Use of ankle brachial pressure index to predict cardiovascular events and death: a cohort study

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Abstract

***Objective*—To determine whether a low ankle brachial pressure index is associated with an increased risk of cardiovascular events and death, and whether the prediction of such events could be improved by including this index.**

***Design*—Cohort study.**

***Setting*—11 practices in Edinburgh, Scotland.**

***Subjects*—1592 men and women aged 55-74 years selected at random from the age-sex registers of 11 general practices and followed up for 5 years.**

***Main outcome measures*—Incidence of fatal and non-fatal cardiovascular events and all cause mortality.**

***Results*—At baseline 90 (5.7%) of subjects had an ankle brachial pressure index ≤0.7, 288 (18.2%) had an index ≤0.9, and 566 (35.6%) ≤1.0. After five years subjects with an index ≤0.9 at baseline had an increased risk of non-fatal myocardial infarction (relative risk 1.38, 95% confidence interval 0.88 to 2.16), stroke (1.98, 1.05 to 3.77), cardiovascular death (1.85, 1.15 to 2.97), and all cause mortality (1.58, 1.14 to 2.18) after adjustment for age, sex, coronary disease, and diabetes at baseline. The ability to predict subsequent events was greatly increased by combining the index with other risk factors—for example, hypertensive smokers with normal cholesterol concentrations had a positive predictive value of 25.0%, increasing to 43.8% in subjects with a low index and decreasing to 15.6% in those with a normal index.**

***Conclusion*—The ankle brachial pressure index is a good predictor of subsequent cardiovascular events, and improves on predictions by conventional risk factors alone. It is simple and accurate and could be included in routine screening of cardiovascular status.**

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Introduction

Coronary heart disease is the main cause of death and disability in elderly people,[1] and numerous primary and secondary prevention trials have attempted to reduce its impact.[2] Important risk factors include hypercholesterolaemia, hypertension, and cigarette smoking,[3] and attempts have been made to target those at greatest risk by using scoring systems such as the Dundee method[4] or by identifying subjects with early asymptomatic atheroma.[5] Several cohort studies have shown that subclinical atherosclerosis is associated with an increased risk of subsequent cardiovascular events,[5-8] but there is currently no universally accepted method for detecting early atheroma in the general population.

One commonly used non-invasive test of cardiovascular disease is the ankle brachial pressure index. It is quick and easy to measure, has high patient acceptability, and is an accurate and reliable indicator of atherosclerosis.[9-11] Increased mortality has been shown to be associated with a low ankle brachial pressure index in patients referred for vascular investigation in Pittsburgh,[12] in asymptomatic working men in Belgium[7] and in elderly white women at risk of fracture.[13] The ankle brachial pressure index has also been shown to predict cardiovascular events in conjunction with other tests,[5 6] and as an independent measure,[7 8 12 13] but before it can be considered as a possible screening tool its ability to predict cardiovascular events must be compared with that of conventional risk factors. We investigated the predictive value of the index in subjects included in the Edinburgh artery study.[14]

Subjects and methods

The Edinburgh artery study began in 1988 as a cross sectional survey of 1592 men and women aged 55-74 years. This population was selected at random, in five year age bands, from 11 general practices serving a range of socioeconomic and geographical areas throughout the city. The response rate was 65%, and follow up of a sample of non-responders showed no substantial bias. Details of recruitment have been described.[14] Participants were followed up over five years to detect cardiovascular events and cause of death. At the end of five years, 102 subjects (6.4%) were lost to follow up. The study was approved by Lothian Health Board ethics subcommittee, and informed consent was obtained from all participants.

