences of coronary ligation.[86] In this study OHP did not prevent infarction or diminish infarct size.

Early reversion of the S-T segment of the electrocardiogram has been thought to indicate a favourable response to OHP therapy after coronary ligation.[5, 7, 87] However in this study the many animals showing reversion displayed large infarcts, air controls showed reversion as often as OHP dogs, and no relation to fibrillation was noted.

Summary

In this study hyperbaric oxygenation did not prevent or limit myocardial infarction following coronary ligation. Ventricular fibrillation occurred more frequently in hyperbaric dogs than in controls. On gross examination marked pulmonary hepatization changes, characteristic of "oxygen toxicity" were noted from four hours onward in hyperbaric oxygen animals and were not seen in controls.

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Résumé

Cette étude a été enterprise pour évaluer les effets de l'oxygénation hyperbarique (OHP) sur l'ampleur de l'infarctus du myocarde et sur la fréquence de la fibrillation ventriculaire. Ces expériences ont été pratiquées sur des chiens chez lesquels on avait ligaturé l'artère coronaire. On a ainsi étudié, du point de vue des changements de toxicité par oxygénation, 130 chiens soumis à la ligature des coronaires et 19 animaux témoins à l'air libre.

Chez 30 chiens, thorax ouvert, cœur suspendu dans un hamac, on a procédé à la ligature de la coronaire antérieure descendante, immédiatement avant de les soumettre à l'OHP, 2 à 3 atmosphères absolues (ata), pendant diverses périodes, allant jusqu'à 24 heures. Chez 20 d'entre eux la zône bleue d'ischémie devint d'abord rose, mais pour un temps limité. L'infarctus se développa chez tous les animaux. Quatre moururent de fibrillation ventriculaire.

Trente-et-un chiens furent soumis, après fermeture du thorax et dans un délai de 20 à 40 minutes après la ligature de la coronaire antérieure descendante, à l'OHP, 2 ou 3 ata, pendant des périodes allant jusqu'à 24 heures. Tous présentèrent de gros infarctus et six moururent de fibrillation ventriculaire.

Chez 11 autres chiens, avec coronaire ligaturée et thorax fermé, on a coloré à la fuchsine acide des coupes microscopiques du myocarde pour tenter de déceler un début d'infarctus. Ces animaux ont été sacrifié après deux, quatre et six heures d'exposition à l'OHP, 2 ou 3 ata. L'infarctus était présent chez tous.

Chez 12 chiens-témoins, à la pression atmosphérique normale, avec une ligature coronaire similaire, mais non exposés à l'OHP, aucun ne présenta de fibrillation ventriculaire, mais à la nécropsie, 24 à 48 heures plus tard, tous présentaient d'importants infarctus du myocarde.

La ligature de la coronaire circonflexe a été effectuée chez huit chiens soumis à l'OHP et six sujets-témoins à l'air libre. Tous ces animaux, de petite taille, présentaient des signes d'infarctus, soit électrocardiographiques soit pathologiques soit les deux. Six des chiens soumis la pression hyperbarique entrèrent en fibrillation et trois des six témoins firent de même.

La coloration au tétrazolium de coupes de ventricules du myocardie, d'une épaisseur de 5 mm., a été faite après sacrifice de 20 chiens de même taille, porteurs d'une ligature de la coronaire antérieure descendante. Un chien de chaque paire avait été ensuite exposé pendant 3 heures à l'OHP, 3 ata, l'autre à l'air libre. Des infarctus de même dimension approximative ont été trouvés chez tous les chiens.

Pour évaluer la grave hépatisation et l'œdème pulmonaire qui accompagnent l'exposition à l'OHP, 19 chiens-témoins, soumis à une anesthésie continue au pentobarbital, ont été exposé à l'air pendant 24 heures; deux avaient subi une thoracotomie, huit une thoracotomie plus une ligature de la coronaire et neuf n'avaient pas été opérés. L'examen macroscopique ne révéla pas d'hépatisation, mais l'examen micropathologique révéla de la congestion légère, mais parfois modérée ou prononcée, de l'atélectasie et de l'œdème.

On a fait couramment des électrocardiogrammes qui montraient les tracés typiques après ligature des coronaires antérieure et circonflexe et des changements caractéristiques d'infarctus.

La pO_2, la pCO_2 et le pH du sang artériel montraient que les conditions hyperbariques prédominaient chez les animaux soumis à l'OHP, que l'acidose, probablement organique, était courante et qu'il n'y avait pas de corrélation entre la paO_2, la pCO_2, le pH et les graves modifications pulmonaires observées.

Dans cette étude, l'oxygénation hyperbarique n'a ni empêché ni limité l'infarctus qui suivait la ligature coronaire. La fibrillation ventriculaire est survenue plus fréquemment chez les chiens soumis à l'OHP que chez les animaux témoins. Une grave hépatisation s'est produite chez les chiens soumis à l'OHP.

THE KEYS TO ORTHOPEDIC ANATOMY. William A. Miller. 155 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $7.00.

It is difficult to know why this book was written. Its avowed aim is to provide anatomic details pertinent to orthopedic and traumatic surgery, but no attempt is made to explain the application of these details. These details are a dull (but concise) catalogue of anatomic facts of the upper and lower limbs, and back. The clinician would be better advised to underline his own undergraduate textbook of anatomy, and save himself $7.00.

THE ANATOMY OF VARICOSE VEINS. Robert E. Mullarky. 89 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $6.50.

This little monograph contains a series of halftone and line drawings depicting the varied anatomy of the superficial veins of the lower limb and their relations to and communications with the deep veins of the limb. Two points of importance to those interested in the surgery of varicose veins emerge from the study, viz., (a) the considerable number of perforating and communicating veins which are present both below and above the knee, and (b) the inconstant nature of the termination of the short saphenous vein. Those surgeons who treat varicose veins will find this monograph of interest.

ETUDES SUR DIFFERENTES SOLUTIONS EMPLOYEES DANS LA PERFUSION LOBAIRE CHEZ LE CHIEN*

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Nous tenons d'abord à rappeler que nos expériences sur les perfusions lobaires avaient pour but de définir une technique et d'identifier le liquide idéal pour ce genre de perfusion; ceci, dans un but thérapeutique pour certaines maladies pulmonaires, néoplasiques ou infectieuses.

Différentes formes de perfusions pulmonaires totales ont été essayées par certains auteurs[1, 2] mais on constata rapidement que la perfusion pulmonaire chez l'animal, au moyen d'une circulation extra-corporelle, comportait de nombreuses difficultés.[3-5] Creech et ses collaborateurs,[6] ont démontré que la mortalité et les complications augmentent avec la durée de la perfusion. Il est généralement admis que les perfusions sont influencées par de nombreux facteurs. Ceux-ci peuvent se classer comme suit:

1.—Les facteurs physiologiques.—Ils sont principalement dus à la présence de deux systèmes vasculaires perfusant le poumon, à la résistance vasculaire pulmonaire,[3] et à la pression intra-alvéolaire des gaz.

Un autre facteur, extrêmement important et probablement une des causes principales des complications pulmonaires au cours des perfusions extra-corporelles, est la cohésion moléculaire de surface (tension superficielle) de la couche liquide qui revêt les alvéoles.

En physiologie normale, il existe une substance phospholipidique lipoprotéinique appelée en anglais "surfactant", qui empêche l'adhésion des surfaces alvéolaires et l'atélectasie. Si cette substance n'existait pas, la formule de LaPlace ($P = 2\ T/r$) s'appliquerait, empêchant la réexpansion alvéolaire. Au cours des perfusions, cette substance phospholipidique lipoprotéinique est apparemment détruite ou éliminée et on assiste à une agglutination des surfaces alvéolaires et exudation sanguine.[7, 8]

2.—Les facteurs mécaniques.—Ces facteurs peuvent aussi affecter le résultat de la perfusion pulmonaire. Ainsi, une pression trop forte ou un débit trop volumineux pourra amener une diffusion du liquide de la perfusion dans les tissus et causer de l'œdème pulmonaire. Par contre, un ralentissement de la circulation pourra favoriser une agglutination des globules rouges et une obstruction de la circulation dans les cas extrêmes.

3.—Les facteurs pathologiques.—Il est facile de comprendre que des altérations pathologiques, comme la fibrose pulmonaire, l'emphysème, l'artériosclérose ou autres lésions, rendraient la perfusion pulmonaire plus difficile.

4.—La nature du liquide utilisé pour la perfusion.—Différentes solutions peuvent donner des résultats variables. Ainsi, les caractères physico-chimiques tels que pH, densité, osmolarité, etc. propres à chaque liquide employé: sérum glucosé, dextran, plasma, sang ou mélange de ces solutions, peuvent amener des perturbations entre le milieu intra-cellulaire et le milieu extra-cellulaire, perturbations qui, si elles sont trop fortes, provoqueront des complications pulmonaires.

Dans le présent travail, nos études ont porté sur une perfusion uniquement lobaire et non pas sur un poumon entier. Nous avons étudié les résultats obtenus avec l'emploi de différents liquides pour cette perfusion lobaire, ainsi que les modifications histopathologiques que ces différents liquides produisent dans le poumon.

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Méthode

Nous avons déjà décrit la méthode que nous employons pour la perfusion des lobes pulmonaires.[9] Nous l'avons quelque peu modifiée pour les besoins de cette expérience (Fig. 1).

Un cathéter à ballon du type Dotter-Lukas est introduit dans la veine fémorale gauche. Durant cette intervention, le chien (hépariné à 2 mg. par kg. de poids) est couché sur le côté gauche, les deux pattes écartées. Le cathéter est ensuite monté successivement dans la veine cave inférieure, l'oreillette droite, le ventricule droit, l'artère pulmonaire souche et ensuite dans l'artère du lobe pulmonaire que l'on veut canuler. Le ballonnet de ce cathéter est ensuite gonflé pour interrompre complètement la circulation qui va à ce lobe.

Dans un deuxième temps, nous insérons dans la veine fémorale droite, un cathéter de polythène de calibre 330 enfilé sur une aiguille transeptale de Ross. Le deuxième cathéter est également introduit dans la veine cave inférieure et l'oreillette droite. Une fois rendue dans l'oreillette droite, le septum inter-auriculaire est transpercé avec l'aiguille Ross et l'on fait glisser sur cette aiguille le cathéter de polythène dans l'oreillette gauche et de là dans la veine pulmonaire du lobe que nous allons perfuser. Par voie thoracique, une ligature temporaire autour de cette veine empêche le sang de revenir dans le cœur. Ce cathéter est relié à l'extérieur à une bouteille contenant le liquide de perfusion pour le retour du sang veineux qui se fera par gravité. Quant à la perfusion elle-même, elle sera faite au moyen d'une pompe sigmamotor (TM-II)* qui aspirera le contenu de cette bouteille et le poussera dans le cathéter artériel.

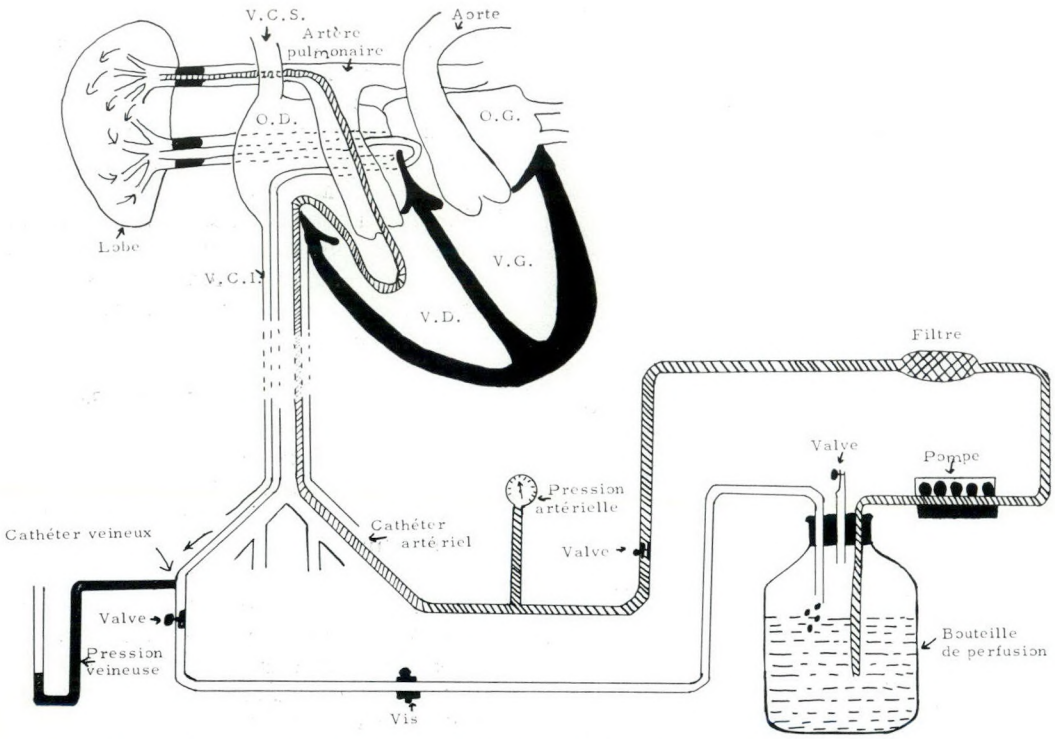

Fig. 1.—Schéma représentant la méthode de perfusion telle que décrite dans le texte.

