HEADACHE OF RENAL ORIGIN

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Headache of renal origin is associated with toxic absorption from an inflamed kidney, it is secondary to an accumulation of waste products due to renal failure, or it may be the result of mechanical changes in circulation following a renal lesion. At some time during the course of disease of the kidney, headache was an outstanding symptom in approximately one-third of our cases.

In acute hemorrhagic Bright's disease, headache commonly accompanies the fever, as is true in the presence of fever from any cause. In this instance, it probably is caused by meningeal irritation which is a result of the toxic products of disturbed metabolic processes. Such headache usually is generalized and dull. In severe cases where marked elevation of blood pressure accompanies the renal lesion, severe throbbing headache may result from increased pressure within the cranium which accompanies the general increase in arterial tension. Such headache may be localized, and it may be increased by effort or a dependent position of the head. Occasionally headache in the presence of acute nephritis is the result of secondary anemia and consequent inanition of the brain, although in the initial stages of renal disease, anemia seldom advances to a stage in which this occurs.

In the active, chronic and terminal phases of hemorrhagic Bright's disease, headache is a common finding and may be one of the early symptoms which induces the patient to consult his physician. In these patients, it may be accompanied by vertigo, anorexia, nocturia, polyuria, edema of the extremities and general weakness. It may be the result of meningeal irritation from the end products of nitrogen metabolism, or it may be related to pressure changes within the cranium as a result of general vascular disturbances. Occasionally, it is the result of cerebral inanition secondary to the marked anemia which frequently is present in these cases. In patients who have chronic nephritis, headaches usually are generalized and lack the localization of the commoner forms of reflex headache. Occasionally in chronic nephritis, it is possible that the retinal inflammation which occurs is in itself a cause of headache of ocular origin.

With the onset of uremia in terminal Bright's disease, headache is a very frequent and often an initial symptom. It may occur in any part of the head—frontal or occipital, or it may be hemicranial, in which case, it simulates migraine. This is especially true since it frequently is accompanied by nausea and vomiting. As a rule, headache in the presence of uremia is not severe in contrast to that observed in the hypertensive encephalopathy to be discussed. Volhard does not regard

headache as a true symptom of uremia, but it has been noted repeatedly that patients who have not had headache previously, do have it as an accompaniment of the abnormal increase of non-protein nitrogen in the blood which is significant of the uremic state.

Headache is a symptom from which almost all patients with essential hypertension suffer at some time, and frequently it is an initial symptom. In fact, it is common to find that patients with essential hypertension have suffered since childhood from headaches which frequently are of the type suggestive of migraine. Evidently such a history indicates some inherent instability of the autonomic nervous system and of vasomotor control which later renders these patients susceptible to essential hypertension. The headaches of hypertensive encephalopathy may be generalized or localized in the occipital or frontal areas; occasionally, they are hemicranial. They may be preceded by visual or auditory aura, and at times they may be accompanied by nausea and vomiting. The headache may vary from a dull area or feeling of increased pressure in the cranium to the most severe type of intense throbbing which is coincident with the heart beat. Janeway describes the typical headache of hypertension as one which awakens the patient prematurely, attains its maximum intensity before breakfast, and disappears during the course of the morning. However in many patients, this is reversed and the maximum pain is attained with the increased mental or physical effort of the day which is associated with consequent effect upon the level of blood pressure.

The reason for the frequent occurrence of headache associated with hypertension is not entirely clear. It is known that the intracranial pressure, as revealed by lumbar puncture, does not vary absolutely with the arterial tension, although it tends to follow in the same direction, and frequently a lumbar puncture relieves much of the headache. There is no necessary parallelism between the height of the blood pressure and the liability to headache. Some patients with severe hypertension never have headaches; therefore, it is apparent that some factor other than mere mechanical elevation of pressure is present. Postmortem findings suggest that the majority of patients with essential hypertension have well-marked cerebral arteriosclerosis, even though they showed no definite evidence of cerebral disease during life, and it therefore is likely that sclerotic vascular changes and disturbances of cerebral or meningeal circulation are responsible for the symptom. The sudden appearance and disappearance of the headache in some cases would suggest that vasoconstriction of the sclerotic vessels may be a factor, possibly by the initiation of some degree of cerebral anemia. Sudden rises in pressure are apt to be accompanied by violent headache. Of course, renal insufficiency in essential hypertension may be accom-

panied by uremic headache, but the vast majority of headaches of patients with essential hypertension are due not to uremia as is indicated by the normal blood findings.

The sequelae of essential hypertension may be the cause of severe headache. The onset of a cerebral hemorrhage may be marked by an uncontrollable localized pain in the head which apparently is the result of meningeal irritation and cerebral damage. Cerebral thrombosis may produce similar symptoms. Occasionally, in hypertensive encephalopathy, edema of the brain develops, and intracranial pressure is increased to such an extent that choked disc appears. In such cases, the clinical picture of violent headache, nausea, vomiting, convulsions and marked elevation of cerebrospinal fluid pressure appears as in any other space-filling lesion of the cranium.

Recognition of the renal origin of the symptom is dependent upon the history of antecedent renal disease or hypertension, urinary examination, renal function and blood chemistry studies. Apart from the use of sedatives and analgesic drugs for symptomatic relief or the judicious use of lumbar punctures in a few cases, treatment must be directed toward the care of the underlying renal condition.