BASELINE INVESTIGATIONS

Subjects were invited to a university clinic where we administered a questionnaire including validated questions on smoking, history of diabetes, and angina from the World Health Organisation questionnaire.[15] A clinical examination was then conducted by two pairs of specially trained nurses. They recorded systolic and diastolic (phase V) blood pressures in the right arm after 10 minutes' rest with a random zero sphygmomanometer. Ankle systolic pressures were measured in the posterior tibial artery of the right then left leg with a Doppler ultrasound probe (Sonicaid, Chichester) and a random zero sphygmomanometer with the cuff positioned proximal to the malleoli. The pulse was located with the Doppler probe, and the cuff inflated until the pulse was obliterated; the cuff was then deflated and the pressure recorded at the point when the pulse reappeared. A 12 lead electrocardiogram was recorded and coded independently by two observers using the Minnesota code.[16]

A sample of fasting blood was taken after five minutes' rest in the supine position to measure serum lipid concentrations, including total cholesterol, on a Cobas Bio analyser (Roche, Welwyn Garden City) with standard kits. Laboratory standardisation was carried out with commercially available standards (Wellcome scheme), and quality was assessed by examining systematic and random error against two control materials (Precipath universal bovine serum, Boehringer Mannheim, Lewes and pooled donated sera). The laboratory is standardised against the WHO Regional Lipid

Table 1—*Five year incidence of non-fatal cardiovascular events and mortality according to baseline ankle brachial pressure index. Values are numbers (percentages)*

	Ankle brachial pressure index*					
	>1.1 (n = 538)	1.1-1.01 (n = 478)	1.0-0.91 (n = 278)	0.9-0.71 (n = 198)	≤0.7 (n = 90)	P value for trend
Non-fatal events:						
Myocardial infarction	23 (4)	23 (5)	14 (5)	13 (7)	8 (9)	0.057
Stroke	4 (1)	9 (2)	7 (3)	5 (3)	3 (3)	0.020
Deaths:						
Myocardial infarction	13 (2)	12 (3)	10 (4)	11 (6)	9 (10)	≤0.001
Stroke	7 (1)	5 (1)	4 (1)	1 (1)	5 (6)	0.139
All cardiovascular causes†	20 (4)	18 (4)	17 (6)	15 (8)	19 (21)	≤0.001
Non-cardiovascular causes	38 (7)	32 (7)	13 (5)	17 (9)	12 (13)	0.188
All causes	58 (11)	50 (10)	30 (11)	32 (16)	31 (34)	≤0.001

*10 subjects not classified due to missing index, of whom two subsequently died.
†Including aneurysm, thromboembolism, stroke, and myocardial infarction.

Reference Laboratory, Prague, Czech Republic. Fasting blood glucose concentration was also measured. Each subject was then asked to consume 75 g of glucose in a drink. A second blood glucose specimen was taken two hours after the oral glucose load.

IDENTIFICATION OF CARDIOVASCULAR EVENTS

We obtained information on cardiovascular events and deaths from general practitioners, who were asked to attach a card to the front of subjects' records at the start of the study and return it after a cardiovascular event or if the patient changed address or doctor; the information and statistics division of the Scottish Office Home and Health Department, which provided annual computer printouts of all hospital discharges in Scotland; hospitals, where medical records were investigated for discharges with relevant ICD-9 codes; and the participants themselves, who received an annual questionnaire asking about the development of chest pain, heart attack, and stroke as well as hospital attendances and general practitioner visits in the previous year.

To identify all deaths occurring in the cohort each participant's record was flagged at the NHS central registry so that certificates would be automatically forwarded. All deaths from cardiovascular causes were further investigated by using hospital or general practitioner records to ensure that the protocol criteria were fulfilled.

The criteria to define fatal or non-fatal myocardial infarctions and stroke were adapted from those proposed by the American Heart Association[17] and are given in the appendix.

DATA ANALYSIS

Information on the questionnaire and recording forms was checked by the clinic staff, coded, and entered on to a DBASE IV database. Error rates were determined by dual entry of all data, and any discrepancies were checked by reference to the original records.

The ankle brachial pressure index for each leg was calculated by dividing the ankle systolic pressure by the brachial systolic pressure. The lower of the indices obtained for the two legs was used as the measure of disease severity in the analysis.