Résultats

Nous avons ainsi perfusé sélectivement un lobe pulmonaire chez 55 chiens pesant de 12 à 31 kg. Nous avons utilisé quatre variétés de solutions pour ces perfusions et nos chiens ont été classifiés en sept

*Sigmamotor Inc., Middleport, New York, N.Y.

groupes suivant le produit employé:

Groupe I: Chiens perfusés avec du sérum physiologique hépariné à 25 mg./500 ml.

Groupe II: Chiens perfusés avec du Dextran hépariné à 25 mg./500 ml.

Groupe III: Chiens perfusé avec du sang hépariné à 25 mg./500 ml.

Group IV: Chiens perfusés avec un mélange de sang et de Dextran à petites molécules (Rhéomacrodex) hépariné au même pourcentage que les autres.

Groupe V: Chiens perfusés avec un mélange hépariné de sang, de Rhéomacrodex et de sérum glucosé à 5%.

Groupe VI: Chiens perfusés avec du plasma hépariné.

Groupe VII: Chiens perfusés avec un mélange de plasma et de sang, avec interruption de la circulation bronchique.

Un certain nombre de chiens ont été soustraits de notre expérience parce qu'ils sont morts à la suite de complications attribuables uniquement à la technique de la perfusion. Ces chiens ainsi décédés étaient au nombre de sept: trois sont morts à la suite d'arrêt cardiaque; un à cause de perforation de l'aorte et hémorragie incontrôlable. Chez un chien, la mort résulta de l'anesthésie et chez un autre, la veine n'a pu être canulée. Voici maintenant les résultats obtenus pour les différents groupes.

Groupe I.—Trois chiens perfusés avec du sérum physiologique hépariné. Ce produit a amené rapidement un œdème pulmonaire marqué et nous avons noté que cet œdème exsudait par le tube endotrachéal. Ces trois chiens moururent dans les 24 heures qui suivirent la perfusion (Tableau I). L'autopsie montra un œdème plus ou moins sévère du parenchyme avec des zones d'atélectasie diffuse, généralisée aux deux poumons et plus particulièrement du côté du poumon où nous avions perfusé le lobe (Fig. 2). L'hémoglobine plasmatique dosée immédiatement après la perfusion n'a pas varié.

Groupe II.—Ce groupe comprend six chiens perfusés avec du Dextran hépariné. Le Tableau I montre les débits et les temps de perfusion. Des six chiens, deux seulement ont survécu, ce qui donne une mortalité de 66%.

L'autopsie des chiens que nous avons perdus dans ce groupe a montré également de l'œdème pulmonaire bilatéral et de l'atélectasie diffuse et en plus, à plusieurs endroits, des hémorragies intra-alvéolaires (Fig. 3).

Groupe III.—Il s'agit de 16 chiens perfusés avec du sang hépariné. De ces chiens, 13 sont morts, ce qui donne une mortalité de 81.3% (Tableau II). Chez ce groupe perfusé uniquement avec du sang hépariné, la morbidité et la mortalité étaient également extrêmement élevées. A l'autopsie, nous avons retrouvé les poumons œdématiés à des degrés variables, légers dans certains cas et très marqués dans d'autres, avec des zones multiples d'até-

TABLEAU I.—Groupe I.—Perfusion avec sérum physiologique

Chien No.	*Poids Kg.*	*Liquide de perfusion*	*Débit ml./min.*	*Temps de perfusion*	*Lobe perfusé*	*Résultat*
1	16.3	Sérum physiol. 500 ml.	11	1 hr.	L.I.D.	Décédé 5 hrs. plus tard
2	16.3	Sérum physiol. 500 ml.	11	1 hr.	L.S.D.	Décédé 3 hrs. plus tard
3	16.3	Sérum physiol. 500 ml.	7	2 hrs.	L.S.D.	Décédé 24 hrs. plus tard
		Groupe II.—Perfusion avec dextran hépariné				
4	21.8	Dextran 500 ml.	8	3 hrs.	L.M.D.	Survie—sacrifié après 23 jrs.
5	16.3	Dextran 500 ml.	11	1 hr.	L.M.D.	Décédé dans la nuit
6	14.7	Dextran 500 ml.	4	1 hr.	L.M.D.	Survie—sacrifié après 6 mois
7	16.3	Dextran 500 ml.	10	1½ hr.	L.M.D.	Décédé le lendemain
8	13.0	Dextran 500 ml.	10	1¼ hr.	L.M.D.	Décédé 4 hrs. plus tard
9	19	Dextran 500 ml.	10	2½ hrs.	L.M.D.	Décédé durant la nuit

L.S.D.: = Lobe supérieur droit. L.M.D.: = Lobe moyen droit. L.I.D.: = Lobe inférieur droit.

TABLEAU II.—Groupe III.—Perfusion avec sang hépariné

Chien No.	*Poids Kg.*	*Liquide de perfusion*	*Débit ml./min.*	*Temps de perfusion en heures*	*Lobe perfusé*	*Résultat*
10	13.6	Sang 500 ml.	9	2½	L.S.D.	Décédé durant la nuit
11	17.2	Sang 500 ml.	7	3	L.M.D.	Décédé 2 hrs. plus tard
12	17.2	Sang 500 ml.	7	1	L.S.D.	Survie—sacrifié après 6 mois
13	20.4	Sang 300 ml.	7	1	L.S.D.	Décédé durant la nuit
14	14.7	Sang 500 ml.	8	1½	L.S.D.	Décédé 4 hrs. plus tard
15	15.4	Sang 500 ml.	8	1	L.S.D.	Décédé 4 hrs. plus tard
16	12.0	Sang 500 ml.	4	1	L.S.D.	Décédé 3 hrs. plus tard
17	17.7	Sang 500 ml.	7	1	L.M.D.	Décédé 4 hrs. plus tard
18	24.9	Sang 500 ml.	7	1½	L.M.D.	Décédé 12 hrs. plus tard
19	22.0	Sang 500 ml.	6	½	L.M.D.	Survie—sacrifié après 3 mois
20	20.4	Sang 500 ml.	7	1	L.S.D.	Décédé durant la nuit
21	12.9	Sang 500 ml.	7	1	L.S.D.	Décédé 4 hrs. plus tard
22	20.9	Sang 500 ml.	10	1	L.M.D.	Décédé durant la nuit
23	14.1	Sang 500 ml.	10	1½	L.M.D.	Survie—sacrifié après 8 jours
24	18.0	Sang 500 ml.	4	1½	L.M.D.	Décédé après 2 hrs.
25	19.1	Sang 500 ml.	4	1½	L.M.D.	Décédé durant la nuit

L.S.D.: = Lobe supérieur droit. L.M.D.: = Lobe moyen droit.

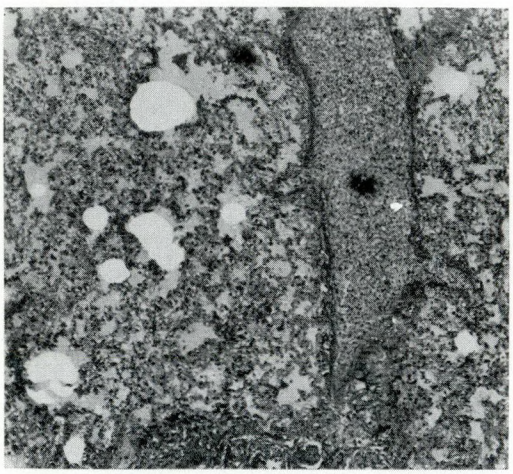

Fig. 2.—Microphoto d'un lobe perfusé avec sérum physiologique pendant deux (2) heures montrant une stase sanguine et un œdème (hémolum-phloxin-safran x 100).

Fig. 3.—Microphoto d'un lobe perfusé avec du dextran durant trois (3) heures montrant de la stase sanguine (hémolum-phloxin-safran x 100).

lectasie des engorgements vasculaires marqués, avec présence de caillots dans les vaisseaux. Microscopiquement, ces constatations étaient encore plus visibles et mettaient en évidence des phénomènes hémorragiques périvasculaires et intra-alvéolaires et des zones d'infarcissement.

Les lésions ainsi produites étaient bilatérales et diffuses; parfois de façon paradoxale, elles étaient plus marquées du côté opposé à la perfusion (Fig. 4).

Le dosage de l'hémoglobine plasmatique a été très élevé (au-dessus de 500 mg. %) chez 18 de ces chiens, de 200 mg. chez le 19e et normal chez le 20e.

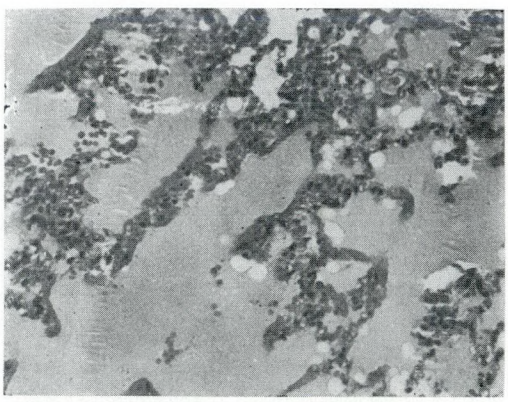

Fig. 4.—Microphoto de poumon perfusé avec du sang pendant une (1) heure montrant une stase sanguine et de l'œdème très important dans les alvéoles (hémolum-phloxin-safran x 250).

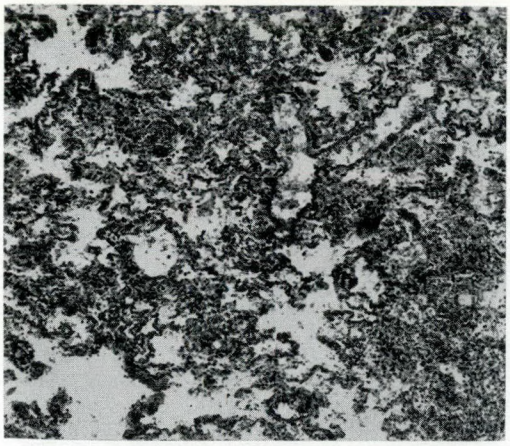

Fig. 5.—Microphoto d'un lobe perfusé avec un mélange de sang et de dextran à petites molécules. On note la présence de stase sanguine et d'hémorragie intra-alvéolaire (hémolum-phloxin-safran x 100).

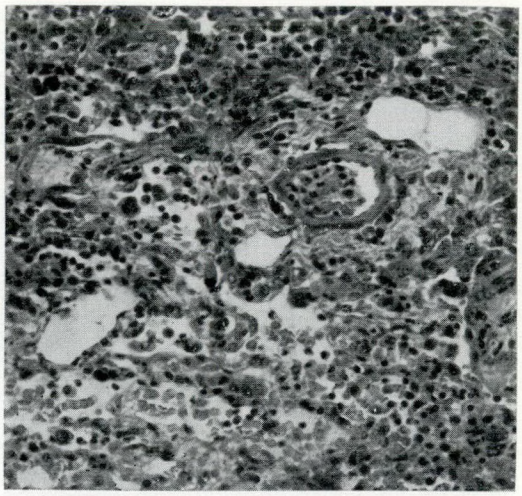

Fig. 6.—Perfusion avec un mélange de sang et de dextran à petites molécules pendant trois (3) heures. Microphoto d'un lobe perfusé, prélevé chez l'animal sacrifié, 15 jours plus tard. Processus pneumonique en voie d'organisation (hémolum-phloxin-safran x 250).

Groupe IV.—Perfusion avec mélange de sang hépariné et de Dextran à petites molécules (Rhéomacrodex) (Tableau III). Nous avons ainsi perfusé huit chiens. Cinq sont morts, ce qui donne une mortalité de 62.5%. Dans cette expérience, il est à noter que le temps de perfusion a été augmenté jusqu'à trois heures et plus. L'autopsie des cinq chiens décédés a montré à peu près les mêmes modifications que chez les chiens du groupe III, sauf que les lésions étaient définitivement moindres (Fig. 5). Un des chiens qui survécut, montrait 15 jours plus tard lors de l'autopsie, un processus pneumonique en voie d'organisation (Fig. 6).

Group V.—Perfusion avec un mélange de sang, de Rhéomacrodex et de soluté glucosé à 5% (Tableau IV). Cinq chiens furent soumis à cette expérience; dans ce groupe, nous avons eu deux morts (une mortalité de 40%). Les poumons des chiens morts montrèrent des changements semblables au groupe précédent.

