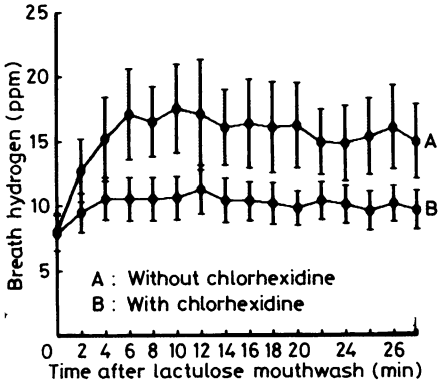

Figure *Breath hydrogen concentration (ppm) before (A) and after (B) chlorhexidine mouthwash.*

brush their teeth and not to smoke for the duration of the test. A measurement of breath hydrogen was then made. They then rinsed their mouths with 20 ml of a 1% lactulose solution for one minute without swallowing it. A further 15 breath hydrogen measurements were made at two minute intervals. The test was then repeated the next day. On one of the two consecutive days the subjects rinsed their mouths with a 1% chlorhexidine solution for one minute before rinsing their mouths with the lactulose solution. Thirteen of the subjects had the chlorhexidine mouthwash on the first day and the other 12 had the mouthwash on the second day.

Multivariate profile analysis was used to show that the mean breath hydrogen concentration profile was significantly ($p < 0.05$) higher in those who had not taken the chlorhexidine mouthwash (figure).

In both groups the time to maximum breath hydrogen concentration varied between two to 30 minutes after the lactulose mouthwash. The maximum mean increase over the fasting breath hydrogen was found to be 14·3 (SEM 4·7, 95% CI) ppm in the group not treated with chlorhexidine and 5·7 (SEM 1·9) ppm in the group treated with chlorhexidine. There was a highly significant difference between the two groups (Student's t test = 3·50 for 48 df, $p = 0.0005$).

In the group treated with chlorhexidine the maximum individual rise in breath hydrogen during the study was 17 ppm; without chlorhexidine the maximum rise was 52 ppm.

Our results confirm the presence of an early hydrogen peak after lactulose mouthwash. Moreover, an early breath hydrogen peak was still observed after oral hygiene with chlorhexidine mouthwash.

It has been proposed that a peak breath hydrogen concentration of 20 ppm before the colonic peak is indicative of small bowel bacterial colonisation.[5] In our study the magnitude of individual results emphasises the possible effect of the early peak on the attempted diagnosis of small bowel bacterial colonisation. We suggest that for making this diagnosis oral hygiene with chlorhexidine is essential and that an increment of 20 ppm of breath hydrogen over the fasting value may be a minimum cut off point.

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Immune complexes in the choroid plexus in systemic hypertension

Immunomorphological investigations were applied to the study of the kidney in patients with systemic hypertension. Various investigators observed, by immunofluorescence microscopy, deposition of immunoglobulins (Ig) and fractions of the complement (C) system in small arteries arterioles, and renal glomeruli in cases of malignant systemic hypertension and, less commonly, in benign cases.[1–3] The choroid plexus is a vascular and epithelial tissue which actively forms cerebral spinal fluid from blood by a process of filtration and secretion. Like the renal glomerulus, its vascular core is composed of capillaries with a fenestrated endothelium.[4] In spite of the structural and functional similarities between the choroid plexus and the renal glomerulus, the existence of similar changes in the choroid plexus in systemic hypertension have not been studied.

We studied choroid plexus specimens obtained from 45 patients at necropsy in the department of pathology of the Federal University Minas Gerais Medical School, in Belo Horizonte. Twenty five of these patients had clinically and histologically diagnosed systemic hypertension (systemic hypertension group), 19 of which had the benign form and six the malignant form, whether preceded by the benign form or not.[5] The remaining 20 patients had died from diseases with no evidence of renal or brain involvement by hypertension or other disease processes. The brain was removed from six to 12 hours after death, and the choroid plexus of the inferior horn of one of the lateral ventricles was taken, embedded in resin, frozen, and stored at −70°C until it was cut with a cyrostat knife. Human Ig (IgA, IgG, IgM) and fractions of C (C3 and Clq) were investigated by the routine direct immunofluorescence technique using monospecific fluoresceinated antisera (Miles Laboratories, Research Products Division, Indiana, USA).

Positive immunofluorescence in the choroid plexus was found in five of the 25 cases from the systemic hypertension group; four cases were associated with benign hypertension and one with malignant hypertension. IgG was more often found than Ig (five cases). C3 and IgA were also present (C3: four cases, IgA: three cases). All the five cases with positive immunofluorescence showed a granular pattern, sparsely distributed in the walls of a few villi of the choroid plexus (figure), resembling that usually seen in the kidney.[1–3] Unfortunately, in this series we did not examine the immunohistology of the kidneys. Histological examination of the choroid plexus showed changes in 12 of the 25 cases from the systemic hypertension group; four of the five cases with positive immunofluorescence showed histopathological changes in the choroid plexus. The most commonly found change was characterised by focal, linear, occasionally nodular, subepithelial deposition of a homogeneous, acidophilic and periodic acid Schiff positive substance,

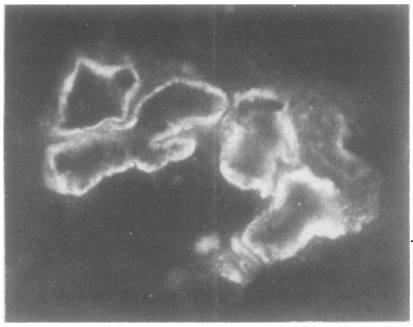

Fig *Deposition of fluoresceinated antisera to IgA in the choroid plexus in a case of systemic hypertension.*

apparently in relation to the epithelial basement membrane, with thickening of this structure. The immunofluorescence in the choroid plexus was negative in all cases of the control group. Histopathological changes of the choroid plexus, with a pattern similar to that of the systemic hypertension group, were found in only two of the control group.

Our results indicate that in about 25% of patients with systemic hypertension the choroid plexus is a site for the deposition of Ig and fractions of C. We suggest that these findings may be secondary to increased vascular permeability produced by hypertension, leading to the passage of plasma and retention of circulating immune complexes in the walls of the choroid plexus, perhaps in a manner analogous to that which occurs in the kidney.[1 3] A question unanswered by this work is the nature of antigens which stimulate the immune complexes formation and lead to their precipitation in the choroid plexus in some cases of systemic hypertension. Further research into the identification of these antigens as well as the demonstration of a pathogenetic association between immune complexes and histopathological changes in the choroid plexus is needed.

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Increased red cell volume distribution width in patients with bone marrow metastases

Marrow infiltration by malignant cells is a common finding in patients with some types of cancer and in patients with Hodgkin's disease and non-Hodgkin's lymphoma. Although in such cases the spread of tumour is usually associated with haematological changes, clinical signs, or radiological findings, marrow metastases can sometimes be found in patients with advanced cancer without any obvious abnormality. For this reason bone marrow biopsy is often carried out as part of the evaluation of patients with cancer.

We report on a haematological abnormality in the peripheral blood that was found in patients with marrow infiltration by malignant cells. As far as we are aware this is the first such study.

The red cell volume distribution width (RDW), which is an expression of the size distribution spread of the erythrocyte population, was measured in 64 patients with bone marrow infiltration by cancer or Hodgkin's disease, in 46 patients with breast and lung cancer, and in 12 patients with Hodgkin's disease who had no evidence of marrow infiltration (figure). The measurement was made on a Coulter S-plus machine. It is computed from the red blood cell histogram and is the coefficient of variation (CV) expressed in per cent of the red blood cell size distribution.

The results are shown in the figure. A gross deviation above the normal limits was observed in over 90% of the patients. The mean value for all three patient groups with marrow infiltration was increased compared with patients with the same type of cancer but without bone marrow metastases; this finding was significant ($p < 0.01$, χ^2).

The precise pathophysiological mechanism of the changes in erythropoiesis is not quite clear. Perhaps anaemia is the expression of quantitative changes which occur in the infiltrated marrow (replacement, fibrosis) while increased RDW is the expression of qualitative disturbances of erythropoiesis.

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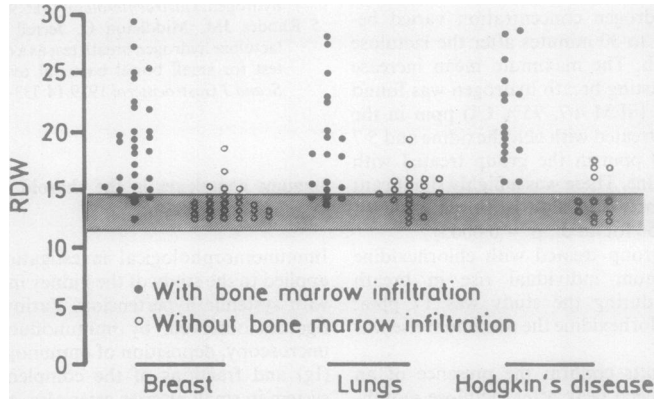

Fig *Measurement of RDW in 122 patients studied.*