Multiple events of the same type occurring in the same subject, such as two myocardial infarctions, were counted only once. The χ^2 test for trend was used to assess differences in non-fatal events and death between categories of subjects determined by baseline ankle brachial pressure index. Survival analysis based on the product limit or Kaplan-Meier estimate of the survival function was used to produce survival curves for non-fatal cardiovascular events and all cause mortality. The trend version of the Wilcoxon (Breslow) test was used to identify any survival differences after adjustment for the confounding effects of age and sex with a stratified analysis. Relative risks of fatal and non-fatal events were calculated for subjects with an ankle brachial pressure index at baseline ≤0.9 compared with those with an index above 0.9. Relative risks were adjusted for age, sex, and presence of angina, myocardial infarction, or diabetes mellitus at baseline by multiple logistic regression.

To determine the usefulness of the index as a diagnostic test we calculated sensitivities, specificities, and likelihood ratios. We used a cut off point of 0.9 to define a low index in our calculations because it is a sensitive and specific measure of peripheral vascular disease in a clinical setting.[9]

Results

All but two of the subjects were white, reflecting the composition of the general population of this age in Edinburgh. The distribution of the ankle brachial pressure index at baseline was slightly negatively skewed, with a mean of 1.03 (SD 0.18). Ten subjects refused to undergo the test, and of the remaining 1582 subjects, 90 (5.7%) had an index ≤0.7, 288 (18.2%) ≤0.9, and 566 (35.8%) ≤1.0. One hundred and forty four subjects (9.0%) had a myocardial infarction during the five year follow up, of whom 55 died. A stroke occurred in 50 (3.1%) subjects, and 22 were fatal. Out of a total of 203 deaths, 89 (44%) were due to cardiovascular causes.

The lower the ankle brachial pressure index at baseline, the greater the occurrence of non-fatal myocardial infarction (P = 0.057) and stroke (P = 0.020) (table 1). A similar relation was seen between the index and death from myocardial infarction (P≤0.001) and all cardiovascular causes (P≤0.001). Deaths from non-cardiovascular causes or stroke were not significantly related to the index at baseline.

Figure 1 shows that the lower the ankle brachial pressure index at baseline, the lower the probability of survival (P≤0.001), independent of age and sex. Similar patterns were found for the probability of surviving without a non-fatal myocardial infarction or stroke. Both the probability of survival and of not having a myocardial infarction or stroke declined most when the index was below 0.9.

Patients with a baseline index ≤0.9 had a slightly increased risk of non-fatal myocardial infarction (P = 0.085) and an increased risk of non-fatal stroke (P≤0.05), independent of age and sex (table 2). A low index was associated with increased relative risks of death from myocardial infarction (P≤0.01), all cardiovascular causes (P≤0.001), and all causes (P≤0.001), independent of age and sex. There was no increased risk of non-cardiovascular death. Adjustment

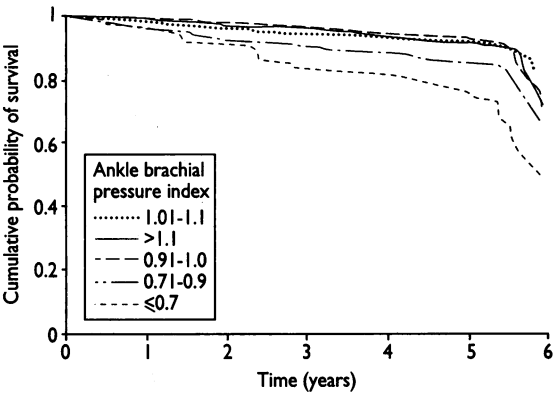

Fig 1—*Product-limit survival curves based on mortality from all causes according to ankle brachial pressure index at baseline*

Table 2—*Relative risk (95% confidence interval) of non-fatal cardiovascular events and death for subjects with an ankle brachial pressure index ≤0.9 at baseline, adjusted for age and sex, cardiovascular disease, and diabetes at baseline*

