tration rate and renal plasma flow are still normal or even supranormal. Small increases of urinary albumin excretion with normal renal function are not known to be associated with "renal hypertension" in any other renal disease.

Yours sincerely,
G. C. Viberti, R. Trevisan and R. Nosadini

References

1. Nørgaard K, Feldt-Rasmussen B, Borch-Johnsen K, Saelan H, Deckert T (1990) Prevalence of hypertension in Type 1 (insulin-dependent) diabetes mellitus. Diabetologia 33: 407–410
2. Parving HH, Mogensen CE, Jensen HAE, Evrin PE (1974) Increased urinary albumin-excretion rate in benign essential hypertension. Lancet I: 1190–1192
3. Losito A, Fortunati F, Zampi I, Del Favero A (1988) Impaired renal functional reserve and albuminuria in essential hypertension. Br Med J 296: 1562–1564
4. Gosling P, Beevers DG (1989) Urinary albumin excretion and blood pressure in the general population. Clin Sci 76: 39–42
5. Leinonen H, Matikainen E, Juntunen J (1982) Permeability and morphology of skeletal muscle capillaries in Type 1 (insulin-dependent) diabetes mellitus. Diabetologia 22: 158–162
6. Bollinger A, Frey J, Jager K, Funer J, Seglias J, Siegenthalen W (1982) Patterns of diffusion through skin capillaries in patients with long-term diabetes. N Engl J Med 307: 1305–1310
7. Trevisan R, Fioretto P, Semplicini A, Opocher G, Mantero F, Rocco S, Remuzzi A, Morocutti A, Zanette G, Donadon V, Perico N, Giorato C, Nosadini R (1990) Role of insulin and atrial natriuretic peptide in sodium retention in insulin-treated IDDM patients during isotonic volume expansion. Diabetes 39: 289–298
8. O'Hare JA, Ferris JB, Brady D, Towmey B, O'Sullivan DJ (1985) Exchangeable sodium and renin in hypertensive diabetic patients with and without nephropathy. Hypertension [Suppl 11] 7: 43–48

Prof. G. C. Viberti
Unit for Metabolic Medicine
UMDS, Guy's Hospital,
London SE1 9RT
UK

Prevalence of hypertension in Type 1 (insulin-dependent) diabetes mellitus

Dear Sir,

Nørgaard et al. [1] reported the prevalence of hypertension in Type 1 (insulin-dependent) diabetic patients and showed that they had a similar prevalence of essential hypertension to that of a normal Danish community. It is possible that blood viscosity could be reason for the similar prevalences.

Since the original report of increased blood viscosity in diabetic patients by Skovborg et al. [2] others, for example [3, 4] have confirmed and extended that observation. But, blood viscosity is also increased in non-diabetic subjects with essential hypertension [5] and in others with high blood pressure [6].

Nørgaard et al. [1] claimed that the similarity of the prevalences of essential hypertension in both non-diabetic subjects and Type 1 diabetic patients "... supports that hypertension is very unlikely to be the cause of diabetic nephropathy." While it is likely that this conclusion is correct, the authors need to examine the possibility that both proteinuria (as an indicator of nephropathy) and raised blood pressure (as an indicator of increased peripheral resistance) could be the consequences of increased blood viscosity.

The authors point out that "... the hypertension of diabetic nephropathy seems to appear *after* the onset of microalbuminuria." But this would be expected if marginally hyperviscous blood was involved, simply because of the haemoconcentrating effects of glomerular filtration. Thus, when only slightly hyperviscous blood has undergone glomerular filtration it will be overtly hyperviscous in the post-glomerular vessels. Hyperviscous blood will increase the resistance to flow in the peritubular plexus and the reduced rate of blood flow will impair the absorptive function of tubular epithelial cells. Direct correlations between blood viscosity and proteinuria have been reported [7, 8] indicating that urinary protein content (whether microalbuminuria or macroproteinuria) depends on the degree of hyperviscosity and therefore on the corresponding rise in intraglomerular pressure.

The finding that essential hypertension had a similar prevalence in both diabetic patients and non-diabetic subjects raises the possibility that dietary intervention could be valuable in the treatment of diabetes. Six weeks on an ovolacto-vegetarian diet lowered blood pressure in healthy normotensive subjects [9] and in subjects with mild hypertension [10]. An important corollary to those studies is that vegetarians have low blood viscosity [11], low blood pressure [12, 13] and a reduced cardiovascular risk [14].

