Table 1. Distribution of Total Cumulative Cups of Regular, Decaffeinated, and All Types of Coffee Consumed by Cases and Controls and Estimates of Odds Ratios.*

Category	Total Coffee Consumption (Cups)					Total Coffee Drinkers
Regular Coffee						
	0	<20,000	20,000–39,999	40,000–59,999	≥60,000	
Cases (no.)	17	41	63	32	18	154
Controls (no.)	45	88	105	51	28	272
Adjusted odds ratio	1.0	1.2	1.6	1.7	1.7	1.5
95% confidence interval	—	0.5–2.4	0.8–3.1	0.8–3.5	0.8–4.0	0.8–2.7
Decaffeinated Coffee						
	0	<2000	2000–3999	4000–5999	≥6000	
Cases (no.)	160	1	0	0	10	11
Controls (no.)	291	1	4	0	21	26
Adjusted odds ratio	1.0	1.8	—	—	0.9	0.8
95% confidence interval	—	0.1–29.8	—	—	0.4–1.9	0.4–1.6
All Coffee						
	0	<20,000	20,000–39,999	40,000–59,999	≥60,000	
Cases (no.)	11	42	66	32	20	160
Controls (no.)	33	90	111	54	29	284
Adjusted odds ratio	1.0	1.4	1.8	1.8	2.1	1.7
95% confidence interval	—	0.5–3.1	0.9–3.8	0.8–4.1	0.9–5.2	0.8–3.5

*Odds ratios were adjusted for age (in decades) and sex.

of regular coffee and of coffee of any type yielded significant results ($P<0.05$).

Such a large effect of coffee consumption on pancreatic cancer was not found again after 1981. Despite the fact that most studies were conducted with hospital controls, published findings do not completely exonerate coffee.

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The above letter was referred to the authors of the article in question, who offer the following reply:

To the Editor: Five reports (with substantial series of cases) on the relation between coffee consumption and the risk of pancreatic cancer have been published since 1981. Overall, the existence of a strong relation is not supported by this body of data, although Dr. Clavel and his colleagues are correct in asserting that "published findings do not completely exonerate coffee." Wynder et al.[1] and Kinlen and McPherson[2] found no association. Gold et al. found an exposure–response relation in women but not in men.[3] Mack et al., on the other hand, found an exposure–response relation only in men.[4] Our own second study showed marginally significant increases in risk for both sexes but only in the heaviest coffee-consumption category.[5] It would be of interest to see the data of Clavel et al. separated according to the sex of the subjects.

One wonders what effect the widespread reporting of the strongly positive association noted in our 1981 paper[6] may have had on subjects' reporting of consumption. However, for most of the studies described above, interviews were completed before that episode.

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LIVER TRANSPLANTATION IN OLDER PATIENTS

To the Editor: The impact of liver transplantation on public health policy has not been delineated, partly because of uncertainty about the upper age limit for candidacy. We have compared the outlook in 363 adult recipients of livers who were less than 50 years old (mean +SD, 35.8+8.5) and 92 recipients who were 50 to 77 years old (mean +SD, 55.7+4.8). Cyclosporine and steroids[1,2] were used for immunosuppression, and monoclonal antilymphocyte globulin[3] and azathioprine were added as needed. The techniques of liver transplantation[4] were not influenced by the age of the patient. The indications for transplantation are shown in Figure 1. In 1980, none of the recipients was as old as 50 years, but by 1985 and 1986, the incidence had increased to 22.7 and 31.5 percent. Seventy (19.3 percent) of the 363 younger patients underwent retransplantation at some time, as compared with 25 (27.2 percent) of the 92 older recipients.

The actuarial patient survival was slightly more favorable among the younger patients, but the differences were not statistically significant over the first five years (Fig. 2). The rate of rehabilitation was the same. Fifty of the 61 living patients in the older age group have returned to full domestic function, full employment, or freedom from medical care during retirement except for routine outpatient follow-up. All 11 of those not yet rehabilitated had their transplantation in the past six months.

The effect of relaxing the age criteria on the demand for liver transplantation will be considerable, but fears about the cost of such

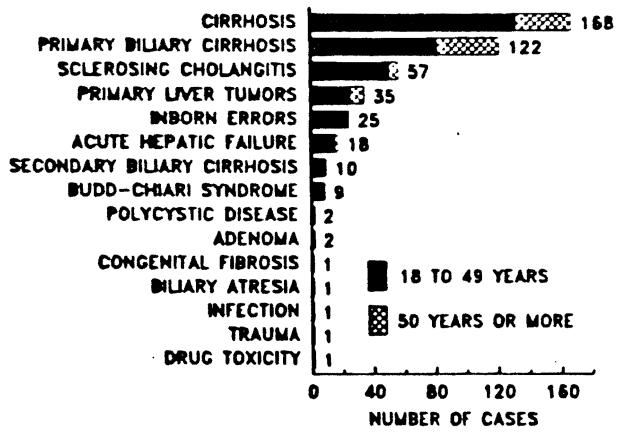

Figure 1. Diseases for Which Liver Transplantation Was Performed in Adults in 1980 to 1986.