	Adjusted for age and sex	Adjusted for age, sex, angina, and myocardial infarction†	Adjusted for age, sex, angina, myocardial infarction, and diabetes‡
Non-fatal events:			
Myocardial infarction	1.39 (0.90 to 2.16)	1.35 (0.86 to 2.10)	1.38 (0.88 to 2.16)
Stroke	1.91* (1.04 to 3.51)	1.85* (1.00 to 3.43)	1.98* (1.05 to 3.77)
Deaths:			
Myocardial infarction	2.21** (1.25 to 3.90)	2.09* (1.17 to 3.73)	1.89* (1.03 to 3.46)
Stroke	1.31 (0.51 to 3.39)	1.08 (0.41 to 2.90)	0.91 (0.31 to 2.66)
All cardiovascular causes§	2.29*** (1.48 to 3.56)	2.05** (1.30 to 3.21)	1.85** (1.15 to 2.97)
Non-cardiovascular causes	0.98 (0.89 to 1.08)	0.98 (0.89 to 1.08)	0.99 (0.89 to 1.09)
All causes	1.79*** (1.32 to 2.43)	1.68*** (1.23 to 2.30)	1.58** (1.14 to 2.18)

†Defined at the baseline examination by the WHO questionnaire.
‡Defined at the baseline examination by the WHO questionnaire and by doctor recall and glucose tolerance test.
§Death from all cardiovascular causes, including aneurysm, thromboembolism, stroke, and myocardial infarction.
*P≤0.05, **P≤0.01, ***P≤0.001.

Table 3—*Sensitivity, specificity, and likelihood ratios of ankle brachial pressure index in predicting cardiovascular events*

Ankle brachial pressure index	Cardiovascular event†		Sensitivity (%)	Specificity (%)	Likelihood ratio
	Yes	No			
≤0.9	52	236	29.6	83.2	1.76
>0.9	124	1170			
≤0.7	25	65	14.2	95.4	3.07
>0.7	151	1341			

†Includes non-fatal myocardial infarction and stroke plus death from all cardiovascular causes.

Table 4—*Positive predictive values for subsequent cardiovascular events for smoking, hypertension, hypercholesterolaemia, and ankle brachial pressure index*

Risk factors	Cardiovascular event†		Positive predictive value (%)		
			Based on risk factors only	Including ankle brachial pressure index	
	Yes	No		≤0.9	>0.9
Smoker‡					
Hypertensive§:					
≥6.5 mmol/l cholesterol	15	66	18.5	22.9	15.2
<6.5 mmol/l cholesterol	12	36	25.0	43.8	15.6
Normotensive:					
≥6.5 mmol/l cholesterol	23	211	9.8	7.6	10.7
<6.5 mmol/l cholesterol	16	122	11.6	20.8	8.9
Non-smoker					
Hypertensive§:					
≥6.5 mmol/l cholesterol	24	178	11.9	16.7	10.4
<6.5 mmol/l cholesterol	17	104	14.0	15.7	13.5
Normotensive:					
≥6.5 mmol/l cholesterol	42	431	8.9	17.8	7.9
<6.5 mmol/l cholesterol	22	231	8.7	23.1	7.9

†Includes non-fatal myocardial infarction and stroke and death from all cardiovascular causes.
‡Smoker includes recent former smokers (≤5 years).
§Hypertension defined using WHO criteria: systolic pressure >160 mm Hg or diastolic pressure >95 mm Hg.

for angina, myocardial infarction, or diabetes mellitus at baseline only slightly reduced the risks.

An ankle brachial pressure index ≤0.9 showed moderate specificity (83.2%), low sensitivity (29.6%), and a likelihood ratio of 1.76 in predicting fatal and non-fatal cardiovascular events after five years (table 3). However, a lower index (≤0.7) showed better specificity (95.4%) and a higher likelihood ratio (3.07) but a lower sensitivity (14.2%). When subjects with diabetes were excluded the likelihood ratios were reduced (1.49 for an index ≤0.9 and 1.52 for an index ≤0.7).