TABLEAU III.—Groupe IV.—Perfusion avec sang hépariné et rhéomacrodex

Chien No.	*Poids Kg.*	*Liquide de perfusion*	*Débit ml./min.*	*Temps de perfusion en heures*	*Lobe perfusé*	*Résultat*
26	23	Sang 350 ml. Rhéom. 150 ml.	10	3	L.M.D.	Décédé 3 hrs. plus tard
27	21	Sang 350 ml. Rhéom. 150 ml.	10	3	L.M.D.	Décédé durant la nuit
28	18.2	Sang 300 ml. Rhéom. 200 ml.	10	3	L.M.D.	Survie—sacrifié après 19 jours
29	27	Sang 300 ml. Rhéom. 200 ml.	10	3½	L.M.D.	Survie—sacrifié après 4 mois
30	16	Sang 350 ml. Rhéom. 150 ml.	10	3	L.M.D.	Survie—sacrifié après 20 jours
31	25	Sang 350 ml. Rhéom. 150 ml.	10	3	L.M.D.	Décédé durant la nuit
32	31	Sang 300 ml. Rhéom. 200 ml.	10	3½	L.M.D.	Décédé à la fin de l'opération
33	21	Sang 300 ml. Rhéom. 200 ml.	10	3	L.M.D.	Décédé 1 hr. plus tard

Rhéom.: = Rhéomacrodex. L.M.D.: = Lobe moyen droit.

TABLEAU IV.—Groupe V.—Perfusion avec un mélange de sang hépariné—glucose 5% et rhéomacrodex

Chien No.	*Poids Kg.*	*Liquide de perfusion*	*Débit ml./min.*	*Temps de perfusion en heures*	*Lobe perfusé*	*Résultat*
34	23.4	Sang 250 ml. Glucosé 125 ml. Rhéom. 125 ml.	10	3	L.M.D.	Survie—sacrifié après 18 jours
35	19	Sang 250 ml. Glucosé 125 ml. Rhéom. 125 ml.	10	3	L.M.D.	Survie—sacrifié après 13 jours
36	22.7	Sang 250 ml. Glucosé 125 ml. Rhéom. 125 ml.	10	3	L.M.D.	Survie sacrifié après 18 jours
37	20.8	Sang 250 ml. Glucosé 125 ml. Rhéom. 125 ml.	8	3¼	L.M.D.	Meurt sur la table
38	19.2	Sang 300 ml. Glucosé 50 ml. Rhéom. 150 ml.	10	3¾	L.M.D.	Meurt durant la nuit

Groupe VI.—Chiens perfusés avec du plasma hépariné (Tableau V). Dans le groupe VI, contrairement à ce que nous espérions, les résultats nous ont déçus, car notre mortalité fut de 80%. Cependant, nous ne pouvons porter de conclusion définitive à ce sujet et nous nous proposons d'en faire un plus grand nombre de cas. L'autopsie des chiens de ce groupe à montré des lésions semblables au groupe précédent.

Groupe VII.—Chiens perfusés avec un mélange de plasma et de sang, avec une circulation bronchique interrompue dans le but de diminuer la congestion circulatoire et d'améliorer les résultats (Tableau VI). Effectivement, dans ce groupe, la mortalité a été de 60%. Les poumons des chiens morts n'ont pas tellement différé en apparence de ceux des trois derniers groupes.

TABLEAU V.—Groupe VI.—Perfusions avec plasma hépariné

Chien No.	*Poids Kg.*	*Liquide de perfusion*	*Débit ml./min.*	*Temps de perfusion en heures*	*Lobe perfusé*	*Résultat*
39	19.16	Plasma 400 ml.	10	2½	L.M.D.	Meurt dans la nuit
40	17.69	Plasma 400 ml.	10	4¼	L.M.D.	Meurt sur la table
41	20.63	Plasma 400 ml.	10	4¾	L.M.D.	Meurt pendant la nuit
42	17.69	Plasma 400 ml.	24	4¼	L.M.D.	Survie
43	18.14	Plasma 400 ml.	10	4	L.M.D.	Meurt durant la nuit

TABLEAU VI.—Groupe VII.—Perfusion avec mélange de plasma et de sang—circulation bronchique interrompue

Chien No.	*Poids Kg.*	*Liquide de perfusion*	*Débit ml./min.*	*Temps de perfusion en heures*	*Lobe perfusé*	*Résultat*
44	18.6	Plasma 300 ml. Sang 50 ml.	10	1¾	L.M.D.	Meurt sur la table
45	17.69	Plasma 300 ml. Sang 50 ml.	10	2½	L.M.D.	Meurt sur la table
46	19.95	Plasma 250 ml. Sang 50 ml.	10	4	L.M.D.	Survie
47	15.87	Plasma 350 ml. Sang 50 ml.	10	3¾	L.M.D.	Survie
48	17.23	Plasma 350 ml. Sang 50 ml.	10	¾	L.M.D.	Meurt sur la table

TABLEAU VII.—RÉSULTATS DES PERFUSIONS DES LOBES PULMONAIRES AVEC DIFFÉRENTS LIQUIDES

Groupe	*Nombre de chiens*	*Liquide de perfusion*	*Moyenne de temps des perfusions*	*Moyenne des débits*	*Mortalité*
Groupe I.....	3	Sérum physiologique	80 min.	9 ml./min.	100 %
Groupe II.....	6	Dextran	100 min.	9 ml./min.	66.6%
Groupe III.....	16	Sang	80 min.	7 ml./min.	81.3%
Groupe IV.....	8	Sang, Rhéom.	187 min.	10 ml./min.	62.5%
Groupe V.....	5	Sang, Rhéom., Dextrose	192 min.	9.6 ml./min.	40 %
Groupe VI.....	5	Plasma	238 min.	13 ml./min.	80 %
Groupe VII*.....	5	Plasma, Sang	156 min.	10 ml./min.	60 %

*Groupe VII.: = Circulation bronchique interrompue.

CONCLUSION ET RÉSUMÉ

Comme on a pu le constater, la perfusion pulmonaire prolongée semble créer de nombreux problèmes. Mais en analysant ces problèmes, il semble que certains moyens peuvent diminuer jusqu'à un certain point les complications pulmonaires:

1.—Manipuler les poumons avec grande délicatesse et le moins possible.

2.—Eviter toute obstruction au retour veineux qui amène rapidement la congestion du lobe ou du poumon en tenant le récipient contenant le liquide de perfusion à un niveau de 50 cm. au-dessous du cœur du chien.

3.—S'abstenir de pressions trop fortes, car une trop forte pression hydrostatique dans les vaisseaux pulmonaires amène un œdème de la région perfusée.

4.—Les expériences faites au cours de nos recherches semblent indiquer que les meilleurs résultats soient obtenus avec des liquides de perfusion contenant du sang dilué avec du dextran à petites molécules. L'emploi du sang total dans les perfusions amène une agglutination des globules rouges[10] et une stagnation de la circulation peuvent aller à l'obstruction complète des vaisseaux.[11] Il s'ensuit des phénomènes pulmonaires d'atélectasie,[12] de congestion et d'hémorragie intra-alvéolaires.[13]

5.—Dans une publication ultérieure, nous donnerons les résultats de nos expériences en cours, qui portent sur l'interruption de la circulation bronchique, en même temps que la perfusion lobaire. Ces expériences semblent démontrer jusqu'à présent que cette interruption de la circulation bronchique contribue à l'amélioration des résultats.

D'après les résultats des expériences (Tableau VII), nous remarquons une amélioration dans les taux de mortalité quand on passe du groupe I (sérum physiologique) au groupe III (sang), au groupe IV (sang-rhéomacrodex), au groupe V (sang-rhéomacrodex-dextrose). Cette mortalité a diminué, malgré une augmentation constante du temps de perfusion. Il semble que cette amélioration soit due à une diminution de l'agglutination des globules rouges dans la circulation. Cette agglutination aurait tendance à causer des thromboses dans les petits vaisseaux ainsi qu'une stase sanguine qui favoriserait la congestion du lobe. Le soluté glucosé dans le groupe V semblerait agir en diminuant la viscosité du sang.[10]

Par opposition, la perfusion avec du liquide électrolytique en solution aqueuse, produit elle aussi une forte mortalité. La faible teneur en pression osmotique dans ces solutions amène leur diffusion rapide et massive au niveau des tissus pulmonaires.

Dans le groupe VI, la perfusion avec le plasma, quoiqu'éliminant le problème de l'agglutination érythrocytaire, ne semble pas avoir donné de résultats spectaculaires. Il semble qu'un autre facteur très important agisse et produise des atélectasies hémorragiques. Nous pensons que ce facteur pourrait être le composé antiatélectatique lipoprotéinique ou "surfactant".[10]

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Summary

The major known causes responsible for the appearance of pulmonary complications following prolonged lobar perfusions, have been classified under the following headings:

Physiologic factors: The presence of a double pulmonary blood supply, the state of the pulmonary vascular resistance and especially, the presence of phospholipid, lipoproteinic substances with antiatelectatic properties, known as surfactant.

Mechanical factors: These involve the flow rate, the perfusing pressure and the state of venous return.

Pathological factors: Various diseases, which tend to cause important changes in the pulmonary vasculature.

The type of perfusing solution used.

In the present study, the isolated pulmonary lobes of dogs were perfused with different solutions as follows:

Group I.—Dogs perfused with heparinized saline. The mortality was 100%.

Group 2.—Dogs perfused with high molecular weight dextran. The mortality was 66%.

Group 3.—Dogs perfused with heparinized blood. The mortality was 81.3%.

Group 4.—Dogs perfused with heparinized blood and low molecular weight dextran. The mortality was 62.5%.

Group 5.—Dogs perfused with heparinized blood, low molecular weight dextran and glucose 5% solution. The mortality was 40%.

Group 6.—Dogs perfused with heparinized plasma. The mortality was 80%.

Group 7.—Dogs perfused with a mixture of heparinized blood and plasma and in which the bronchial circulation has been interrupted. The mortality was 60%.

From these experiments, it appears that the groups in which blood has been diluted with low molecular weight dextran (Rheomacrodex) have a lower mortality probably due to the antisludging properties of Rheomacrodex and the increased intravascular osmolarity produced by this substance.

The lungs of the dogs that died revealed various degrees of congestion in most of the lobes, as well as areas of necrosis, intravascular stasis, intra-alveolar hemorrhages and diffuse patches of atelectasis. These changes seemed generally to be less severe in the groups perfused with Rheomacrodex.

FOUNDATIONS OF ANESTHESIOLOGY. Albert Faulconer, Jr. and Thomas E. Keys. Vols. 1 and 2. 1337 pp. plus indices. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $46.25 set.

This is a two-volume history of anesthesiology by eminently qualified authors.

Its main distinction is that it documents the development of this specialty by reproducing, and translating where necessary, the original articles which constitute the milestones of its history.

While open to the criticism that such an effort can never be completed in two volumes, to my mind it does two things in superb fashion. First, it makes the "Classics" of anesthesiology easily available for the first time to teachers and authors. Second, it provides the fascination of living history for the student and practitioner.

The material is organized into eight sections and chronologically within each section. Each section begins with an introduction which is, in fact, a summary of the contents of the section. Each original paper is preceded by a very complete biography of the author.

This is a very readable work which should be in every medical library. It is, I am certain, destined to become a standard reference in the history of medicine. In addition, every anesthesiologist, with more than passing interest in the history of his specialty, will want a set for his own.

EFFECT OF A MONOAMINE OXIDASE INHIBITOR ON BLOOD AMMONIA AND GASTRIC SECRETION IN DOGS: II. DOGS WITH SIDE-TO-SIDE AND END-TO-SIDE PORTACAVAL ANASTOMOSIS*

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WHEN portal blood is diverted into the systemic circulation before passing through the liver, some products of intestinal digestion escape hepatic degradation and may activate the intestinal phase of gastric secretion. This process is a possible explanation of gastric hypersecretion observed under clinical and experimental conditions, associated with the inability of the liver to detoxify certain metabolic products which may act as gastric secretagogues. The identity of these secretagogues has not been established. Ammonia has been suspected as the cause of gastric hypersecretion in the presence of hepatic insufficiency. The problem of ammonia intoxication was examined in the first part of this study and the pertinent literature on the subject was reviewed.[1] Since ammonia intoxication can be prevented by a monoamine oxidase inhibitor,[2] we wondered whether this inhibitor would also influence gastric hypersecretion which is associated, under certain conditions, with high levels of blood ammonia.

Previously, we had produced experimental hyperammonemia associated with increased gastric secretion in normal dogs with Heidenhain pouches. We also found that a monoamine oxidase inhibitor did reduce blood ammonia in these animals without affecting gastric secretion.[1] The previous study, however, dealt with normal dogs in which the inactivation of a gastric secretagogue by the liver was not experimentally altered. In the present study dogs with portacaval bypass were used and again the effect of a monoamine oxidase inhibitor on both blood ammonia and gastric secretion was investigated.

METHODS

Surgery.—Mongrel dogs weighing 25 to 36 kg. were used. Heidenhain pouches were prepared and the animals were allowed to recover for three to five weeks before biochemical studies were begun. Base-line determinations of blood ammonia, blood urea nitrogen and pouch juice were performed daily for at least four days, after a 12-hour fasting period. In addition blood ammonia was also determined in some dogs six hours after feeding meat in the amount of 10 g./kg. body weight.

Portacaval shunts were established in the Heidenhain pouch dogs from three to six months after their initial surgery. A side-to-side portacaval anastomosis using 4-0 arterial silk was performed on all dogs. The lumen of the completed shunt averaged 15 mm. in diameter. Pressure studies before and after shunting revealed that the values were considerably higher in the portal vein than in the inferior vena cava. This finding assured an adequate diversion of portal blood into the systemic circulation. Surgery was well tolerated and the blood loss was insignificant. The shunted animals were placed on special low-protein and low-potassium diets for the first two weeks following the surgery. The majority gained weight. Subsequently regular standard diets were offered. Again all animals tolerated these feedings without any evidence of food intoxication.

