oscopy, false positive results are not uncommon with double contrast radiology, and up to 29% of polyps may be missed, although this figure falls to under 10% for polyps over 1 cm in diameter.¹³ The presence of diverticular disease makes the diagnosis of associated lesions more difficult,^{10,14} some authorities recommending that colonoscopy should be performed in all patients with symptomatic diverticular disease.¹⁴ Another important group comprises patients with overt or occult rectal bleeding with no cause found on rigid sigmoidoscopy or barium examination: lesions may be shown in 27-81% of such cases, including tumours in 3-11%.^{4,15}

Accurate cost comparisons in our hospitals are difficult to make, but the greater necessity for colonoscopy after an initial barium study would suggest that a policy of performing endoscopy initially is likely to work out less costly. For this reason and for reasons of accuracy and expediency and to avoid multiple investigations total colonoscopy may be the preferred initial investigation for those with suspected colonic disease. We have shown that radiology is not a prerequisite for adequate colonoscopic examination.

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SHORT REPORTS

Apolipoprotein B polymorphism and altered apolipoprotein B and low density lipoprotein cholesterol concentrations in Finnish children

With a monoclonal antibody we studied the relation between genetic variation in apolipoprotein B and serum concentrations of lipoprotein cholesterol and apolipoprotein B. The polymorphism detected by this antibody (MB-19) is identical to the Ag(c/g) polymorphism, one of five allelic variations described by Büttler *et al* in the review by Tikkanen.¹ Antibody MB-19 distinguishes among three apolipoprotein B allotypes (immunophenotypes) encoded by the two allelic genes apoB(c) and apoB(g). Because of codominant transmission genotypes may be inferred from allotypes.

We report an association between apoB(c) and serum concentrations of apolipoprotein B and low density lipoprotein cholesterol in Finnish children.

Subjects, methods, and results

Serum samples were obtained from 513 children who participated in an ongoing multicentre study on atherosclerosis precursors.² The children were randomly selected from major cities and rural areas representing different parts of the country. Concentrations of serum lipids and low density lipoprotein and high density lipoprotein cholesterol and body mass index (kg/m²) were determined² at the beginning of the study (1980), when the children were 9 years old. Serum apolipoprotein B concentrations were determined and allotypes assayed¹ in frozen serum that had been stored at -20°C for about five years. To evaluate the possible effects of storage on serum apolipoprotein B we correlated the present serum

apolipoprotein B concentrations with those of the total and low density lipoprotein cholesterol obtained in 1980. Apolipoprotein B concentrations correlated well with the total cholesterol ($r=0.757$, $p<0.001$) and low density lipoprotein cholesterol concentrations ($r=0.809$, $p<0.001$), which supports the hypothesis that the apolipoprotein B had remained intact. Two tailed Bonferroni adjusted *t* tests were used for multiple comparisons between genotypes with standard BMDP software (University of California).

The table shows the subjects grouped according to the presence or absence of the apoB(c) allele. Children who had at least one apoB(c) allele (genotypes apoB(cc) or apoB(cg)) showed, as a group, higher serum concentrations of apolipoprotein B and low density lipoprotein cholesterol and lower high density lipoprotein/total cholesterol ratios than the group who did not have an apoB(c) allele (genotype apoB(gg)). Analysis by apolipoprotein B genotype showed that 8% of the children belonged to the apoB(cc) group, 35% to the apoB(cg) group, and 57% to the apoB(gg) group. The apoB(cg) children had higher serum concentrations of apolipoprotein B (+7.9%, $p=0.007$) and low density lipoprotein cholesterol (+5.4%, $p=0.029$) and lower high density lipoprotein/total cholesterol ratios (-6.1%, $p=0.006$) than children who had the apoB(gg) genotype. Children who had a double dose of the apoB(c) allele, however, did not differ appreciably from those who had a single dose.

Comment

Analysis of restriction fragment length polymorphisms shows variation at the gene locus that may or may not be expressed as alterations in protein structure. Conversely, monoclonal antibodies detect allotypic differences in polymorphic proteins and are suited for monitoring expressed genetic variations. Our study was designed to show possible relations between apolipoprotein B allotype and serum lipoprotein and apolipoprotein B concentrations. Assuming that genetic variation causing alteration in these concentrations could be more evident before hormonal and environmental factors had their full impact, we studied 9 year old children.

We showed significant increases in serum apolipoprotein B and low density lipoprotein cholesterol concentrations in children who had at least one apoB(c) allele compared with children who had none. This receives support from a study that compared the XbaI gene polymorphism with the apoB c/g polymorphism, which showed a strong association between the apoB(c) allele and the smaller molecular weight XbaI allele in Finnish subjects (Dunning *et al*, unpublished). The smaller molecular weight XbaI allele is reportedly associated with an increased serum concentration of apolipoprotein B.³ An explanation for the lack of a gene dosage effect for the apoB(c) allele is not evident from the present results. As suggested for apolipoprotein B gene polymorphisms,⁴ mapping of additional variations at the gene locus (haplotype analysis) may be necessary to clarify such apparent inconsistencies. Studies along these lines are in progress with antibodies specific for other allotypes.

