Summary of a Paper read at the Annual Meeting 1980

NEURORADIOLOGICAL EVALUATION IN PROGRESSIVE POST-TRAUMATIC CYSTIC MYELOPATHY (PPCM)

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The authors review the neuroradiological evaluation methodology and findings in 25 patients with surgically proven progressive post-traumatic cystic myelopathy. As Barnett recorded in his monograph on syringomyelia in 1973, 'Unequivocal evidence exists that the ascending myelopathy occurring as a sequel to traumatic spinal injury is due to progressive cavitation within the central spinal cord.' In the course of following patients with spinal cord injury at Craig Hospital and the Rocky Mountain Regional Spinal Cord Injury Care System, neuroradiologic algorithms were developed to confirm and define cystic myelopathy. The algorithm used in the early 1970s and mid-1970s emphasised the use of myelography to identify an enlargement of the spinal cord, secondary to the presence of a cyst. Myelography was performed using Pantopaque or air and in early cases included in the series direct cyst puncture and positive contrast injection verified the extent and location of the cyst. Subsequently diagnostic cyst puncture was largely abandoned because of potential attendant risk. The recent modified algorithm employs myelography usually from the C-1, 2 lateral approach with metrizamide and subsequent computerised axial tomography of the suspicious level at two to four hours.

In 21 of 25 cases, the neuroradiological findings confirmed the suspected clinical diagnosis. In four of 25 cases the neuroradiological findings were debatable or inconclusive and progressive neurological findings prompted surgery and a cyst was found. Five of the 21 patients had a large cord at contrast myelography which led to diagnostic cyst puncture which was followed by surgery. Seven patients had a large cord demonstrated at contrast myelography and had subsequent surgery and in nine patients metrizamide CT demonstrated the cyst filling with contrast material which was followed by surgery. Pertinent neuroradiological findings included: normal cord size in approximately 50 per cent of these proven cases; partial or complete block and tethered cord was demonstrated in all 25 cases; all cysts originated at or immediately adjacent to the injury level and in the majority extended cranially; computerised tomography without metrizamide did not identify the cyst in the spinal cord but CT with metrizamide at two to four hours demonstrated filling of the cyst in nine patients. The mechanism of cyst fill is unknown but does not appear to be from direct communication with the subarachnoid space, and may be due to transmyel migration of contrast material. Conclusions are that progressive post-traumatic cystic myelopathy is uncommon but potentially a devastating complication in the spinal cord injury patient. The condition must be clinically recognised and radiologically confirmed and defined for surgical treatment which at this institution was cyst-peritoneal shunt procedure. The current algorithm emphasises the use of computerised tomography with metrizamide to identify the cyst, and in the future with improved resolution CT without metrizamide may be successful in identifying these cysts.