The positive predictive value for a future cardiovascular event was 17.6% (95% confidence interval 13.1% to 22.1%) for subjects with an ankle brachial pressure index ≤0.9 compared with 9.6% (8.0% to 11.2%) for those with an index >0.9. Positive predictive values were also higher for smokers (13.2%, 10.2% to 16.2%) than non-smokers (10.0%, 8.2% to 11.8%) and for hypertensive patients (15.0%, 11.7% to 18.3%) than those with normal blood pressure (9.4%, 7.7% to 11.1%), but the predictive value was lower for those with high cholesterol concentrations (10.5%, 8.6% to 12.4%) compared with those with normal values (12.0%, 9.3% to 14.7%). However, the ability to predict subsequent events was greatly increased by combining a low ankle brachial pressure index ≤0.9 with other risk factors (table 4). In general, predictive values increased in the presence of a low index, particularly in normotensive non-smokers, and in smokers with low cholesterol concentrations. In most cases the predictive values for those with normal ankle brachial pressure indices were similar to those produced by risk factor status alone, with the exception of hypertensive smokers with a low cholesterol concentration, where the predictive value dropped from 25.0% to 15.6%.

Discussion

Over 18% of subjects in this study had an ankle brachial pressure index ≤0.9 at baseline. Thus almost one in five subjects would be identified as at risk should a population of this age be screened for early atherosclerosis. Other studies, however, have shown differing prevalences. In the cardiovascular health study only 12.4% of the population had an ankle brachial pressure index below 0.9,[18] and in 40-55 year old men in Belgium only 3.8% had a low index.[7] These differences undoubtedly reflect variations in the age structure of the study populations and the techniques of measurement as well as the underlying occurrence of atherosclerotic disease.

A relation between low ankle brachial pressure index and subsequent cardiovascular events might have been expected as lower limb disease is known to coexist with coronary and cerebrovascular disease.[19-20] Indeed, previous studies in selected groups of subjects have all shown that a low ankle brachial pressure index is associated with reduced survival.[7-13] This study shows that a low index is also associated with an increased risk of subsequent non-fatal cardiovascular events as well as death in the general population, independent of age, sex, and the presence of angina, myocardial infarction, and diabetes at baseline.

In this study, as in others, myocardial infarction was more common than stroke in subjects with lower limb disease.[12-21] One Swedish study also showed an increase in non-cardiovascular mortality in those with a low ankle brachial pressure index, possibly because this population contained a relatively high proportion of smokers dying from other smoking related diseases such as neoplasm.[22]

WHAT SHOULD THE CUT OFF VALUE BE?

Most studies,[7-13] including ours, show a significantly increased risk of death for an ankle brachial pressure index of 0.9 or lower. However, in the Pittsburgh study risk of a cardiovascular event became significant only for an index ≤0.7,[12] possibly because the overall prevalence of peripheral arterial disease in this sample was high, with over 75% of subjects having an index ≤0.9. In our study the likelihood ratio of a subsequent event was greater for subjects with an index ≤0.7 than those with an index ≤0.9, reflecting the more advanced atheroma in those with a lower score. For the purposes of screening, however, an index ≤0.9 would be more

likely to identify those with early disease without symptoms, who would not otherwise seek medical attention.

An index of ≤0.9 had a significantly greater predictive value for a subsequent cardiovascular event than higher values. Similarly, subjects who had hypertension or who smoked had higher predictive values than those who did not, but these differences were non-significant and lower than the predictive values associated with a low index. A low index reflects the combined effect of many risk factors over time and, once atherosclerosis has developed, would be expected to be a better predictor than any one risk factor alone. However, hypercholesterolaemia was associated with a lower predictive value than normal serum cholesterol concentration, possibly because in Edinburgh almost 65% of the baseline population had a cholesterol concentration ≥6.5 mmol/l. Alternatively, the predictive values of both high cholesterol concentration and hypertension may have been attenuated as a result of treatment given by general practitioners after the baseline examination. Total cholesterol is a poorer predictor than low density lipoprotein cholesterol,[23] and although the predictive value could have been slightly improved by analysing low density lipoprotein, we used total cholesterol because it is the most widely measured parameter in general practice.