At the conclusion of the biochemical studies on the side-to-side shunted dogs, three of the animals were subjected to further operation three months later. This last procedure was the conversion of the side-to-side portacaval anastomosis to an end-to-side shunt. The latter was accomplished by ligating the portal vein on the liver side of the previously established side-to-side anastomosis. No portal vein branches were excluded from entering the inferior vena cava by this procedure. The animals tolerated the surgery well. They were able to maintain their weight on low-protein and low-potassium diets but showed slight deterioration when they were transferred to regular standard diets.

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Sampling.—In the experiments described below, dogs were fasted for 12 hours before treatment and collections of samples. Experiments were spaced at least one week apart. In every case venous blood was collected at zero, two, four and six hours after the beginning of infusion or after the termination of tube feeding of whole human blood. Simultaneously, the pouch was emptied for gastric analysis.

Treatment.—The following methods were used: (1) Intravenous infusion of 5% glucose, 2 ml./min. for six hours. (2) Intravenous infusion of 2% of ammonium citrate in 5% glucose, 2 ml./min. for six hours. (3) Monoamine oxidase inhibitor, nialamide (Niamid) 5 mg./kg./body weight was given intramuscularly one hour before the beginning of infusion. An identical amount of ammonium citrate, as in Experiment 2, was then infused for six hours. A second dose of nialamide (5 mg./kg./body weight) was injected at the second hour after the beginning of infusion. (4) The dogs received, by gastric tube, whole human blood, 40 ml./kg. body weight, in divided doses over a 60-minute period. (5) Nialamide, 5 mg./kg. body weight was given intramuscularly, at the beginning of intragastric feeding of blood, and a second similar dose was injected two hours after the feeding ended. Blood was given as in Experiment 4.

Biochemical study.—Total blood ammonia was determined by microdiffusion technique.[3] Blood urea nitrogen (BUN) was studied.[4] The content of the gastric pouch, emptied at the time of blood sampling, was measured and hydrochloric acid was titrated with NaOH using Töpfer's reagent as the indicator.

Results

It is apparent from Table I that intravenous infusion of ammonia in glucose produced significant hyperammonemia as compared with the effect of the control infusion of glucose alone. Associated with this increase of blood ammonia were marked elevation of both volume and acidity of pouch secretion. The injection of nialamide reduced blood ammonia levels but did not produce a significant change in volume or acidity of gastric secretion. When total human blood was fed to these dogs both blood ammonia and BUN were increased, as compared with control fasting values. Associated with these changes in blood were increase of volume and acid-

TABLE I.—Blood ammonia (μg.%), urea nitrogen (mg.%) and secretion from Heidenhain pouch in dogs with side-to-side portacaval shunt. Sampling at zero hours taken after 12 hours of fasting.

Treatment	*Sample*	*Hours after the beginning of infusion* 0	2	4	6
Infusion of 5% glucose* (control)	Blood ammonia	64.1	80.2	71.0	76.3
	Blood BUN	7.8	8.9	9.9	8.7
	Gastric juice ml.	—	9.0	12.1	7.0
	Gastric HCl mEq./l. (6 dogs)	—	62.0	81.0	85.0
Infusion of 2% ammonia citrate in 5% glucose	Blood ammonia	74.0	257.0***	223.6***	230.9***
	Blood BUN	6.3	8.1	9.4	8.6
	Gastric juice ml.	—	24.0†	23.0†	24.0†
	Gastric HCl mEq./l. (6 dogs)	—	113.0†	116.0†	113.0†
Infusion of ammonia citrate Nialamide**	Blood ammonia	56.1	132.0	110.0	92.0
	Blood BUN	7.0	8.1	8.9	9.0
	Gastric juice ml.	—	23.0†	22.0†	21.0†
	Gastric HCl mEq./l. (5 dogs)	—	112.0†	113.0†	111.0†

*Infusion rate, 2 ml./min. for six hours.
**A monoamine oxidase inhibitor, 5 mg./kg./body weight intramuscularly, two doses, the first one hour before, and the second two hours after beginning the infusion.
***P <1% vs. other groups.
†P <1% vs. control group.

ity of pouch secretion. In this experiment (Table II), nialamide also reduced the level of blood ammonia but did not affect gastric secretion significantly. In dogs with end-to-side portacaval shunt the reaction to blood feeding and to nialamide (Table III) was comparable to that observed in animals with side-to-side portacaval shunt.

In three dogs side-to-side anastomosis has been converted to end-to-side anastomosis. The effect of meat meal and of blood feeding on ammonia and pouch secretion were studied before and after this conversion and the results compared. It may be seen from Table IV that postprandial blood ammonia, particularly after total blood

TABLE II.—Blood ammonia (μg.%), urea nitrogen (mg.%) and secretion from Heidenhain pouch in dogs with side-to-side portacaval shunt after intragastric administration of whole human blood, 40 ml./kg./body weight. Sampling at zero hours taken after 12 hours of fasting.

Treatment	*Sample*	*Hours after the administration of blood*			
		0	*2*	*4*	*6*
Intragastric blood	Blood ammonia	59.9	147.2	196.0	197.1
	Blood BUN	8.8	11.1	20.0	21.0
	Gastric juice ml.	—	49.0	47.0	43.0
	Gastric HCl mEq./l.	—	115.0	119.0	113.0
	(6 dogs)				
Intragastric blood and nialamide*	Blood ammonia	70.1	117.0	98.0**	97.2**
	Blood BUN	6.9	10.2	18.0	20.3
	Gastric juice ml.	—	39.0	50.0	39.0
	Gastric HCl mEq./l.	—	98.0	111.0	107.0
	(5 dogs)				

*A monoamine oxidase inhibitor, 5 mg./kg./body weight intramuscularly, two doses, at the beginning of blood feeding and at two hours after.
**P < 1% vs. non-treated dogs.

TABLE III.—Blood ammonia (μg.%), urea nitrogen (mg.%) and secretion from Heidenhain pouch in dogs with end-to-side portacaval shunt after intragastric administration of whole human blood, 40 ml./kg./body weight. Sampling at zero hours taken after 12 hours of fasting.

Treatment	*Sample*	*Hours after the administration of blood*			
		0	*2*	*4*	*6*
Intragastric blood	Blood ammonia	102.0	169.0	141.2	170.0
	Blood BUN	8.9	9.2	17.0	22.1
	Gastric juice ml.	—	21.0	36.0	18.0
	Gastric HCl mEq./l.	—	128.0	130.0	122.0
	(3 dogs)				
Intragastric blood and nialamide*	Blood ammonia	101.0	139.0	140.0	99.2**
	Blood BUN	7.1	8.8	19.0	23.2
	Gastric juice ml.	—	32.0	28.0	12.0
	Gastric HCl mEq./l.	—	118.0	129.0	122.0
	(3 dogs)				

*A monoamine oxidase inhibitor, 5 mg./kg./body weight intramuscularly, two doses, at the beginning of blood feeding and at two hours after.
**P < 1%, vs. non-treated group.

TABLE IV.—Blood ammonia (μg.%) and secretion from Heidenhain pouch in dogs before and after conversion of side-to-side, to side-to-end portacaval anastomosis. Average values for three dogs.

Sampling	*Side-to-side anastomosis*			*Side-to-end anastomosis*		
	Blood ammonia	*Gastric juice vol. ml.*	*Gastric juice HCl mEq.*	*Blood ammonia*	*Gastric juice vol. ml.*	*Gastric juice HCl mEq.*
After 12 hours fasting	70	199	21.67	98	200	27.40
Six hours after meat meal	118	57	4.73	125	85	10.20
Six hours after intragastric blood	119	41	4.88	170	35	4.44

feeding, is higher in dogs with end-to-side anastomosis than in the same animals with side-to-side shunt. Also there was marked increase of gastric juice volume and acidity after the conversion of shunt, in dogs fed meat meal.

Comment

It has been recognized for a long time that postprandial intolerance of Eck fistula dogs to a protein-rich meal was due to a substance which was elaborated by the intestine and entered the systemic circulation without passing through the liver. Research directed at the identification of the toxic substance producing "meat poisoning" in Eck fistula dogs began with the studies of the Pavlov group in 1893.[5] Association of this toxic state with hyperammonemia indicated that ammonia may be the agent responsible. Association of the Eck fistula syndrome with marked gastric hypersecretion pointed to ammonia as the gastric stimulant of intestinal origin.[6, 7] At the present time the role of ammonia as a possible cause of gastric hypersecretion in portacaval shunt is not, however, explained.

In our experiments, the methods of producing increased blood ammonia were used in dogs with side-to-side and with end-to-side portacaval shunts. These two types of surgery are used clinically and their action on liver function and on ascites is known.[8] In side-to-side bypass a considerable part of splanchnic blood is shunted away from the liver. End-to-side anastomosis eliminates the contribution of portal venous blood to hepatic inflow. Postprandial blood ammonia in dogs fed meat was previously found to be higher after end-to-side shunt than after side-to-side shunt[9] and this may be easily explained by the difference in the amounts of portal blood diverted from the liver by these operations. End-to-side shunt is more injurious systemically and it also may result in liver-cell damage.[10]

In the present experiment, experimental hyperammonemia was produced in dogs with two types of shunt. The signs of exogenous ammonia intoxication previously described[1, 2] have been observed in dogs, particularly after the conversion of side-to-side shunt to end-to-side shunt. Particularly toxic was intravenous infusion of ammonia, and under these conditions, the effect of hyperammonemia on gastric secretion cannot be properly evaluated. There is, however, experimental evidence that, when nialamide was injected, the blood-ammonia level was significantly reduced in all dogs and that the type of shunt made no difference as far as the reaction to nialamide was concerned. We demonstrated that, under the conditions of this experiment, nialamide reduced blood ammonia but did not alter the secretion of Heidenhain pouch. Hyperammonemia was associated with increased secretion of pouch juice but there is no evidence that ammonia was a direct stimulant of this secretion, nor was it proved that nialamide has an effect on gastric secretion. It is therefore apparent that the increased quantity of circulating ammonia, under the conditions of this study, is not likely to be a direct cause of the gastric hypersecretion. In a recent work on gastric hypersecretion in dogs with portacaval transposition, another substance, which is a suspected gastric stimulant of intestinal origin, was found to be elevated in blood.[11] This substance, histamine, was not, however, definitely proved to be the cause of gastric hypersecretion in animals with liver bypass.[11] A hypothesis[11] concerning basic increase in the responsiveness of the mucosa to secretion stimuli, by a still-unknown mechanism appears to be an interesting possibility still awaiting experimental verification.

Summary

A significant rise of blood ammonia was observed in dogs with side-to-side and with end-to-side portacaval shunts, after intravenous infusion of ammonium citrate or after feeding total blood. Associated with hyperammonemia was a marked increase in the output of acid from Heidenhain pouch. Nialamide, a monoamine oxidase inhibitor, reduced blood ammonia in these dogs but did not affect gastric secretion.

There is no evidence, therefore, that the increased blood ammonia is a cause of gastric hypersecrtion under the conditions of this experiment.

This project was supported by a research grant from the Medical Research Council of Canada. Pfizer Co. Ltd., Montreal, Que., kindly supplied nialamide (Niamid) for this experiment. Mrs. M. Foster, Mr. J. Olekszyk and Mr. T. Germaine are thanked for their technical assistance.

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Résumé

Dans les cas cliniques et expérimentaux où le sang portal ne traverse plus le foie mais se déverse directement dans la circulation systémique, on note une hypersécretion gastrique dont le facteur étiologique n'a pas pu être identifié. Certains auteurs ont incriminé l'ammonium comme sécrétagogue. Comme l'inhibiteur d'enzyme monoamine oxidase, nialamide (Niamid) est un antidote actif contre l'hyperammoniémie, les auteurs ont voulu étudier son effet sur la sécrétion gastrique. Après avoir preparé une poche gastrique de type Heidenhain et produit un "shunt" portocave chez les chiens, ils ont provoqué un état d'hyperammoniémie par un apport exogène d'ammonium. L'état d'hyperammoniémie artificiellement produit, a été ensuite traité à l'aide de l'inhibiteur monoamine oxidase. Cet inhibiteur a réduit l'hyperammoniémie sanguine, mais n'a exercé aucune influence sur la sécrétion gastrique. Il apparaît donc que, dans les conditions expérimentales décrites, l'ammonium sanguin ne soit pas la cause de l'hypersécrétion gastrique.

HERNIA. Edited by Lloyd M. Nyhus and Henry N. Harkins. 836 pp. Illust. J. B. Lippincott Company of Canada Ltd., Montreal, 1964. $31.00.

In no form of hernia is there uniformity of approach and no single monograph can completely remedy this fact. However, the book edited by Nyhus and Harkins makes some significant contributions to understanding, if not to unity. The editors have gathered contributions from 105 international authorities who deal with aspects of hernia and its repair in which they have a particular interest. Each essay is followed by "special comments" from other specialists with similar or divergent views and a liberal sprinkling of editorial comment sums up each section. This format gives the reader the opportunity to compare, side by side, within the same text, points of outstanding controversy.

