hypoglossal nuclei, nuclei ambigui, motor nuclei of the vagi and glosopharyngei. There was also atrophy of the pyramidal tracts in all regions. The most characteristic feature was the peculiar vascular pattern in the right side of the medulla oblongata, pons and inferior part of mesencephalon (fig). All capillary vessels of these regions showed irregular fibrosis and thickening of their walls and slight variations of their lumina. The endothelium was flat and the wall irregularly enlarged with proliferation of collagen fibres. There was a slight loss of reticular neurons and diffuse proliferation of glial cells. No thrombi or occlusive changes or endothelial proliferation were demonstrated. There were no cavernous areas or arteriovenous fistulas. The subarachnoid vessels in the vicinity were normal.

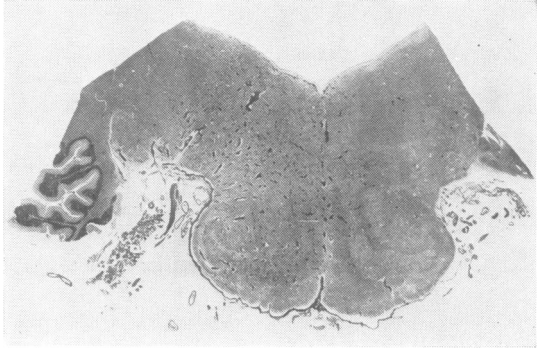

Figure *Cross section of the brainstem showing in the left side the walls of capillaries and small vessels. Van Gieson's Method* × 2·2.

Our case is uncommon because of the presence of abnormal vessels exclusively in the right side of the hindbrain; also this type of vascular abnormality is unusual in the central nervous system. The vascular changes affected especially the capillaries and small vessels, but because of the atypical structure of their walls, it was not possible to decide whether they were arterioles, venules or only capillaries. Genuine telangiectasis, convoluted blood vessels, and cavernous areas were not present, as they are in other form of angiomatosis.[4 5] The most important and characteristic vascular changes in our case was a hyperplasia of collagen fibres with thickening of the wall. Manuelidis[6] described vascular thickening in the cerebral cortex in a case of Sturge-Weber syndrome and interpreted it as "dysgenetic dysorie". This vascular condition is different from the well known vascular maldevelopment or vascular tumour of the hindbrain. However, Horanyi-Hechst[7] described very similar vascular changes in the occipital cortex of a idiotic patient associated with microgyria and demyelination. It is important to note that the vascular proliferation and thickening affected exclusively the right side of the hindbrain, unrelated to any vascular territory. Meningeal vessels in the vicinity are not affected. We think that our case should be interpreted as mesenchymal dysplasia as described by Divry and Van Bogaert[8] and independent of amyotrophic lateral sclerosis. This interpretation is supported by the localisation and the vascular distribution of the vascular lesions. As far as we know, the present case is the first description of this rare type of angiodysplasia of the brainstem.

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Matters arising

Sir: We congratulate Dr Harrison and Dr Andrew (1981;**44**:558) on their lucid exposition and explanation of why hemifacial spasm recurs after facial-hypoglossal anastomoses. This phenomenon has been noted before (Potter JM, personal communication), but their explanation reinforces the belief that the origin of hemifacial spasm lies proximally, rather than distally. However, we cannot agree with the statement that exploration of the facial nerve in the posterior fossa has a "significant morbidity". Indeed, we believe that this is the treatment of choice, although there is debate as to the mechanism by which the spasm occurs in the first place and how it is abolished by the operation.

In 1978[1] we published our experiences treating this condition by facial nerve wrapping. We are in the process of compiling a follow up report on sixteen patients treated since 1976. The results are superior to any other procedure, the morbidity is negligible and the mortality is zero.

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