The present report links apolipoprotein B allotype with apolipoprotein B concentration and differs from another study that used antibody MB-19, which showed no such association in adults in southern California.⁵ Subtle

Factors related to atherosclerosis in 9 year old children according to presence or absence of apoB(c) allele. Values given are means (SD)

	Apolipoprotein B genotype		Significance
	apoB(cc) or apoB(cg) n=223	apoB(gg) n=290	
Cholesterol (mmol/l)	5.37 (0.79)	5.25 (0.89)	$p=0.11$
Triglyceride (mmol/l)	0.73 (0.29)	0.70 (0.29)	$p=0.27$
Cholesterol:			
Low density lipoprotein (mmol/l)	3.49 (0.72)	3.34 (0.80)	$p=0.029$
High density lipoprotein (mmol/l)	1.55 (0.27)	1.59 (0.33)	$p=0.12$
High density lipoprotein/total	0.29 (0.06)	0.31 (0.06)	$p=0.006$
Apolipoprotein B (mg/100 ml)	93.4 (21.2)	88.3 (20.8)	$p=0.007$
Body mass index (kg/m ²)	16.5 (2.2)	16.7 (2.5)	$p=0.35$

genetic associations may be masked by ethnic heterogeneity and, with age, by the increasing impact of hormonal and environmental factors.

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Transporting critically ill patients by ambulance: audit by sickness scoring

Patients who are critically ill may be safely transported by specialist teams¹ but no prospective studies to investigate the efficacy of transporting such patients by ordinary ambulances attended by junior doctors have been reported. Such studies are important because most critically ill patients are probably transported in this way. Financial constraints may result in increasing use of ordinary ambulances to transfer such patients to centralised units. We performed an audit of non-specialist transport within a district general hospital group in which a sickness score was used to control for severity of illness.

Patients, methods, and results

Vascular and general surgery, coronary care, and most medical specialties are sited within a five mile radius of this intensive care unit; patients are therefore commonly transferred to the unit by ambulances, the journey taking a maximum of 30 minutes. The unit accepts all patients except those with uncomplicated head injuries.

We studied 50 consecutive patients transferred to the unit. Severity of illness was assessed with a sickness score,¹ which is a modification of the acute physiology and chronic health evaluation (APACHE II) score.² The sickness score was calculated from data collected immediately before and after transport. Arterial blood samples for blood gas analysis were taken before transport, packed in ice, and analysed together with a sample drawn on arrival. Patients were not monitored during the journey. Controlled ventilation was provided with an Ambu bag; patients who had not been intubated received a controlled supply of oxygen from MC facemasks. The partial pressure of inspired oxygen was measured with an oxygen meter. Complications occurring during transfer and the seniority and specialty of the medical attendant were noted. Survival was taken as discharge home.

Of the 50 patients, 31 had had operations and 19 had medical conditions (repair of an aortic aneurysm 13; acute renal failure, 12; sepsis, seven; cardiac arrest, five; and respiratory problems, 13). Seven patients, three of whom had had operations, developed eight serious complications during transfer: obstruction of an endotracheal tube, respiratory arrest on arrival at the hospital, accidental disconnection of arterial and central venous cannulas, withdrawal of crucial inotropic and bronchodilator infusions (two cases), and unrecordable blood pressure on arrival (three). Six of the seven patients were attended by junior staff (registrar grade or below) who were not anaesthetists ($\chi^2=10.79$, $p<0.005$).

The table shows the numbers of patients and their mean sickness scores before and after transfer. The difference in scores between the survivors and non-survivors was highly significant ($p<0.0001$). The mean score for non-survivors showed a small increase after transport, though this was not significant. Patients who suffered complications and those accompanied by junior staff or staff other than anaesthetists tended to have higher scores, but the differences were not significant.

Sickness scores (and 95% confidence intervals) before and after transport categorised by outcome, grade and specialty of attending staff, and complications

	No of patients	Mean sickness score	
		Before transport	After transport
Survivors	34	10.2 (8.9 to 12.0)	10.4 (9.1 to 12.2)
Non-survivors	16	19.0 (16.3 to 21.6)	20.6 (17.7 to 23.4)
Senior staff	27	12.0 (9.7 to 14.3)	12.5 (10.3 to 14.7)
Junior staff	23	14.1 (11.3 to 16.9)	15.0 (11.8 to 18.2)
Anaesthetists	34	12.4 (10.5 to 14.4)	13.0 (11.0 to 15.0)
Other	16	14.1 (10.4 to 17.9)	15.1 (10.8 to 19.3)
Complications	7	15.2 (10.1 to 20.4)	16.5 (10.2 to 22.9)
No complications	43	12.6 (10.7 to 14.5)	13.2 (11.2 to 15.2)

Comment

This study showed that life threatening complications may occur in critically ill patients when conventional ambulances are used for transport. Complications were more common in patients attended by junior doctors and doctors other than anaesthetists and were not due to more severe illness among these patients. The training in resuscitation received by anaesthetists may be an advantage in caring for patients when monitoring is not available. The complications were not the direct cause of death in any patient, probably because of the short journey; longer transport times might have resulted in a significant increase in sickness scores.

Because severity of illness was controlled for, these results suggest that inexperience in the management of patients who are critically ill is the dominant factor in the development of complications during transfer, confirming earlier work.³ The results support the need for improved training in resuscitation⁴ and suggest that blood pressure should be monitored during transfer.

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Is altered cardiac sensation responsible for chest pain in patients with normal coronary arteries? Clinical observation during cardiac catheterisation

Most patients with chest pain characterised as angina pectoris have obstructive atheromatous disease of the coronary arteries. In a few patients with angina pectoris exercise testing indicates abnormalities but coronary angiograms are normal.¹ Various theories have been proposed to explain these findings, termed syndrome X, and which include abnormalities of coronary reserve or myocardial metabolism and abnormal histological appearances.^{2,4} During routine cardiac catheterisation we observed that patients with syndrome X were unusually sensitive to intracardiac instrumentation. We report the findings of a preliminary study.

Patients, methods, and results

We studied seven patients with syndrome X (exertional chest pain associated with ST segment depression of more than 1 mm during exercise and normal coronary arteries); four patients with typical angina but negative results on exercise testing and normal coronary arteries; seven patients with atherosclerotic coronary artery disease; and nine patients with mitral valve disease.