This style, which is most commendable, detracts from the value of the book to the novice studying the subject without previous experience. It is of great interest, however, to the surgeon with some familiarity with the field or to the teacher who wishes to broaden his knowledge.

The major portion of the book deals with hernias of the groin with particularly excellent discussions on the problems relating to infants and children and to the aged. It includes, of course, the rare hernias of the lumbar, sciatic, obturator and pelvic regions and serves as an excellent reference text. A major segment concerns diaphragmatic hernias with consideration especially centred at the esophageal hiatus and the problem of reflux.

The use of prosthetic materials is discussed in some depth and the chapters on medicolegal and industrial aspects is to be commended.

In general, this is a most valuable book. The multiple authorship gives rise to variability in style and repetition of content, but, with the exception of an overabundance of historical data, it is well edited. It would be hoped that in future editions the editors will attempt to unify the illustrations which vary greatly in their effectiveness.

EFFECTS OF LIGATION OF THE CIRCUMFLEX CORONARY ARTERY IN DOGS

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Surgical revascularization of the heart has played an important part in the management of selected patients with coronary artery disease during the past few years.[1-4] While the assessment of the results of these operations has been difficult from the clinical point of view, evaluation of the relative merits of these procedures in the experimental laboratory is based on one or more of certain criteria.[5] These criteria include protection against the effects of acute interruption of the anterior descending coronary artery, demonstration of increased collateral circulation by dyes and radiopaque substances, and measurement of coronary blood flow. Of these methods, "test ligation" of the anterior descending branch of the left coronary artery is the most popular. Previous studies have cast considerable doubt on the validity of the "test ligation" because of extreme variability in the mortality rate occurring in control animals.[6, 7] One of the explanations of this variability are the differences in the area supplied by the major branches of the left coronary. The preponderance of one or other of these two branches apparently affects the severity of the infarction following interruption of the major vessel. A recent study suggested a possible relationship between the mortality rate following ligation of the anterior descending artery and the factors controlling coronary blood supply.[7] That this relationship may be applicable to the circumflex coronary artery seems logical. The purpose of the present study was to determine the relationship of the acute effects of ligation of the circumflex coronary artery to some of the determinants of the coronary circulation, and to assess ligation of the circumflex branch of the left coronary artery as a test for evaluating myocardial revascularization procedures.

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Materials and Methods

An unselected series of mongrel dogs was used. On the day of surgery, the weight in kilograms, sex, age and hemoglobin level were recorded. Age was assessed by the application of the dental formula,[8] and hemoglobin estimated by the technique of Sahli.[9] Animals were anesthetized with thiopental sodium using an average dose of 20 mg./kg. body weight and were maintained in a light anesthetic plane. After introduction of an endotracheal tube, respiration was controlled with an Inco* respirator aerating room air. Electrocardiographic tracings, including Leads I, II, III, aVr, aVl and aVf were recorded. Using clean, but not aseptic technique, the left pleural cavity was entered through an incision in the left fourth intercostal space. After opening the pericardium, the circumflex coronary artery was mobilized for a distance of approximately 1 cm., no branches of this vessel being sacrificed. The main stem circumflex artery was securely ligated with a No. 20 cotton ligature. After the usual surgical toilet, the pericardial edges were loosely approximated with fine cotton sutures. The chest was closed in layers. Antibiotics were not administered and defibrillation procedures were not employed at any time. Electrocardiographic tracings, using the same leads as those recorded before operation, were taken immediately after ligation of the artery and one-half hour later. Surviving animals were sacrificed at the end of 24 hours, the electrocardiogram being repeated before the administration of an overdose of thiopental sodium. The time of death was recorded in animals that died before the end of the 24-hour period. A complete autopsy excluding the central nervous system was carried out in every animal. The heart with short segments of the great vessels was carefully excised and washed in running water.

Gross and radiological visualization of

*Manufactured by Inco Ltd., Ambala, India.

the coronary system was carried out by employing the injection of a lead acetate solution according to a method previously described.[10] Part of the injection solution was coloured red with congo red and another part green with brilliant green. The red material was injected into the anterior descending artery and the green into the right coronary artery. For the injection a No. 16 cannula was used attached to a latex tubing which was connected through a Y-tube to a mercury sphygmomanometer. The flask containing the solution was attached to the third limb of the Y. The cannula was introduced through the left coronary ostium into the anterior descending artery. It was fixed in place by a cotton ligature close to its distal end. The injection medium was introduced into the coronary system at a constant pressure of 150 mm. Hg over a 10-minute interval. On removal of the cannula, the artery was ligated to prevent reflux. The same procedure was repeated with the right coronary artery. The heart was cleansed by gently washing it in tap water. Radiographs of the injected hearts were taken in anteroposterior planes. Subsequently, the hearts were preserved in formalin and the coronary arteries were later dissected and accurate details of the coronary vessels, including the extent of the anastomotic channels and size of the circumflex and anterior descending arteries, were recorded.

Results

A series of 98 dogs was studied. Six animals were excluded from this study following unavoidable accidents during surgery. The overall mortality was 23.9% (22 dogs), 7.6% (seven dogs) dying immediately and the remainder 16.3% (15 dogs) within 24 hours. Details regarding age, sex and hematological status are recorded in Tables I, II and III respectively. The

TABLE I.—Relationship Between Mortality Rate and Age of Dogs

Age (Years)	*Total No. of dogs*	*Expired*	*Mortality rate %*
0 - 1	8	1	12.5
1 - 2	9	3	33.3
2 - 4	26	5	19.2
4 - 6	24	5	20.8
6 - 8	18	5	27.7
Over 8	7	3	42.8

TABLE II.—Mortality Rate in Relationship to Sex

Male:	
Total no. of dogs	67
Expired	19
Mortality rate	28.3%
Female:	
Total no. of dogs	25
Expired	3
Mortality rate	12%

mortality rate of female dogs was progressively greater with increasing age (Table IV). The relationship of the degree of coronary collateral vessels, interpreted radiologically (Figs. 1-3), to the percentage

TABLE III.—Mortality Rate and Hemoglobin Level

Hemoglobin g.%	*Total No. of dogs*	*Expired*	*Mortality rate %*
Below 8	1	1	100.0
8 - 10	9	4	45.5
10 - 12	38	13	30.4
12 - 14	44	4	9.0

death rate is recorded in Table V. The extent of intercoronary anastomoses seen on gross dissection and its bearing on the mortality rate is noted in Table VI. When

TABLE IV.—Mortality of Female Dogs Related to Age

Age (Years)	*No. of dogs*	*Expired*	*Mortality rate %*
0 - 2	2	0	0
2 - 4	5	0	0
4 - 6	8	1	12.58
6 - 8	7	1	12.28
Over 8 years	3	1	33.3

the anterior descending coronary artery was predominant, the mortality was 16.7%; when the circumflex artery was predominant, it was 31.8% (Table VII). The vari-

TABLE V.—Relationship of Degree of Intercoronary Anastomoses as Seen on Radiographs to the Mortality

Anastomoses	*Mortality rate %*
Marked	9.6
Moderate	21.8
Few	45.0

ous electrocardiographic observations are summarized in Tables VIII, IX and X. The preoperative electrocardiogram demonstrated normal sinus rhythm in 67 dogs, sinus tachycardia in 10 and ischemic changes in seven dogs. Ischemic changes on the electrocardiogram were recorded with ST-segment depression, increased PR interval or T-wave changes. All the animals with ischemic changes were over six years of age. Electrocardiograms taken immediately after ligation showed ischemic changes in 64 dogs and ventricular tachycardia in 10. Left bundle branch block occurred in two dogs and eight had sinus tachycardia. The dogs with ventricular tachycardia all died within one-half hour after surgery. Atrial fibrillation was seen in four dogs. Electrocardiograms taken 24 hours after ligation demonstrated persistent ischemic changes in 26 dogs. Sixteen dogs showed some improvement in the ischemic changes seen immediately after ligation. Ventricular fibrillation was noted in six dogs and sinus tachycardia in 11. Conduction defects consisting chiefly of left bundle branch block were observed in 13 dogs.

TABLE VI.—Relationship of Degree of Intercoronary Anastomoses Seen Grossly to Mortality

Definite anastomoses:	
Total number of dogs	42
Mortality rate	6.6%
Suspected anastomoses:	
Total no. of dogs	30
Mortality rate	17.6%
No anastomoses:	
Total no. of dogs	20
Mortality rate	75.8%

TABLE VII.—Mortality Rate According to Coronary Artery Predominance

Ant. descending predominant:	
No. of dogs	48
Expired	8
Mortality rate	16.7%
Circumflex predominant:	
No. of dogs	44
Expired	14
Mortality rate	31.8%

TABLE VIII.—Preoperative Electrocardiographic Findings

Sinus tachycardia	10
Ischemic changes	7
Conduction defect	0
Low voltage	8
Normal	67

TABLE IX.
Postoperative Electrocardiographic Findings

Sinus tachycardia	12
Ventricular tachycardia	10
Auricular fibrillation	4
Ischemic change	64
Conduction defect	2 (LBBB)

TABLE X.—Electrocardiographic Findings 24 Hours Postoperatively

Sinus tachycardia	11
Vent. fibrillation	6
Auricular fibrillation	1
Conduction defect	13
Ischemic change persistent	26
Improved ischemic change	16

Discussion

The overall mortality rate of 23.9% in this study is comparable with that of 20% obtained when the anterior descending ar-

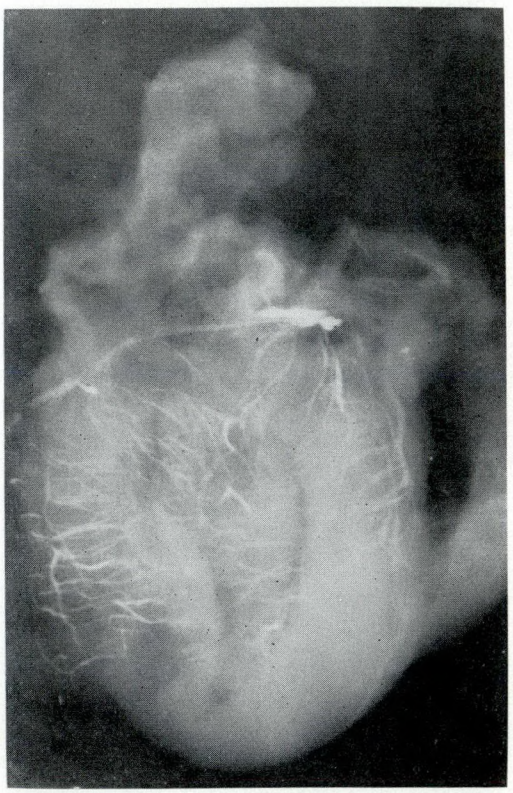

Fig. 1.—A few intercoronary anastomoses are present.

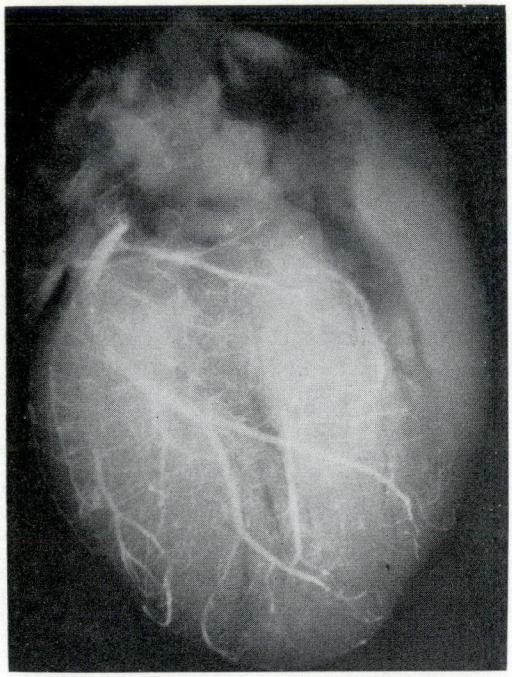

Fig. 2.—A moderate number of anastomosing vessels are noted.

tery was interrupted under identical conditions.[7] These results, however, are not in keeping with those of other investigators.[2, 11-16] The mortality rate of the series reported in the literature has varied between 57 and 100% (Table XI). It should

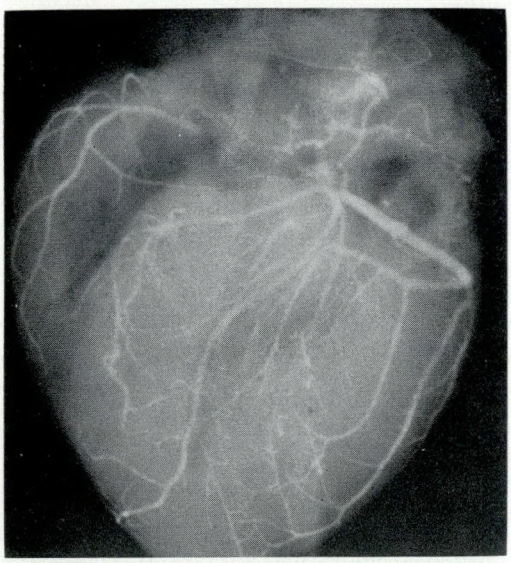

Fig. 3.—A marked number of intercoronary channels are seen.