Although this information is readily available and despite the lack of understanding of the cause of microalbuminuria another expert, without reference to the haemorheological problems of diabetic patients, has concluded that "microalbuminuria is the most simple and sensitive parameter" for predicting diabetic nephropathy [15]. Surely it is time for diabetologists to recognise that presently disregarded but significant scientific information obtained from non-diabetic studies could be of value in understanding the diabetic state.

Yours sincerely
L. O. Simpson

References

1. Nørgaard K, Feldt-Rasmussen B, Borch-Johnsen K, Saelan H, Deckert T (1990) Prevalence of hypertension in Type 1 (insulin-dependent) diabetes mellitus. Diabetologia 33: 407–410
2. Skovborg F, Nielsen AaV, Schlichtkrull J, Ditzel J (1966) Blood viscosity in diabetic patients. Lancet I: 129–131
3. Dintenfass L (1979) Haemorrheology of diabetes mellitus. Adv Microcirc 8: 14–36
4. Caimai G (1983) Blood viscosity and erythrocyte filterability: their evaluation in diabetes mellitus. Horm Metab Res 15: 467–470
5. Letcher RL, Chien S, Pickering TG, Laragh JH (1983) Elevated blood viscosity in patients with borderline essential hypertension. Hypertension 5: 757–762
6. Harris I, McLoughlin G (1930) The viscosity of the blood in high blood pressure. Quart J Med 23: 451–464
7. Solerte SB, Fioravanti M (1987) Hemodynamic alterations in long-term insulin-dependent diabetic patients with overt nephropathy: role of blood hyperviscosity and plasma protein changes. Clin Nephrol 28: 138–143
8. Gordge MP, Faint RW, Rylance PB, Neild GH (1988) Abnormal blood rheology in progressive renal failure: a factor in non-immune glomerular injury? Nephrol Dial Transpl 3: 257–262
9. Rouse IL, Beilin LJ, Armstrong BK, Vandongen R (1983) Blood-pressure-lowering effect of a vegetarian diet: controlled trial in normotensive subjects. Lancet I: 5–10
10. Margetts BM, Beilin LJ, Vandongen R, Armstrong BK (1986) Vegetarian diet in mild hypertension: a randomised controlled trial: Br Med J 293: 1468–1471
11. Ernst E, Matrai A, Pietsch L (1987) Vegetarian diet in mild hypertension. Br Med J 294: 180 (letter)
12. Sacks FM, Rosner B, Kass EH (1974) Blood pressure in vegetarians. Am J Epidemiol 100: 390–368
13. Rouse IL, Armstrong BK, Beilin LJ (1982) Vegetarian diet, lifestyle and blood pressure in two religious populations. Clin Exp Pharmacol Physiol 9: 327–330

14. Rouse IL, Armstrong BK, Beilin LJ, Vandongen R (1984) Vegetarian diet, blood pressure and cardiovascular risk. Aust NZ Med J 14: 439–443
15. Mogensen CE (1990) Prediction of clinical diabetic nephropathy in IDDM. Alternatives to microalbuminuria? Diabetes 39: 761–767

Dr. L. O. Simpson
Department of General Practice
Medical School, University of Otago
Dunedin, New Zealand

Response from the authors

Dear Sir,

We have studied with interest the comments of Dr. Stephenson, Professor Viberti and Dr. Simpson to our paper "Prevalence of hypertension in Type 1 (insulin-dependent) diabetes mellitus [1].

We certainly agree with Dr. Stephenson that a study of the prevalence of hypertension can never prove or disprove the hypothesis that the rise in blood pressure in Type 1 diabetic patients with renal complications is a consequence of and not a cause of nephropathy. The aim of our study was to compare the prevalence of essential hypertension in a Danish background population with the prevalence of essential hypertension in a representative sample of Type 1 diabetic patients. If hypertension contributes to the development of diabetic nephropathy we would have expected a very low prevalence of hypertension in normoalbuminuric diabetic patients. However, our study clearly demonstrated the prevalence of essential hypertension to be more or less the same in the two populations. We admit that essential hypertension is sometimes accompanied by microalbuminuria. It might therefore be difficult in patients with Type 1 diabetes and microalbuminuria to discriminate between patients with essential hypertension and patients with incipient nephropathy [2]. We have discussed these difficulties in our paper and shall not repeat them here. We agree with Dr. Stephenson that the hypothesis of hypertension as a consequence and not a cause of nephropathy can be tested by a prospective study which compares the incidence of nephropathy in a group of normoalbuminuric hypertensive diabetic patients with the incidence of nephropathy in a group of normoalbuminuric normotensive diabetic patients. Such a study would be difficult but our preliminary results indicate the cumulative incidence in normoalbuminuric hypertensive diabetic patients not to be higher than among normoalbuminuric normotensive diabetic patients. Our observations from a prospective study among 200 Type 1 diabetic patients with normoalbuminuria also indicate that the urinary albumin excretion rate clearly increases before blood pressure begins to rise. The observation in our actual paper that patients can suffer from long-standing hypertension without nephropathy strongly suggests together with the former observation that hypertension is not causally related to the onset of nephropathy. This of course does not exclude that blood pressure elevation highly aggravates diabetic nephropathy once it has started [3, 4].