In an attempt to increase the accuracy of prediction of cardiovascular events from risk factors we included baseline ankle brachial pressure index in the calculation of positive predictive values. As expected, predictive values were generally higher in those with low ankle brachial pressure indices than in those with higher values, with the biggest effect occurring in those with a low cholesterol concentration. It is difficult to draw firm conclusions about the types of subjects in whom the index adds the most in terms of prediction, but it is possible that it will be most useful in subjects with few other risk factors who would otherwise be considered at low risk of an event.

PRACTICAL USE

A low ankle brachial pressure index is related to an increased risk of both fatal and non-fatal cardiovascular events. Since the measurement is simple and quick it could be carried out in general practice when screening for cardiovascular disease. In addition to the routine measurement of brachial blood pressure, systolic ankle pressures can be quickly measured with an ultrasound probe, which general practitioners already often use for obstetric purposes. It is probably unnecessary to measure the index in younger people, in whom atherosclerosis is unlikely to be present, but in middle aged and elderly subjects it should be measured when blood pressure, serum cholesterol, and smoking are assessed. A low index would indicate those subjects who require additional monitoring. In the absence of other cardiovascular risk factors, a low index might indicate a high risk individual who could benefit from aspirin or other secondary preventive measures. Before such preventive therapies are routinely recommended, however, it is essential that randomised controlled trials are performed to show their effectiveness in this group of high risk individuals.

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Key messages

- The ankle brachial pressure index is a simple test which indicates the presence of generalised atherosclerosis
- In this study individuals with a low ankle brachial pressure index had an increased risk of fatal and non-fatal cardiovascular events
- The index was a good predictor of subsequent cardiovascular events, and improved that of conventional risk factors alone
- The ankle brachial pressure index could be included in routine screening of cardiovascular status
- Individuals with a low ankle brachial pressure index require additional monitoring, and might benefit from aspirin or other secondary preventive measures

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Appendix

The criteria used to define cardiovascular disease and deaths were as follows

MYOCARDIAL INFARCTION

Non-fatal, definite—Two of the following three criteria: (*a*) prolonged cardiac pain anywhere in the anterior chest, left arm, or jaw (possibly also affecting the back, shoulder, right arm, or abdomen) and lasting at least 20 minutes; (*b*) diagnostic electrocardiographic codes, including Minnesota codes: 1.1.1-1.2.5, 1.2.7, or 9.2 plus 5.1 or 5.2; (*c*) raised enzyme concentrations (creatine phosphokinase greater than twice the upper limits of normal, and one of the following also greater than twice the upper limits of normal: lactate dehydrogenase, aspartate aminotransferase, or the MB isoenzyme of creatine phosphokinase). The enzymes must have been measured within 72 hours of an acute event.

Non-fatal, possible—(*a*) One of the above definite criteria, plus either equivocal electrocardiographic codes (1.2.8-1.3.6, 4.1-4.3, 5.1-5.3, or 9.2) or equivocal enzyme levels (above normal but not twice normal, or one above twice normal but could be attributed to another cause); or (*b*) equivocal electrocardiographic codes and enzyme concentrations.

Fatal, definite—(*a*) Postmortem evidence of acute myocardial infarction; (*b*) definite criteria for myocardial infarction within the four weeks before death; or (*c*) ICD-9 codes for cause of death 410-414 plus history of a definite or possible myocardial infarction or 410-414 plus definite or possible criteria for myocardial infarction immediately preceding death or 410-414 plus postmortem evidence of severe coronary atherosclerosis or previous myocardial infarction.