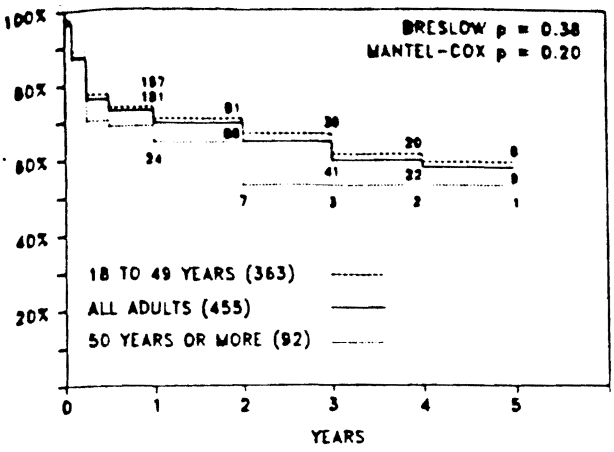

Figure 2. Actuarial Survival of Adult Patients under and over 50 Years of Age.

care may be unfounded. Patients with the liver diseases for which elderly persons have received liver transplantation have required repeated and costly hospitalizations while slowly dying. Such expenditures can be diverted in properly selected cases to a resolution of the problem by transplantation if enough organs can be found. Questions of graft allocation and the influence of age on recipients' candidacies could be made moot if the supply of organs were used more efficiently. Makowka et al.[5] have shown that the criteria for liver donor status have been made unreasonably rigid, probably causing a waste of organs. Popper[6] has pointed out that the liver is the only organ not subject to senescence in aging potential donors. It may be that a choice between the old recipient and the young one will not have to be made.

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ATRIAL NATRIURETIC PEPTIDE AND ATRIAL PRESSURE

To the Editor: Raine and his colleagues (Aug. 28 issue)[1] conclude that the release of atrial natriuretic peptides into the bloodstream is at least partly regulated by right and left atrial pressures. Their data and conclusions are consistent with data obtained from more specific experiments in which the right and left atria of conscious dogs were stretched independently by partial obstruction of the mitral or the tricuspid valve.[2] Distention of either atrium produced an increase in the concentration of atrial peptides in plasma, and circulating atrial peptide levels reverted to control values shortly after atrial pressure was allowed to return to normal. The average increase in the plasma concentration of atrial peptides in response to an elevation of pressure in either atrium was essentially identical (10.2 pmol per liter and 11.0 pmol per liter for each 1-mm Hg increase in left and right atrial pressure, respectively). These values in laboratory animals are quite close to the increase in atrial peptides (14 pmol per liter) calculated to occur with each 1-mm Hg increase in right atrial pressure in the patients described by Raine et al.

The important question regarding whether the rise in the concentration of atrial peptides in plasma has any appreciable effect on sodium balance in patients with cardiac disorders remains unanswered.[3,4] The available data suggest that a persistent increase in circulating atrial peptide levels for hours to days, even in normal laboratory animals and human subjects, does not produce a persistent natriuresis.[5,6] Consequently, the lack of any apparent natriuretic effect of the elevated plasma levels of atrial peptides in patients with heart disease is hardly surprising. Indeed, the possibility that atrial peptides are important regulators of sodium excretion under normal conditions remains to be established.[3,4] Although pharmacologic doses of the atrial peptide consistently elicit a natriuretic response, such consistency is not apparent from the various studies in which smaller (presumably physiologic) increases in the concentration of atrial peptides in plasma were produced. On the other hand, experiments performed in our own laboratory indicate that the ability of atrial peptides to reduce cardiac filling pressure is consistent and dose-dependent. The decrease in cardiac filling pressure may be a consequence of an increase in venous compliance initiated by a sodium nitroprusside–like action of the atrial peptides[7] or by a rapid reduction of intravascular volume elicited by an action of those peptides on systemic capillaries.[8] The observation (by at least five separate groups of investigators) that atrial peptides reduce atrial pressures, coupled with the evidence that an increase in atrial pressure causes the release of atrial peptides, has led us to speculate that atrial peptides may serve as part of a negative-feedback system that allows the heart to influence its own filling pressure by means of the release of atrial peptides.[5]

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The above letter was referred to the authors of the article in question, who offer the following reply:

To the Editor: We are grateful to Dr. Goetz for his comments and agree with him that the steady-state relation between atrial pressure and plasma atrial peptide concentration defined in our study remains when atrial pressure is increased acutely in conscious dogs[1] or during increases or decreases in atrial pressure produced in humans by exercise or decreased venous return, respectively.[2] The suggestion by Dr. Goetz that this tightly regulated release of peptide causes a negative-feedback reduction of atrial filling pressure is appealing, and is consistent with some[3] — but not all[4] — published data.

There is evidence that atrial peptides may act as potent vasodilators — at least in the arterial system. We have found that when human alpha-atrial natriuretic peptide is infused intraarterially in the forearm in humans, it has 60 percent of the vasodilating ac-