Dr. Viberti believes that our conclusions are based on the incorrect premise that essential hypertension has no effect on urinary protein excretion. Also, we know very well that essential hypertension in non-diabetic subjects is sometimes accompanied by microalbuminuria. However, the diastolic blood pressure is usually >130 mm Hg before microalbuminuria appears (>20 µg/min). Thus, among 44 non-diabetic patients with essential hypertension but diastolic blood pressure ≤ 130 mm Hg only 2–3 had microalbuminuria [2, 5]. It would therefore be interesting to know the diastolic blood pressure in those few patients of Dr. Viberti who developed microalbuminuria after antihypertensive therapy was stopped. In our patients with microalbuminuria not a single patient had blood pressure $>200/100$. We therefore do not believe that the prevalence of renal hypertension has been overestimated. We also find no indication for the assumption that the development of moderate hypertension in Type 1 diabetic patients necessarily leads to increased protein excretion and subsequent renal damage. As described in our paper [1] we have identified 51 patients who despite long-standing Type 1 diabetes and long-standing hypertension had normal urinary albumin excretion rate and serum creatinine. Finally, we suggest that the marginal elevation of blood pressure in diabetic patients with advanced microalbuminuria (70–200 µg/min) might well be associated with renal pathology [6] and might very well contribute to keeping the total glomerular filtration rate in the normal range on its downhill course [7].

Dr. Simpson's idea of increased blood viscosity being the common cause of hypertension and proteinuria deserves to be tested in a prospective long-term study. We have until now restrained from such a study because it is our impression that patients with Type 1 diabetes who develop persistent microalbuminuria (and later nephropathy) do not have higher haematocrit and/or higher plasma fibrinogen compared to those who do not develop nephropathy. We agree with Dr. Simpson that an ovolacto-vegetarian diet is probably beneficial especially in patients with diabetic nephropathy and indeed we recommend it to some patients. Also, we are happy for scientific information from studies in non-diabetic subjects. Without such information our understanding of the pathogenesis of diabetes and its complications would be very poor.

Your sincerely,
K. Nørgaard, K. Borch-Johnsen, B. Feldt-Rasmussen and T. Deckert

References

1. Nørgaard K, Feldt-Rasmussen B, Borch-Johnsen K, Sælan H, Deckert T (1990) Prevalence of hypertension in Type 1 (insulin-dependent) diabetes mellitus. Diabetologia 33: 407–410
2. Christensen CK, Krusell LR, Mogensen CE (1987) Increased blood pressure in diabetes: essential hypertension or diabetic nephropathy? Scan J Clin Lab Invest 47: 363–370
3. Christensen CK, Mogensen CE (1985) The course of incipient diabetic nephropathy: studies of albumin excretion and blood pressure. Diab Med 2: 97–102
4. Parving HH, Andersen AR, Smidt UM, Svendsen PÅ (1983) Early aggressive antihypertensive treatment reduces rate of decline in kidney function in diabetic nephropathy. Lancet I: 1175–1179
5. Parving HH, Jensen HÆ, Mogensen CE, Evrin PE (1974) Increased urinary albumin-excretion rate in benign essential hypertension. Lancet I: 1190–1193
6. Ellis EN, Steffes MW, Goetz FC, Sutherland DER, Mauer SM (1986) Glomerular filtration surface in Type 1 diabetes mellitus. Kidney Int 29: 889–894
7. Hall JE, Guyton AC, Coleman TG, Mizelle HL, Woods LL (1986) Regulation of arterial pressure: role of pressure natriuresis and diuresis. Federation Proc 45: 2897–2903

Dr. K. Nørgaard
Steno Memorial Hospital
Niels Steensenvej 2
DK-2820 Gentofte
Denmark