Fatal, possible—Death certificate codes 410-414 but no other evidence.

STROKE

Non-fatal, definite—(*a*) History of onset of symptoms of less than 48 hours, plus clinical confirmation of a focal or global disturbance of cerebral function lasting more than 24 hours; or (*b*) computed tomography showing evidence of cerebral infarction or haemorrhage.

Non-fatal, possible—Primary or secondary discharge diagnosis including ICD-9 codes 431, 432, 434, 436, or 437.

Fatal, definite—One of the following: i) postmortem evidence of cerebral infarction or haemorrhage; b) criteria for definite stroke met within six weeks before death.

Fatal, possible—Death certificate codes of underlying or immediate cause of death were ICD 431-437, but no other evidence.

OTHER FATAL CARDIOVASCULAR EVENTS

Other fatal cardiovascular events (such as ruptured aneurysm or thromboembolism) were recorded if the diagnosis was confirmed by laboratory, radiological, surgical, or postmortem evidence.

A MEMORABLE PATIENT

Some monkey business

As a young research assistant in anaesthesia I found myself occasionally undertaking tasks that, to say the least, were unconventional. The most bizarre perhaps was when I was asked by our professor of surgery to anaesthetise his pet monkey for a total dental clearance.

Jacko, a small rhesus monkey, had several party tricks, not all of which were socially acceptable. On inquiring further, it seemed that he had taken to biting people, including the hand that fed him. It was decided that his teeth should be removed, and the task was to be undertaken by a senior dental surgeon, apparently in the professorial operating theatre. I had my doubts, and these were confirmed when the theatre sister made it clear that no monkey would enter her theatre.

Unabashed, the professor recruited his chief technician to organise an operating theatre in his home. I need scarcely add that the professor was a bachelor. On my arrival the surgeon was already scrubbing, and the technician was waiting to assist me. Jacko was crying like a baby and reacting to any approach by scratching, biting, and kicking. It was obvious that despite the professor's entreaties to "be a good boy, Jacko" I had little hope of entering a vein. After I had muttered repeatedly that induction was often easier with the patient's relatives out of the room the professor reluctantly disappeared. I thereupon seized a large needle and deposited a substantial dose of thiopentone in the peritoneal cavity; Jacko went out like a light. A suitable dose of relaxant given intravenously enabled me to pass a tube into the trachea after which anaesthesia was maintained with a mixture of nitrous oxide and oxygen supplemented occasionally with trichloroethylene—halothane had yet to be marketed.

The operation proceeded uneventfully and although the surgeon had been wildly optimistic in his assessment of the duration of the procedure, he did ultimately indicate accurately when the operation would finish. Accordingly, when I withdrew the endotracheal tube, Jacko sat up almost immediately albeit somewhat confused. Simultaneously, his master reappeared carrying four large glasses, one small glass, and a bottle of whisky which was emptied into the glasses and handed round the assembled company. Jacko was given the small glass and its contents were immediately swallowed in one gulp. For about 30 seconds he sat still on the end of the operating table and then took a flying leap at the chandelier, which he missed by about two feet, and fell unconscious on the floor. At this stage I hastily pointed out that the patient had been conscious, swallowing, and mobile when he left my hands.

Almost 50 years on, I still clearly remember, firstly, the advantages of intraperitoneal thiopentone in a recalcitrant patient; secondly, the first intensive care unit I had ever seen either in hospital or in a private home; and thirdly, the sight of a professor of surgery sitting quietly all evening with a patient, the first and only time I have ever had that experience. In addition, my conviction that good whisky provides excellent postoperative sedation and analgesia was reinforced. Jacko slept all afternoon and evening and was apparently none the worse when I visited him the next day. Whether or not tolerance was a factor, I will never know. What I do know is that Jacko lived to a ripe old age, teeth or no teeth.—J P PAYNE, *emeritus professor of anaesthesia, London*

We welcome filler articles of up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk.