Aneurysms of the abdominal aorta: a 20-year study[1]

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Summary: One hundred and eighty-eight patients in whom the diagnosis of aneurysm of the abdominal aorta (AAA) was established after 1 January 1960 were followed until their deaths or to 31 December 1979. By the actuarial method, the cumulative 5-year risk of an intact aneurysm progressing to rupture was 35%; the observed 5-year survival rate for patients who had medical management for intact AAA was 33%, for patients who had elective surgery for AAA 74%, for patients who had emergency surgery for ruptured AAA 35%, and for those who did not have surgery for ruptured AAA 0%.

Comparison of the non-operated and electively-operated groups of patients showed that the former was disproportionately weighted with older higher-risk patients, suggesting that the difference in survival rates for the two groups might be a reflection of patient selection rather than of surgical intervention. Comparison of the cumulative 5-year risk of rupture of an intact AAA with the cumulative 5-year mortality rate associated with elective surgery for intact AAA showed that elective surgery for intact AAA might be expected to result in a reduction in the cumulative 5-year mortality rates of patients with intact AAA.

Introduction

Current concepts regarding aneurysms of the abdominal aorta (AAA) have, to a large extent, been shaped by retrospective reviews of affected patients attending major medical centres (Estes 1950, Crane 1955, Schatz *et al.* 1962, Fielding *et al.* 1981). Undoubtedly, the conclusions from these studies have been coloured by their retrospective viewpoint and by the patient catchment. The present study has addressed AAA from a different perspective. All medically-recognized patients with AAA from all sources of medical attention in a community – whether doctors' offices, coroner's jurisdiction, or general hospital – who met the authors' criteria for the diagnosis of AAA were enrolled during the data collection phase of the study extending from 1 January 1960 through 31 December 1979. This paper analyses the information obtained from these patients.

Methods

With the prior approval of the medical staff of the Kelowna General Hospital and the sustained cooperation of its members, the medical records of all patients from the Central Okanagan Valley in British Columbia, Canada, in whom the diagnosis of AAA was suspected were made available to the authors. The medical records of patients with the diagnosis of AAA suspected prior to moving to the Okanagan Valley were obtained by correspondence with their previous physicians.

Patients were accepted for inclusion in the study if they satisfied any one of the following criteria for the diagnosis of AAA: (1) radiological evidence (Janower 1961) of loss of parallelism of the walls of the abdominal aorta – typically, there was crescentic calcification in the left lateral wall; (2) a positive aortogram – a negative aortogram did not exclude a patient

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from this study because thrombus lining the aneurysm may cause the channel to appear deceptively normal (Jackson 1963); (3) a positive echoaortogram (Mulder *et al.* 1973); (4) the finding of an AAA by a surgeon at laparotomy; or (5) the finding of an AAA by a pathologist at necropsy.

Palpation of a midabdominal pulsatile mass was not accepted as a sufficient criterion for inclusion. Only those patients in whom the diagnosis of AAA was first established between 1 January 1960 and 31 December 1979, according to the listed criteria, were included. Data concerning the sizes of the aneurysms were not uniformly collected.

The diagnosis of rupture of AAA was confirmed by laparotomy or by necropsy in many cases. In the remainder, the diagnosis of rupture of AAA was based on prior knowledge of the presence of an intact AAA; and on clinical evidence of intra-abdominal haemorrhage, namely abdominal and/or lumbar pain, arterial hypotension, and falling hemoglobin levels.

Decisions regarding patients' management were made by their own physicians. The authors were involved in the treatment only of those patients under their personal care. Decisions regarding the most appropriate treatment for each patient were reached on an individual basis without any concern for their eventual bearing on the conclusions of this study. The surgical procedures were carried out by a number of western Canadian surgeons.

Follow-up data were obtained by the authors by telephone contact or written correspondence with the patients, their relatives, or their physicians whether the patients continued to live in the Okanagan Valley or moved elsewhere. The patients were followed until either their death, in which case the data of death and the cause of death were sought, or until 31 December 1979 in which case the state of their health and details of any intervening treatment for AAA were sought. Follow up was 100% complete.

The annual incidence of newly diagnosed cases of AAA was calculated for those patients who resided in School District No. 23, a geographically-circumscribed area in the Okanagan Valley. The designation 'resident' was applied rather arbitrarily on the basis of longstanding residence in School District No. 23 or on the basis of apparent intent to become a long-term resident. (Difficulties in defining 'resident' arose because the Okanagan Valley has long been a popular tourist and retirement haven.) The population data for School District No. 23 were provided by the Division of Vital Statistics, Ministry of Health, Province of British Columbia and were based on census data collected in 1961, 1966, 1971 and 1976 together with extrapolations and interpolations by the authors to cover the years 1960 through 1979.

For those patients whose aneurysms were intact at the time of enrolment and who were observed to progress to rupture of the AAA before 31 December 1979, the rate of rupture was calculated by the actuarial approach (Anderson *et al.* 1974).

The observed survival rates were calculated using the life table method (the standard errors by Greenwood's formula) (Anderson *et al.* 1974, Irwin 1971) for 4 categories of patients: those who had medical management for intact AAA; those who had elective surgical resection for intact AAA; those who had emergency surgical intervention for ruptured AAA; those who did not have surgical intervention for ruptured AAA. The relative survival rates (Irwin 1971) were computed from the formula:

$$\text{Relative survival rate} = \frac{\text{observed survival rate}}{\text{expected survival rate}} \times 100\%$$

The expected survival rates were based on the p_x values (proportion surviving) in Life Tables, Canada and Provinces, 1970–1972, using the data for British Columbia males and females.

Results

Between 1 January 1960 and 31 December 1979 the diagnosis of AAA was established in 188 patients. They are subdivided in Table 1 into several categories on diagnostic and therapeutic grounds. Eighty-four percent were diagnosed during life. The most common explanations for failure to diagnose AAA during life were that (1) the patient did not have antemortem medical

Table 1. Classification and numbers of patients (in parentheses) with AAA (total 188 cases)

1. Not recognized during life; first diagnosed at necropsy
   - *1.1* Intact (18)
   - *1.2* Ruptured (12)
2. Diagnosed during life
   - *2.1* Intact when first diagnosed
     - *2.1.1* Aneurysm not operated (53)
     - *2.1.2* Aneurysm remained intact; operated at time of election (69)
     - *2.1.3* Aneurysm subsequently ruptured; operated at time of rupture (3)
   - *2.2* Ruptured when first diagnosed
     - *2.2.1* Not operated (5)
     - *2.2.2* Operated (28)

Table 2. Determination whether AAA intact at diagnosis did or did not later rupture. Numbers of patients in parentheses●

Aneurysm remained intact
Patients still alive 31 December 1979 and aneurysm presumed not to have ruptured (11)
Patients who died prior to 31 December 1979 and were believed, on the basis of noninvasive studies, to have had intact aneurysms at death (16)
Patients proven by laparotomy to have maintained intact aneurysms (71)
Patients proven by necropsy to have maintained intact aneurysms (7)
Aneurysm later ruptured
Aneurysm believed on the basis of noninvasive studies to have ruptured (9)
Aneurysm proven by laparotomy to have ruptured (3)
Aneurysm proven by necropsy to have ruptured (4)

●Total 121 cases. In 4 cases no information was available on status of aneurysm at time of death

attention during the terminal illness; and (2) 'small' aneurysms were not suspected on physical examination.

At the time of first diagnosis of AAA, rupture was noted in 24% of cases, a higher frequency (40%) being observed in those cases first diagnosed at necropsy than in those first diagnosed during life (21%)

The annual incidence of newly-diagnosed cases of AAA in School District No. 23 (derived from a total of 147 cases in 955 498 citizen-years) was 1:6500 (1:3800 for males and 1:21 000 for females).

The sex distribution of patients whose AAA did not rupture during the period of observation, based on a total of 123 cases, was 101 males and 22 females (82% and 18% respectively). The sex distribution of patients whose AAA ruptured during the same period, based on a total of 61 cases, was 51 males and 10 females (84% and 16%). The average age of patients at the time of first diagnosis of an intact AAA, based on a total of 142 cases (117 males and 25 females) was for males 71.6 years (range 55.6–88.7) and for females 75.8 years (range 56.7–98.1). The average age of patients at the time of rupture of AAA, based on a total of 61 cases (52 males and 9 females) was for males 76.1 years (range 53.3–92.6) and for females 74.5 years (range 55.7–93.2).

Five patients (3%) had a positive family history of AAA, 4 with a total of 6 affected brothers and one with one affected sister. Serologic tests for syphilis were performed in only a few patients and were positive in 2 (1%).

Haemodynamically important aortic valve disease was recognized in 4 patients: 2 had aortic stenosis, one combined stenosis and regurgitation, and one sufficient regurgitation to require aortic valve replacement. Four patients had associated saccular aneurysms of the aortic arch, 4 had saccular aneurysms of the descending thoracic aorta, 19 had iliac aneurysms, and 3 had femoral aneurysms. Two patients had dissections of the thoracic aorta proven at necropsy: one had survived for 18 months following his dissection; the other died at dissection.

The data used to determine whether AAA intact at the time of initial diagnosis did or did not subsequently rupture are outlined in Table 2. For the 121 patients for whom information was available (category 2.1), this determination was based on noninvasive studies in 36 cases (30%), on laparotomy in 74 cases (61%), and on necropsy findings in 11 cases (9%).

The average age at death of those patients with an intact AAA both at diagnosis and at death (based on a total of 43 cases, 33 males and 10 females) was for males 76.1 years (range

Table 3. Observed survival rates (%) for patients with AAA diagnosed during life

Patient categories	Years					Standard error of 5-year survival rate
	1	2	3	4	5	
AAA intact at diagnosis; medical management; survival from date of diagnosis●	78	61	44	39	33	7.5
AAA intact at diagnosis; operated at time of election; survival from date of surgery■	91	87	83	77	74	6.9
AAA operated at primary rupture; survival from date of surgery▲	50	46	41	35	35	9.9
Not operated at primary rupture of AAA; survival from date of rupture*	0	0	0	0	0	—

●Includes 53 patients from category 2.1.1 (Table 1); 68 patients from category 2.1.2 (one patient omitted because age at AAA diagnosis unknown); and 3 patients from category 2.1.3 from date of diagnosis to date of surgery
■Includes 69 patients from category 2.1.2
▲Includes 3 patients from category 2.1.3 and 28 patients from category 2.2.2 (patients with secondary rupture of AAA included once only, i.e. from date of operation for primary rupture to date of secondary rupture)
*Includes 13 patients from category 2.1.1 and 5 patients from category 2.2.1

Table 4. Relative survival rates (%) for patients with AAA diagnosed during life●

Patients	Years				
	1	2	3	4	5
AAA intact at diagnosis; not operated	82	69	52	49	45
AAA intact at diagnosis; operated at time of election	94	95	94	92	92
Operated at rupture of AAA	53	51	48	43	46
Not operated at rupture of AAA	0	0	0	0	0

●Footnotes to Table 3 also apply here
If the patients with AAA diagnosed during life had survived as long as an equivalent cohort from the general population, their relative survival rates at annual intervals would have remained constant at 100%. The extent to which patients failed to live as long as the general population may be attributed to the AAA, to its treatment or to other disease associated with AAA

61.3–86.1) and for females 81.7 years (range 61.8–98.4). The observed survival rates for those patients with AAA diagnosed during life are presented in Table 3, and their relative survival rates in Table 4.

Of 125 living patients with an intact AAA at the time of diagnosis (category 2.1), 13 (10%) were noted to progress to rupture within 5 years of diagnosis. Expressing the incidence of rupture of AAA in this way disregards those patients in whom the possibility of rupture during those 5 years was removed: e.g. 65 patients in category 2.1.2 who had elective surgery; 23 patients who died from other causes; and 8 patients who were still alive on 31 December 1979. Using the actuarial approach to analysis, it was calculated (Table 5) that the cumulative risk of rupture of AAA within 5 years of diagnosis of AAA in 120 of 125 of these cases was 35%.

Of the 31 patients who had emergency surgical treatment for ruptured AAA, 3 died from secondary ruptures 1.6, 7.9 and 9.0 years later.

Discussion

Previous studies of large groups of patients with AAA have been retrospective; that is, records of patients with the diagnosis of AAA have been extracted from clinic or hospital files, and the course of these patients has been reviewed from the time of diagnosis of AAA to death, to surgery, to rupture, or to the date of the study. The present investigation was initiated as a

prospective study in the original sense of the term 'prospective'; that is, patients were enrolled at or about the date of diagnosis of AAA and their course followed until death or to 31 December 1979, the closing date for enrolment. It may be argued that our study is not 'prospective' in the current sense of this term because patient management was not predetermined by a protocol defined by the authors. This evolving connotation of the adjective 'prospective' may perhaps best be accommodated by suggesting that the data in this study have been obtained by a prospective observational approach rather than by a prospective experimental approach.

Two major considerations in the conduct of a prospective study are the diagnostic criteria for inclusion of patients in that study and the possible loss of patients to follow up. The diagnostic criteria in the present study have been stringent; some might argue unduly stringent. Palpation of a pulsatile midabdominal mass was considered insufficient because there may be difficulty in distinguishing on physical examination between a tortuous elongated abdominal aorta and an AAA, a difficulty emphasized by Osler as early as 1892 and more recently by Freeark & Weinberg (1967). One might speculate that the authors' policy, while it undoubtedly strengthened the diagnoses of AAA in this study, may have delayed these diagnoses and thus influenced the conclusions. None of the patients were lost to follow up. Irwin's (1971) dictum that, statistically, the only correct way of handling patients lost to follow up is to have none, has been heeded.

The omission from this report of data concerning the sizes of AAA merits comment. Prior to the introduction of echoaortography in this community, three methods of determining the sizes of AAA were available, each with its own drawbacks: (1) measurement from radiographic films; (2) estimation by palpation by surgeons; and (3) measurement by pathologists at the time of necropsy when the *in vivo* distending pressure was no longer present. Measurement of the sizes of AAA by echoaortography became available approximately mid-way during the data collection phase of this study, but it was felt that data concerning the sizes of AAA collected up to that time could not reasonably be added to nor compared with the data collected after the introduction of echoaortography. Moreover, if the survival data for non-operated and operated patients were further subdivided, depending upon whether echoartography had or had not been performed, the number of patients in each sub-set would have been so small that their significance would be questionable. For these reasons, all data concerning AAA size have been omitted from this report.

This study was made possible by an unusual combination of circumstances, among which the following are probably the most important: (1) a relatively circumscribed population residing in an intermountain valley permitting relatively easy follow up; (2) the cooperative camaraderie of the medical staff of the hospital serving this population; and (3) the continuity provided by the senior author's long-term practice in that hospital. While it cannot be claimed that all patients with AAA in this community have been included in this study, the goal that all diagnosed cases should be enrolled in this study has probably been attained. The multiple sources of information about patients, e.g. their attending physicians, the hospital Departments of Radiology and Pathology and the Medical Records Department of the hospital, served as separate screens for the identification of affected patients. Thus, the incidence data for diagnosed patients are thought to be accurate. The demographics of the population in this study are known not to be representative of the general population of Canada in that the former has a higher incidence of elderly patients than does the latter. For this reason, it is probable that the incidence figures reported here are higher than might be expected from a countrywide study.

In this study, the annual incidence of patients with newly-diagnosed AAA coming to medical attention was 1:6500 citizen-years. The sex distribution, as in previous studies, showed a 5:1 predominance of males over females whether the aneurysm remained intact or ruptured during the period of observation; the explanation for this gender predilection remains unknown. AAA is a disorder of later life. In this study, the average age at the time of first diagnosis of an intact AAA was 71.6 years for males and 75.8 years for females; the average age at the time of rupture of AAA was 76.1 years for males and 74.5 years for females.

Table 5. Rates of rupture of intact AAA

Year	Population exposed to risk of rupture at onset of interval	No. of ruptures (*a*)	Deaths from other causes	Withdrawn alive	Population exposed to risk of rupture during interval (*b*)	Rate of rupture during the year ($a/b \times 100\%$)	Cumulative rate of rupture■ (%)
0–1	120●	7	13	60	83.5	8	8
1–2	40	2	5	10	32.5	6	14
2–3	23	1	4	1	20.5	5	18
3–4	17	1	1	1	16	6	23
4–5	14	2	0	1	13.5	15	35
5+	11	3	2	6	—	—	—

● 5 cases omitted: one because date of diagnosis unknown, and 4 because status of aneurysm at death uncertain
■ Calculated by multiplying annual rates of non-rupture to yield cumulative rates of non-rupture, and then subtracting these values from 100%

The occurrence of AAA in siblings of 3% of our patients was surprising. None of our patients had affected parents, children, or spouses. Unfortunately, our protocol did not include a search for affected cousins. Could AAA be inherited as a recessive trait? None of our patients was considered to have Marfan's syndrome (which may be inherited as a dominant trait). The older medical literature (from the era in which late manifestations of syphilis were not uncommon) emphasizes the importance of syphilis in patients with AAA. Two patients did have positive serologic tests for syphilis, probably coincidental rather than aetiological. The frequency of associated saccular aneurysms of the aortic arch, the descending thoracic aorta, and the iliac and femoral arteries was also surprising. These numerous associated saccular aneurysms and the 2 associated aortic dissections support the concept of atherosclerosis as a diffuse disorder affecting major arteries in segmental fashion.

The medical literature abounds with data on the frequency with which intact AAAs progress to rupture (Schatz *et al.* 1962, Klippel & Butcher 1966, Szilagyi *et al.* 1966, Bernstein *et al.* 1967). These studies are difficult to compare because of differences in the criteria for the diagnosis of AAA, in the duration of follow up, and in the methods of calculating and presenting the results. In this study, the cumulative risk of rupture of an intact AAA over a 5-year period following diagnosis was 35% when calculated by the actuarial approach.

Attempts to evaluate the benefits of surgery in prolonging the lives of certain patients with AAA have been the major concern of several previous studies (Szilagyi *et al.* 1966, Bernstein *et al.* 1967, Foster *et al.* 1969). The present study illustrates that at least part of the difficulty in this evaluation can be attributed to the longevity of patients with AAA not treated surgically. For example, in this study, the average age at death of 43 patients who did not have elective surgery and whose aneurysms remained intact at the time of their death was 77.4 years. The average age at rupture in 61 patients was 75.9 years. Undaunted by such favourable survival data in unoperated patients, surgeons have attempted to augment the longevity of patients with AAA.

There can be no doubt that emergency surgical intervention increases the survival rates for patients with ruptured AAA. In this study, the observed 5-year survival rate for 31 patients operated at the time of rupture was 35% and for 18 patients not operated at the time of rupture was 0%. The corresponding relative survival rates were 46% and 0% respectively.

Our data can be used to predict the expected survival rates of patients with AAA intact at the time of diagnosis managed by withholding elective surgery, and performing emergency surgery when and if the AAA ruptures. The data required for these calculations include: (1) annual risks of rupture of an intact AAA (Table 5); (2) annual survival rates for an equivalent cohort from the general population (Life Tables 1974); (3) annual survival rates following

Table 6. Demonstration that the group of patients who did not have elective surgery for AAA is weighted with high-risk patients

	Years				
	1	2	3	4	5
Observed survival rates for non-operated patients (from Table 3) (*a*)	78	61	44	39	33
Cumulative rates of rupture of intact AAA (from Table 5) (*b*)●	8	14	18	23	35
Predicted survival rates for non-operated patients (*a*+*b*)	86	75	62	62	68
Observed survival rates for electively-operated patients (from Table 3)	91	87	83	77	74

●The possibility of salvaging perhaps half of these patients by emergency surgery at the time of rupture of AAA is disregarded here

emergency surgery for ruptured AAA (Table 3). The projected survival rates for this management protocol at 1, 2, 3, 4, and 5 years following the diagnosis of an intact AAA are 92%, 84%, 72%, 61% and 47% respectively, distinctly less favourable than the survival rates following a policy of management by elective surgery for selected patients with intact AAA (Table 3).

The question has been debated whether elective surgical resection of an intact AAA increases the duration of survival of affected patients. One might hope that comparison of the survival rates of operated and non-operated patients might answer this question. The survival rates (Tables 3 and 4) at first glance suggest impressive benefits from elective surgery for intact AAA. Unfortunately, there is objective evidence that a significant process of selection influenced the composition of the operated and non-operated groups. For example, there was an important age difference between patients whose only treatment was conservative (category 2.1.1: 53 patients, average age 76.7 years at diagnosis) and those treated by elective surgery (category 2.1.2: 69 patients, average age 68.8 years at surgery). This age difference is not adjusted for in the observed 5-year survival rates of 33% and 74% respectively, but is adjusted for statistically in the relative survival rates of 45% and 92% respectively. Moreover, evidence that the non-operated group is disproportionately weighted with high-risk patients can be developed from the following reasoning: if the non-operated group was comparable with the operated group, one would predict that, if the potential mortality rates from rupture of an intact AAA are added to the observed survival rates of non-operated patients with intact AAA, figures should be obtained which approximate (or exceed, because of the mortality rate attendant upon elective surgery) the observed survival rates of patients who did have elective surgery for intact AAA. The numerical expression of this hypothesis is presented in Table 6. The fact that the observed survival rates in the operated group were higher than can be predicted from the observed survival rates in the non-operated group, clearly shows that the non-operated group is more highly weighted with poor-risk cases than in the operated group. To permit firm conclusions from comparison of survival rates of operated and non-operated groups will require studies using protocols such as matching pairs of patients in the operated and non-operated groups or random assignment to operated and non-operated groups.