TABLE XI.—MORTALITY RATE FOLLOWING LIGATION OF THE CIRCUMFLEX CORONARY ARTERY IN DOGS AS REPORTED BY PREVIOUS INVESTIGATORS.

Author	*Mortality rate* %
Erichsen[11]	100
Conheim and Schulthess-Rechberg[12]	100
Porter[13]	64
Smith[14]	57
Beck and Leighninger[1]	68
Lumb, Shacklett and Cook[15]	85
Allen and Laadt[16]	65

be appreciated that the number of animals used in this project was significantly greater than in those previously reported. Of further interest in comparing the mortality rate with that obtained by others is the influence of age, sex, hematological status, degree of intercoronary anastomoses, and coronary artery preponderance on the acute effects documented in this study.

A distinct increase in the immediate death rate was noted in animals above six years of age. This bears a close correlation with the human counterpart of acute coronary occlusion.[17] The major difference in the two situations is that the majority of cases of acute myocardial infarction in the human are due to involvement of the coronary arteries with atherosclerotic disease.[18] No occlusive coronary lesions were demonstrable either radiologically or grossly in the canine hearts that were studied. It is therefore reasonable to assume that the effect of advancing age in decreasing the dog's ability to withstand a major coronary insult may be due to the operation of a myocardial factor in some animals. Since no histopathological studies were carried out on the excised hearts, it is difficult to draw any conclusions on this point. Even if histological studies had been performed, any pre-existing disease may have become less evident as a result of the infarction. The existence of cardiac disease before ligation is suggested by the presence of electrocardiographic abnormalities in a high percentage of older animals initially. In human cases, in addition to previous coronary obliteration, other myocardial lesions are known to play an important role in myocardial infarction.[19]

The premenopausal female who is free from hypertension or diabetes is protected to a remarkable degree from coronary ar-

tery disease.[20-23] Although a completely satisfactory explanation for this phenomenon has never been offered, several interesting hypotheses have been proposed. The intima of the coronary arteries in females is distinctly thinner than that in males even in infancy.[24] Bilaterally oophorectomized women have a greater degree of atherosclerosis than a control group in the same decades of life.[25] In addition specific effects of estrogen on vessels and blood have been documented.[26, 27] Furthermore, because the average female heart is smaller than the male, the demand for coronary blood flow is probably less.[28] Despite all these considerations, the exact nature of the protective mechanism in the premenopausal female is still unknown.[29] The mortality rate among dogs in the present study is almost twice as great in males as in females. Since female dogs do not pass through a menopausal period,[30] any comparable changes present in the human female will not apply. Nevertheless, it is more than coincidental that at about eight years of age, a time that may correspond to the fifth decade in the human, mortality in this procedure among female dogs is markedly increased.

The effects of anemia in preventing myocardial infarction in the human was originally suggested by Porter.[31] While observing the marked infrequency of myocardial infarction in patients with severe hookworm anemia, he hypothesized that the anemic state was responsible for a marked increase in coronary collateral circulation. Eckstein,[32] in experiments conducted on anemic dogs, concluded that in such dogs the mortality after ligation of a coronary artery is less. He postulated that this is due to an augmentation of coronary collateral channels which results from the anemia. In contrast to these observations, the mortality rate in this present study varied inversely with the hemoglobin level. In animals with a normal hemoglobin the mortality was less than in those with lower hemoglobin levels. However, it should be pointed out that the number of anemic dogs in which ligation was performed was quite small. Hence, no definite conclusions regarding the protective effect of anemia in myocardial infarction can be drawn from the present study. The only satisfactory explanation for this discrepancy is that anemic animals are poor surgical and anesthetic risks.[33] It is quite possible that the mortality rate in anemic dogs would be less if the circumflex artery were occluded in the closed-chest unanesthetised animal.[34]

Consideration of the animals in this project in three major groups depending on the degree of intercoronary anastomotic channels clearly demonstrated the protective influence of these anastomoses following a major vascular insult. These results confirm the findings of others who emphasized the importance of intercoronary connections.[35-38] Since the surviving animals were sacrificed within 24 hours of surgery, it is unlikely that the occlusive process had stimulated the development of these communications in a short interval of time.[39] Since the original observations of Lower[40] in 1669, a great deal of interest has centred round the practical importance of intercoronary channels. These anastomoses which occur with greater frequency in the presence of occlusive coronary disease,[41] myocardial hypertrophy,[42] valvular disease,[43] anemia and pulmonary emphysema[44] are observed in only 9% of human hearts.[43] The validity of this belief has been challenged because techniques for demonstrating intercoronary anastomoses have not given consistent results. The recent introduction of *in vivo* coronary arteriography[45] should provide more accurate physiological data concerning these anastomoses. In any event, protective intercoronary anastomosing channels were seen in a significant number of dogs in the present study.

Electrocardiography is of great importance in the diagnosis and management of coronary artery disease.[46] Disturbances of rate, rhythm and conduction are frequent consequences of acute myocardial infarction.[47] These disturbances are due to the production of an irritable focus in the myocardium, interference with the blood supply of the conduction system and the effects of injury to the heart muscle.[48] Conduction disturbances are an ominous sign, although they are much less frequent than changes in rate and rhythm.[49] The main electrocardiographic alterations recorded in this study bear some resemblance to the

changes noted in the human. Sinus tachycardia, seen in the majority of patients with acute coronary occlusion, was the most frequent electrocardiographic change in this study.[47] The most common disturbance of rhythm was the appearance of ventricular premature beats which were noted immediately upon occlusion but did not constitute a grave prognostic sign unless they persisted or occurred intermittently. Other uncommon features were atrial fibrillation, supraventricular tachycardia and nodal rhythm. When ventricular fibrillation appeared it generally terminated in death in a few minutes. Changes in conduction consisting of left bundle branch block or A-V block were also observed, and S-T segment changes were almost universally encountered. These, however, reverted to normal in the large majority of surviving animals. It is interesting to note that although all electrocardiographic leads were not recorded, the information derived from the leads taken in this study would often have been adequate to predict death from the occlusive process.

Although the myocardium of the dog is supplied by the right and left coronary arteries, the relative importance of these two vessels is different. The right coronary artery can be sacrificed in a large majority of dogs with a mortality rate of only 4% under experimental conditions similar to those employed in this study.[50] The left coronary and its two major subdivisions however are critical because they are largely responsible for supplying the area containing the conduction system.[51] The anterior descending artery, through its anterior septal branch, anastomoses with the posterior septal branch of the circumflex artery or right coronary artery. If anastomoses between these two septal vessels are not impaired, one of them may be sacrificed without any serious effects.

The relationship of the ventilating medium to the sequelae of acute experimental coronary occlusion may be of some importance. According to the concept of Beck[52, 53] electrical instability of the heart with resultant ventricular fibrillation is observed when the oxygen differential between normal and infarcted cardiac muscle becomes significant. Such an oxygen differential occurs when pure oxygen is administered to the experimental animal subjected to myocardial infarction. In the present study, all animals were ventilated with room air and the same ventilatory dynamics applied in all experiments, thus minimizing the likelihood of the development of any areas of oxygen differential in the dogs' myocardium and maintaining identical experimental conditions throughout the study.

Gross examination of the coronary vessels of these dogs has emphasized the anatomic variation that exists with respect to the predominance of the anterior descending and circumflex coronary arteries over each other. In an almost equal proportion of dogs, one or other of these vessels is of significantly larger size. The mortality rate following ligation bears a close relationship to these anatomic features. When the anterior descending artery was predominant, ligation of the circumflex artery produced a mortality rate of 16.7%. When the circumflex predominated, the mortality rate rose to 31.8%. The larger vessel is obviously supplying a greater portion of the myocardium and the conduction system. These observations are important from the point of view of the relationship of the anatomic status of the coronary system to the clinical effects of coronary artery disease. The production of different degrees of narrowing of the left circumflex artery in a recent study[15] has indicated that with greater degrees of occlusion of this vessel there is a proportionate increase in the mortality rate. While the results of these investigators are logically acceptable, the high mortality they report following minor degrees of occlusion is difficult to explain since these authors did not specify the age, sex and hemoglobin levels of their animals.

It is reasonable to conclude from the results of the present study that employment of circumflex coronary artery ligation as a model for evaluating myocardial revascularization operations is not acceptable for the following reasons. Utilization of the mortality figures of other authors (Table XI) as a base-line could lead to incorrect interpretations regarding the supposed beneficial effect of the procedure being tested. Attempts to restrict the selec-

tion of dogs for study to those of the same age, sex, hematological status and extent of coronary collateral vessels would not only be difficult, but would leave such investigation open to criticism. Lastly, variation in the degree of preponderance of the anterior descending and circumflex branches is unpredictable and leads to marked differences in survival rates.

Summary and Conclusions

The effects of acute, permanent ligation of the circumflex branch of the left coronary artery have been studied in 92 dogs. The overall mortality was 23.9%. Age, sex, hemoglobin levels, the presence of intercoronary collaterals, and coronary artery preponderance of these dogs is described and the possible relationship of these factors to the mortality rate is considered. In the light of the observations recorded in this study, the use of ligation operations employing the circumflex coronary artery in dogs as an experimental model for the evaluation of myocardial revascularization appears inadvisable.

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RÉSUMÉ

L'étude de la revascularisation chirurgicale du myocarde a fait le sujet de commentaires nombreux sur la conduite à tenir dans la maladie coronarienne. L'évaluation des résultats cliniques est fort difficile cependant; tandis que l'évaluation des résultats expérimentaux a permis de juger de la valeur des techniques de revascularisation en se basant sur certains critères. Comme le "test de la ligature" de la branche antérieure descendante de la coronaire gauche n'avait qu'une valeur discutable pour juger des procédés de revascularisation, les auteurs ont analysés l'effet de la ligature de l'artère circonflexe. Ainsi ont-ils voulu, par ce procédé, estimer le degré de circulation coronarienne collatérale et évaluer les résultats de la chirurgie de revascularisation.

Les auteurs ont opéré 98 chiens chez lesquels ils ont pratiqué des ligatures de l'artère coronaire circonflexe. En discussion, ils analysent les résultats de ces opérations, en comparant le taux de mortalité et les critères suivants: l'âge, le sexe, le taux d'hémoglobine, la circulation collatérale inter-coronarienne et enfin l'anatomie relative des coronaires gauche et droite. Ils ont également étudier les altérations électrocardiographiques per-opératoires et l'examen macroscopique du cœur après autopsie. Les chiens on été groupés en trois catégories selon l'importance des anastomoses inter-coronariennes mises en évidence à l'aide de colorant injecté dans la lumière artérielle. En conclusion, il semble que le "test de la ligature" de l'artère circonflexe n'ait pas de valeur pratique pour juger des opérations qui veulent revasculariser le myocarde.

The Royal College of Physicians and Surgeons of Canada

1966 ANNUAL MEETING

The 1966 Annual Meeting of the College will be held at the Queen Elizabeth Hotel, Montreal, January 20, 21 and 22.

The scientific program for the meeting is beginning to take shape and promises to maintain the high standard of recent years. The Lecturer in Medicine will be Sir Peter Medawar, Director of the Medical Research Council, London, who, in addition, will be admitted to Honorary Fellowship in the College at the Annual Convocation. Sir Peter's topic will be "The Current Position of Research on Transplantation". Dr. Albert Jutras, Director and Professor of Radiology at the University of Montreal, will deliver the Lecture in Surgery and has chosen as the subject of his paper "Physiopathologic Factors in the Detection of Intramucosal Carcinoma of the Stomach". Dr. Allan C. Barnes, Director of the Department of Obstetrics and Gynecology, Johns Hopkins School of Medicine, has chosen "The Postmenopause" as the topic of the Lecture in Obstetrics and Gynecology.

This meeting will also mark the initiation of the Gallie Lectureship in Surgery which will become an annual feature of the scientific program in the Division of Surgery. This Lectureship, supported out of funds subscribed by members of the Gallie Club and contributed to the Educational Endowment Fund, honours the memory of the late Dr. Edward Gallie, one of Canada's outstanding surgeons and teachers. It is fitting that Dr. R. I. Harris, a close personal friend and colleague of the late Dr. Gallie, has accepted an invitation to deliver the first Gallie Lecture.

Closed-circuit colour television will again form an important adjunct to the scientific program through the courtesy of Smith Kline & French. The following subjects will be presented on television: "Disorders of Growth", "Hypophysectomy — Its Place in the Management of Breast Cancer and Diabetic Retinopathy" (Demonstration: Transphenoidal Approach), "Angiography and its Clinical Applications", "The Disabled Hand" and "Learning Difficulties in Children of Normal Intelligence".

To broaden the interest of the morning scientific sessions for as many as possible of those attending the meeting, two symposium-type sessions will be presented on each of the Friday and Saturday morning programs. The titles of these symposia are:

The Secretary,
The Royal College of Physicians and Surgeons of Canada,
74 Stanley Avenue,
Ottawa 2, Ontario.

I desire to register to attend the Scientific Sessions of the Annual Meeting of The Royal College of Physicians and Surgeons of Canada to be held at the Queen Elizabeth Hotel, Montreal, January 20, 21 and 22, 1966.