Another approach to assessing the role of elective surgery in increasing the duration of survival of patients with intact AAA is for the mortality rates associated with elective surgery to be compared with the risks of rupture of an intact AAA. (It should be emphasized that the mortality rate of elective surgery is associated both with the surgical procedure and with manifest arteriosclerotic disease expected to be greater than that of the general population.) As long as the mortality rates of elective surgery are less than the potential mortality rates due to rupture, elective surgery can be expected to increase the duration of survival of patients with intact AAA. The mortality rates associated with elective surgery can be calculated from the equation:

Table 7. Comparison of the mortality rates after elective surgery for AAA with the potential mortality rates resulting from rupture of intact AAA●

	Years				
	1	2	3	4	5
Observed survival rates after elective surgery (from Table 3)	91	87	83	77	74
Annual mortality rates after elective surgery (*a*)	9	4	5	7	5
Survival rates for comparable cohort from general population (Life Tables 1974)	96	93	89	85	80
Annual mortality rates for comparable cohort from general population (*b*)	4	3	3	3	7
Excess deaths annually after elective surgery (*a–b*)	5	1	2.	4	–2 (substitute 0)
Cumulative rates of excess deaths after elective surgery■	5	6	8	12	12
Cumulative rates of rupture (from Table 5)	8	14	18	23	35

●The footnote to Table 6 applies here also
■See footnote ■ to Table 5 for calculation method

Calculated mortality rate associated with management of AAA by elective surgery = Expected survival rate for a comparable cohort from the general population minus observed survival rate after elective surgery for AAA

Comparison of the first and third lines of Table 7 shows that elective surgery in this study achieved survival rates approaching those for the general population, and that there is scant margin for improving survival rates in patients managed by elective surgery. The 74% 5-year survival rate after elective surgery for AAA compares favourably with the 68% reported by Fielding *et al.* (1981) for a retrospective study embracing the same time period (1960 through 1979). When the cumulative rates of excess deaths after elective surgery (line 6, Table 7) are compared with the cumulative rates of rupture for AAA (line 7, Table 7) it will be seen that elective surgery can be expected to reduce the mortality rates for patients with intact AAA over a 5-year period of follow up. Moreover, extrapolation of the data in Table 7 suggests that the benefits of elective surgery may be expected to become even more substantial with longer follow up.

What does the future hold for elective surgery for AAA? On the one hand, it seems reasonable to predict that improvements in elective surgical treatment of patients with intact AAA may further reduce the mortality rates associated with this surgery. On the other hand, these same improvements may result in elective surgery being extended to higher-risk patients, thus affecting adversely the mortality rates associated with elective surgery. Clearly, this is a subject which will require continuing careful scrutiny.

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References

Anderson R P, Bonchek L I, Grunemeier G L, Lambert L E & Starr A (1974) *Journal of Surgical Research* **16,** 224–230
Bernstein E F, Fisher J C & Varco R L (1967) *Surgery* **61,** 83–93

Crane C (1955) *New England Journal of Medicine* **253,** 954–958
Estes J E (1950) *Circulation* **2,** 258–264
Fielding J W, Black J, Ashton F, Slaney G & Campbell D J (1981) *British Medical Journal* **283,** 355–359
Foster J H, Bolasny B L, Gobbell W G & Scott H W (1969) *Surgery, Gynecology and Obstetrics* **129,** 1–9
Freeark R F & Weinberg M (1967) *Medical Clinics of North America* **51,** 193–214
Irwin A C (1971) *Canadian Medical Association Journal* **105,** 489–494, 497
Jackson T L (1963) *Journal of Abdominal Surgery* **5,** 61–62
Janower M L (1961) *New England Journal of Medicine* **265,** 12–15
Klippel A P & Butcher H R (1966) *American Journal of Surgery* **111,** 629–631
Life Tables, Canada and Provinces, 1970–1972 (1974) Catalogue 84–532. Statistics Canada, Ottawa; pp 50–53
Mulder D S, Blundell P E, Scott H J, Cole C M & Winsberg F (1973) *Annals of the Royal College of Physicians and Surgeons of Canada* **6,** 13
Osler W (1892) The Principles and Practice of Medicine. D Appleton & Co, New York; pp 680–681
Schatz I J, Fairburn J F & Juergens J L (1962) *Circulation* **26,** 200–205
Szilagyi D E, Smith R F, DeRusso F J, Elliott J P & Sherrin F W (1966) *Annals of Surgery* **164,** 678–697