Enclosed is a cheque/money order in the amount of $15.00 in payment of the Registration Fee.

Name of Certificant .....

Address .....

.....

Name of Specialty .....

(Please print)

Friday Morning: Highway Accidents and the Problem of Multiple Injuries. Molecular Basis of Human Disease.

Saturday Morning: The Interesting World of the Fetus. Team Approach to the Treatment of Emergencies in Hospital.

Through the generous support of Lederle and of Davis & Geck, simultaneous translation facilities will be provided for the Annual Business Meeting, all of the major lectures, all of the television presentations, and at least one of the morning and afternoon scientific sessions on each day of the meeting.

INVITATION TO CERTIFICATED SPECIALISTS OF THE COLLEGE TO ATTEND THE 1966 ANNUAL MEETING

The Council of the College extended a cordial invitation to all Certificated Specialists of the College to attend the Annual Meeting and to participate fully in the scientifiic sessions. Those who desired to attend were asked to complete a registration application form and a copy of the program was sent about mid-December to all those who registered in advance.

INTERNATIONAL FEDERATION OF SURGICAL COLLEGES

The International Federation of Surgical Colleges held its Annual Meeting in Philadelphia, Pa., and Atlantic City, N.J. October 14-17.

The Council and Committee meetings took place in Philadelphia. Professor J. Englebert Dunphy of the University of California was elected Vice-President in succession to Professor I. S. Ravdin, who did not seek re-election: Professor J. F. Nuboer (Holland) was re-elected to the Executive Committee, and a new member of that Committee is Professor W. T. Rudowski of Poland, who takes the place of his compatriot Professor K. Debicki who had resigned.

The Council elected to membership in the Federation the Brazilian College of Surgeons, which has its headquarters in Rio de Janeiro and was founded in 1929.

The Committee on the Training of Surgeons reported good progress with a brochure on Surgical Education and Training, which it hopes to publish within the next year.

The Committee on Interchange of Young Surgeons produced the first number of the *Interchange Bulletin*, containing the names and addresses of the Professors of Surgery and other heads of surgical divisions who participate in the Federation's Interchange Scheme. This document also contains a list of interchanges effected and announcements of vacant interchange posts. It is to be published annually.

The European Group of the Research Committee reported a promising venture by a group of young surgeons from Sweden, France, Germany and the United Kingdom who have drawn up a program of investigation, on an international basis, into malignant disease of the stomach.

Social events in Philadelphia included a visit to the Rosenbach Museum and a tour of the University of Pennsylvania.

The representatives then moved on to Atlantic City to join in the Clinical Congress of the American College of Surgeons, prior to which the Federation held an open discussion on "Surgery and Surgical Teachings of the Undergraduate Period of Medical Education". The Chairman on this occasion was Professor I. S. Ravdin (U.S.A.) and the invited speakers were Professor Sir John Bruce (U.K.), Professor J. Englebert Dunphy (U.S.A.), Professor Warren H. Cole (U.S.A.) and Professor Walter MacKenzie (Canada); the discussion was summed up by the President, Professor Sir Harry Platt (U.K.). The unanimous view was that the place of surgery in the undergraduate curriculum must be fully maintained and that the teaching must be carried out by surgeons themselves.

The next meeting of the International Federation is to be held in Warsaw in September 1966, when there will be an open discussion on "Surgical Ethics, with Special Reference to the Problems Arising from Transplantation". Subsequent meetings are scheduled to be held in Vienna in 1967 and in Israel in 1968. The Executive Committee will meet in Stockholm in May 1966.

CANADIAN JOURNAL OF SURGERY

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

The Journal is published quarterly. Subscription is $10 per year ($5 per year for trainees in surgery), and starts with the January issue of each year. Single copies are $2.50 each, payable in advance. (It would be greatly appreciated if subscribers would please add bank exchange to their cheques.)

INSTRUCTIONS TO CONTRIBUTORS

Manuscripts

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

Reprints

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

References

References should be referred to by numerals in the text. They should include in order: the author's name and initials in capitals; title of the article; abbreviated journal name; volume number, page number and year. The abbreviations of journal names should be those used by the National Library of Medicine, Washington, D.C., as published in *Index Medicus*. References to books should include in order: author's name and initials; title of book; number of edition (e.g., 2nd ed.); title of publishing house; city of publication; year of publication; page number if a specific reference.

Illustrations

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

Language

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

JOURNAL CANADIEN DE CHIRURGIE

Toute communication concernant le Journal devra porter la mention "Journal canadien de chirurgie" et être adressée à l'Editeur, Publications de l'A.M.C., 150 St. George Street, Toronto 5.

Le journal est publié trimestriellement. Le prix de l'abonnement est de $10. par an ($5. par an pour les médecins qui sont résidents en chirurgie) et commence avec le numéro de janvier de chaque année. Un exemplaire isolé coûte $2.50 et est payable d'avance. (Nous serions reconnaissants aux souscripteurs de vouloir bien ajouter à leur chèque le montant des frais bancaires éventuels).

INSTRUCTIONS A NOS COLLABORATEURS

Manuscrits

Les manuscrits d'articles originaux, de rapports cliniques etc. seront envoyés en deux exemplaires, accompagnés d'une lettre demandant qu'on veuille bien considérer leur publication dans le *Journal canadien de chirurgie*. Ils ne seront acceptés qu'à la condition qu'ils n'aient été soumis qu'à notre Journal et qu'ils ne soient pas réimprimés sans le consentement exprès de l'éditeur et l'auteur. L'acceptation ou le refus des articles soumis relève du Conseil de la publication. Si la place est disponible, un nombre limité d'histoires cliniques pourront être publiés. Les articles seront dactylographiés sur un seul côté d'un papier non ligné, à double espace et avec une large marge. L'auteur devra toujours conserver une copie au papier carbone du texte soumis. Tout article devra être accompagné d'un résumé. L'orthographe sera celle adoptée par le dictionnaire Larousse. Quant à la terminologie scientifique, elle sera basée sur le Dictionnaire des termes techniques de médecine ou tout autre ouvrage de référence sérieux. Le Conseil de la publication se réserve le droit d'apporter au texte les changements qu'il jugerait à propos pour assurer la correction grammaticale et l'orthographe, pour éliminer d'éventuelles obscurités ou pour rendre la présentation conforme au style du *Journal canadien de chirurgie*. Aucun changement important ne sera apporté au texte sans que l'auteur aît été préalablement consulté. Les auteurs recevront avant la publication des épreuves d'imprimerie de leur texte, auxquelles ils sont priés d'apporter le minimum de corrections.

Tirés-à-part

On pourra commander des tirés-à-part sur une formule qui est envoyée avec les épreuves. Il est important de les commander avant la publication de l'article, sous peine de devoir payer un supplément pour une nouvelle composition.

Bibliographie

Les références bibliographiques seront indiquées par des numéros dans le corps du texte. Elles comprendront dans l'ordre: le nom de l'auteur et ses initiales, en majuscules, le titre abrégé du Journal, le numéro du volume, le numéro de la page et l'année. Les abréviations admises pour les noms de revues sont celles qui figurent dans *l'Index Medicus* de la Bibliothèque Nationale de Médecine, Washington, D.C. Les renvois aux livres comprendront dans l'ordre: le nom de l'auteur, ses initiales, le titre de l'ouvrage, le numéro de l'édition (p. ex. 2ème éd.), le nom de la maison d'édition, la ville où elle est située et l'année de la publication; enfin, le numéro de la page s'il s'agit d'un renvoi précis.

Illustrations

Le journal accepte de publier gratuitement un nombre raisonnable d'illustrations en noir et blanc. Les reproductions de clichés en couleurs seront publiées aux frais de l'auteur. Les photographies seront imprimées sur papier brillant, ne seront ni montées ni calibrées et d'un format maximum de 8" x 10". En ce qui concerne les radiographies, nous demandons des copies et *non pas l'original*. On devra toujours fournir un agrandissement de microphotographies. Il ne faut jamais écrire ou dactylographier un texte quelconque sur les photographies. Une légende les identifiant pourra être jointe au dos. Dans les illustrations montrant des malades, ceux-ci ne pourront être reconnus, à moins qu'ils n'en aient donné le consentement écrit préalablement à la publication. Les graphiques et diagrammes seront dessinés à l'encre de Chine sur un bon papier à dessin blanc. Le lettrage devra être écrit en caractères assez grands pour que, après réduction proportionnelle au format du Journal, ils soient encore lisibles. Les légendes devant accompagner les illustrations seront dactylographiées sur une feuille indépendante du texte. Les illustrations ne seront ni roulées ni pliées.

Langue véhiculaire

Il doit être clairement établi que les collaborateurs ont pleine liberté de soumettre leurs articles en français ou en anglais, à leur choix. L'acceptation de l'article sera entièrement indépendante de la langue choisie par l'auteur. Si le collaborateur le désire, il peut décrire le contenu de l'article en un sommaire ne dépassant pas 300 mots et dans une langue différente de la langue choisie pour l'article lui-même. Par exemple, un article écrit en français doit comporter un résumé en français et peut, si l'auteur le désire, être accompagné d'un sommaire plus détaillé en anglais.

BOOK REVIEWS

(See also pages 38, 56, 77, 80, 90, 98, 103)

COAGULATION AND TRANSFUSION IN CLINICAL MEDICINE. Shirley A. Johnson and Tibor J. Greenwalt. 203 pp. Illust. Little, Brown and Company, Boston; J. B. Lippincott Company of Canada Ltd., Montreal, 1965. $10.50.

This small book, directed toward the practising clinician, contains brief and up-to-date accounts of all the important disorders of hemostasis, with most emphasis on the bleeding syndromes, their clinical features, laboratory diagnosis and practical management by replacement (transfusion) therapy. The use of anticoagulants and thrombolytic agents is also briefly outlined.

The first chapter reviews the mechanisms of hemostasis; the principles of tests of hemostatic function are critically described, but the reader is referred elsewhere for the technical details of the more complex procedures.

The second chapter outlines the principles of replacement of deficient factors by transfusion, and emphasizes the value of specific blood component therapy, when a precise laboratory assessment has been made.

Subsequent chapters deal with the congenital and acquired hemorrhagic disorders and thrombosis. Of particular interest to the reviewer was the authoritative chapter on disorders of platelet function and their recognition.

These authors have had wide experience in the fields dealt with in their book and what they have to say is generally accurate and useful. The book is, nevertheless, somewhat disappointing. The text is often repetitious, sometimes ambiguous and occasionally inelegant in expression. Pruning of redundancies would have left space to discuss more adequately such vexing problems as the differentiation of primary fibrinolysis from fibrinolysis secondary to the defibrination syndrome. Although the international numerical terminology for coagulation factors is chiefly employed, the terms Ac globulin (for Factor V) and antihemophilic globulin (for Factor VIII) are retained. It is also difficult to see what the four half-tone photographs add to the book, other than to its cost.

These minor criticisms aside, this book is recommended as an introduction to an important and rapidly developing aspect of clinical medicine.

LECTURE NOTES ON GENERAL SURGERY. Harold Ellis and Roy Yorke Calne. 382 pp. Illust. Blackwell Scientific Publications. Oxford; The Ryerson Press, Toronto, 1965. $9.25.

This little volume has been written by the Professor of Surgery and his Senior Assistant at the Westminster Hospital Medical School in London. It is intended for the medical student in his clinical years and its size is such that it can be carried in his pocket. In their preface the authors say "There is a serious need for a book which will set out briefly the important facts in general surgery classified, analyzed and as far as possible rationalized for the revision student. These lecture notes represent our own final-year teaching; they are in no way a substitute for the standard textbooks."

Within these limits the authors succeed admirably in achieving their object. The subject matter includes general surgery, urology and neurosurgery, (but excluding fractures and orthopedics) and the text is concise and clear. Medical students in their clinical years will find it of value for quick reference and revision purposes.

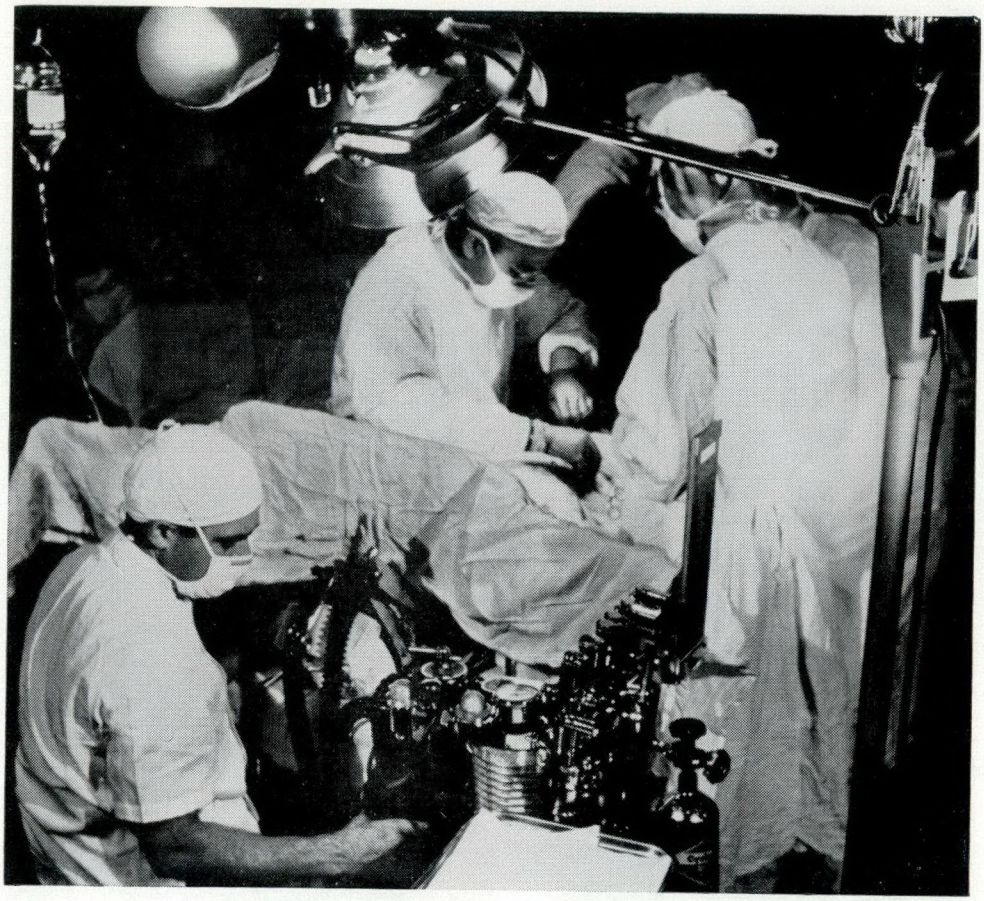
AN OUTLINE AND ATLAS OF GYNAECOLOGICAL CYTODIAGNOSIS. Second revised and enlarged edition. H. Smolka and H.-J. Soost, 208 pp. Illust. Edward Arnold (Publishers) Limited, London; The Macmillan Company of Canada Limited, 1965. $21.60.

This English translation of the 2nd edition of a German textbook is more text than pictorial, since the printed word occupies more than one-half of the 208 pages. The 220 illustrations are mostly small but high-quality photomicrographs, a few of which are in colour.

The text reflects a lack of emphasis on carcinoma which is uncommon on this continent. The section on carcinoma occupies only 26 pages and while the content is sound, it would not ordinarily be considered a sufficient text in this subject for either gynecologists or cytotechnologists. On the other hand, 75 pages are devoted to the description and discussion of hormonal effects. This may constitute useful information for the gynecologist but maximum service to the patient requires not only detailed knowledge but the closest co-operation between clinician and cytopathologist.

The authors have emphasized the clinical correlation and the pitfalls in interpretation and techniques. Dr. Smolka is the Director of the Department of Obstetrics and Gynaecology of the Robert Koch Hospital, Hanover, and Dr. Soost, the Director of the Cytology Laboratory Hospital for Women, Munich, and their book will be useful to both clinicians and cytologists. The terminology used is the commonly accepted international one, and the terms and indices are explained. The reference documentation is extensive, and although more than one-half of the 19-page bibliography is in German, the English and French literatures are also extensively surveyed.

On the whole, this book should provide a readable and informative addition to the library of gynecologists and cytologists.



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CURRENT SURGICAL MANAGEMENT III. Edwin H. Ellison, Stanley R. Frieson and John H. Mulholland. 519 pp. Illust. W. B. Saunders Company, Philadelphia and London; McAinsh & Co. Limited, Toronto, 1965. $12.45.

This book is both valuable and exciting. It is the third in the series dealing with contentious areas, mainly in the field of general surgery. The fact that three volumes should have appeared in such a short time testifies to the continuing interest in the problems considered, and it is certainly to the advantage of surgeons, to continue to demand a critical reappraisal of many fields, when dogma would be an easier diet. To the editors' credit they have chosen for consideration subjects which continue to arouse interest, and the authors of the sections have carried out their appraisal in a balanced and judicial manner, depending on their own factual knowledge for the assessment. In the areas of controversy discussed, no clear-cut advantages accrue to any methods of treatment advocated, or there would be no controversy. These problems can only be solved by a new approach, or by careful reassessment of existing methods. It is important that those in training appreciate that controversies do exist. Surgeons in the fields concerned, who are aware of the problems, want to be kept informed of the present position with respect to them. Both groups will be stimulated by the present volume. Neither will be satisfied, for, if they were, the present volume would have failed in presenting a balanced picture on either side.

The subjects discussed fall into the field of general surgery, and most of them are the everyday problems on which much might be said. In fact, much is not said. The case is put succinctly and in a most readable way. Among the problems dealt with are thrombophlebitis, inguinal hernia, ulcerative colitis, the dumping syndrome, the bleeding ulcer, nasogastric tubes, chronic pancreatitis, acute cholecystitis, and radical mastectomy. Although these are continuing problems, it is still possible to illuminate them by new information or a balanced combination of known facts.

The present volume must be read by surgical residents, and should be read by general surgeons. It should be in every hospital library, and it will probably be bought for most personal libraries.

PRINCIPLES OF X-RAY DIAGNOSIS OF THE SKULL. G. H. Du Boulay. 370 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1965. $33.75.

This text is a welcome addition to the

two volumes by George Simon on the principles of X-ray diagnosis of the chest and bones, respectively. As did these, this book begins with a description of specific radiological findings, e.g. a localized increase in density, and discusses the possible causes — instead of the more usual practice of discussing each disease entity in turn and listing its various manifestations. This method follows more closely the logical sequence of thought required when viewing a radiograph; but although it is a valuable approach, it does make for rather slower reading. Du Boulay's book is an improvement on the two earlier volumes in this series, since it has more internal cross-references, is more profusely illustrated and is printed on a better grade of paper. It is also proportionately more expensive.

Most of the illustrations are well chosen and of high quality, although there are a number of disappointing exceptions. A few typographical errors may cause confusion, e.g. "osteoporosis" for "osteopetrosis" on page 212, and "orbito-mental" plane instead of "orbito-meatal" plane on page 351.

The author has confined his text to the interpretation of routine skull radiographs (plain films), without attempting to deal with the interpretation of angiograms and pneumograms. His wide experience and thoughful interpretation is evident throughout the book. Although it is intended for students of radiology, it should be worthwhile reading for otolaryngologists and particularly residents training in neurology and neurosurgery. It is too detailed for medical students and most residents in general surgery. It will prove valuable as a reference volume, e.g. if radiographs disclose widening of the sphenoidal fissure, one will find a useful list of possible causes and a discussion on the differential diagnosis of this feature. No library on neurosurgery, neurology or radiology will be considered complete without this work.

ACCIDENT SURGERY. Vol. 3. Edited by H. Fred Moseley. 362 pp. Illust. Appleton-Century-Crofts, Inc., New York, 1965. $12.00.

This third volume on Accident Surgery was based upon a series of lectures given during a postgraduate course on Emergency and Accident Trauma at the Royal Victoria Hospital, Montreal. The volume is carefully indexed and contains numerous reference lists. Illustrations are freely used to enhance the text.

The first three chapters deal with ambulance service, organization of Emergency Departments and organization of a clinical service dealing with trauma victims. The last two chapters outline the principles of disability evaluation and the role of the medico-legal expert. Chapters 4 through 13 describe the diagnosis and treatment of a wide variety of traumatic injuries. Chapter 13 is devoted to the problems of limb replantation and includes the presentation of a case.

This book is well written under the very able and experienced editorship of Dr. H. F. Moseley and is very highly recommended to all those who face the responsibility of treating patients who have sustained severe trauma. Volume 3 complete the series on accident surgery and represents an important contribution to this field of surgery.

CLINICAL SURGERY—5. THORAX. Edited by A. L. d'Abreu. 540 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto. 1965. Vols. 1-16 and Index sold only as a set. $314.00.

This is one of a series of volumes on clinical surgery under the general editorship of Charles Rob and Rodney Smith. This particular volume deals with thoracic disease in a broad sense, including the heart. There are 26 contributors from various parts of Great Britain.

The avowed aim of the book is to meet the needs of those whose medical interests are not confined predominantly to diseases of the thorax but who require an acquaintance with the field of thoracic surgery. On the whole this objective has been achieved very well. The various sections are written clearly and concisely and are well illustrated. The book represents a very good exposition of British practice in thoracic surgery at the present time, but the bibliography at the end of each section does refer freely to the North American literature as well as the British.

This volume will be valuable not only to graduate students of surgery but also to practising thoracic surgeons as a reference work.

TUMORS OF BONE AND SOFT TISSUE. A Collection of Papers Presented at the Eighth Annual Clinical Conference on Cancer, 1963 at The University of Texas M.D. Anderson Hospital and Tumor Institute, Houston, Texas. 448 pp. Illust. Year Book Medical Publishers, Inc., Chicago, Ill., 1965. $41.30 U.S.

Some 18 well-known authors contributed the papers for the conference and, apparently fortuitously, compiled the work into book

form. Bone tumours make up the major part of the book, the remainder consisting of papers on various soft-tissue tumours. The book was not intended as a complete study of these tumour groups but as a collection of current concepts of cell physiology, cell pathophysiology, tumour histogenesis, specific tumour pathology, and tumour management. Certain tumours, such as giant cell tumours of bone, rhabdomyosarcoma, were made the sole subject of a paper because of their controversial nature. A generous section of the book is devoted to the results of surgical, chemotherapeutic, and radiological management of individual tumours.

The pathologist, the surgeon and the radiotherapist will gain a certain amount of satisfaction in sharing the frustrations and perplexities of these outstanding workers as they attempt to explain the nature of some of these tumours. The book contains excellent food for thought and is recommendable.

FLUID AND ELECTROLYTES IN NEUROLOGICAL SURGERY. Burton L. Wise. 117 pp. Illust. Charles C Thomas, Springfield, Ill., The Ryerson Press, Toronto, 1965. $7.25.

The first four chapters of this very readable monograph give a concise discussion of fluid and electrolyte disturbances accompanying neurosurgical lesions and operative procedures. A chapter is devoted to both hypotonicity and hypertonicity. The concluding chapters describe the management of patients with pituitary and hypothalamic lesions, the effects of mannitol and urea, and the influence of the nervous system on fluid and electrolyte metabolism. An excellent index and bibliography are appended.

The use of illustrative cases makes the subject come alive for those who may feel somewhat lost in this increasingly complex field. Presentation is brief, pertinent, and clear, and "balance" details are charted on simplified Moore and Ball diagrams. Brief summaries of signs and symptoms, pathology, prevention and treatment make the clinical application complete.

Dr. Wise has contributed much to the basic knowledge of this subject; by preparing this monograph he has also performed a service to all neurosurgeons — for whom it is required reading. Students, researchers and clinicians treating patients with central nervous disorders will also benefit from reading it.

BOOK REVIEWERS, 1965

The physicians, other medical scientists and members of allied health professions, who month by month assist the Editor of the *Canadian Journal of Surgery* by contributing book reviews, make a significant contribution to continuing medical education. The Editorial Board and the Editors wish at this time to acknowledge with gratitude the assistance in this service during the year 1965 of their colleagues whose names appear below.

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Books Received

Books are acknowledged as received, but in some cases reviews will also be published. Other books received to be found on page 65.

Cardiovascular Pathology. Reginald E. B. Hudson. Vols. 1 and 2. 2123 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1965. $75.00 set.

Coagulation and Transfusion in Clinical Medicine. Shirley A. Johnson and Tibor J. Greenwalt. 203 pp. Illust. Little, Brown and Company, Boston; J. B. Lippincott Company of Canada Ltd., Montreal, 1965. $10.50.

Comparative Cardiology. Annals of the New York Academy of Sciences. Vol. 127, Art. 1. Harold E. Whipple, Editor; Paul E. van Reyen, Associate Editor. 875 pp. Illust. New York Academy of Sciences, 2 East Sixty-third Street, N.Y. 21, N.Y.

Current Concepts in Surgery. A Clinical Interpretation of Basic Knowledge. Edited by John H. Davis. 390 pp. Illust. McGraw-Hill Canada, Toronto, 1965. $16.30.

Current Surgical Management III. Edwin H. Ellison, Stanley R. Frieson and John H. Mulholland. 519 pp. Illust. W. B. Saunders Company, Philadelphia and London; McAinsh & Co. Limited, Toronto, 1965. $12.45.

Les Entretiens de Bichat 1965. Chirurgie — Spécialités. 426 pp. plus advertising inserts. Expansion Scientifique Française, Paris, 1965.

Foundations of Anesthesiology. Albert Faulconer, Jr. and Thomas E. Keys. Vols. 1 and 2. 1337 pp. plus indices. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $46.25 set.

Fibrinolysis. George R. Fearnley. 191 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1965. $6.25.

The Keys to Orthopedic Anatomy. William A. Miller. 155 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1965. $7.00.

Lecture Notes on General Surgery. Harold Ellis and Roy Yorke Calne. 382 pp. Illust. Blackwell Scientific Publications, Oxford; The Ryerson Press, Toronto, 1965. $9.25.

Lumbar Disc Lesions. 3rd ed. J. R. Armstrong. 307 pp. Illus. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1965. $11.75.