blood (Lomaz). The divergence of opinion, however, about the capillary pressure in hypertension renders further investigation and information necessary before this correlation can be considered. Izquierdo and Cannon's discussion of ''emotional'' polycythaemia in relation to the sympathetic and the medulliadrenal action of the spleen, and Lamson's earlier view that excess of adrenaline in the blood may drive the red cells out of the spleen into the circulation, might suggest that those cases of essential hypertension with polycythaemia have an excess of adrenaline in the blood. The simplest explanation of the association of polycythaemia and essential hypertension is the onset of congestive heart failure.

In conclusion, the clinical significance of essential hypertension is obviously that it is the potential antecedent of cardiovascular and renal complications.

BIBLIOGRAPHY

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Abrami, Santenoise, and Bernal: Presse Méd., 1933, xli, 329.
Allbutt, T. Clifford: British Medical Journal, 1877, i, 157.
Idem: Ibid., 1893, ii, 997.
     Idem: Trans. Hunterian Soc. Lond., 1895-6, p. 38. Idem: Med.-Chir. Trans., 1903, lxxxvi, 323.
                                                    Diseases of the Arteries, including Angina Pectoris, 1915, i,
                               383.
    383.
Idem: Arteriosclerosis: A Summary View, 1925, p. 4.
Allen and Sherrill: Journ. Metabol. Res., 1922, ii, 429.
Ayman, D., and Pratt, J. H.: Arch. Int. Med., 1931, xlvii, 675.
Berger and Fineberg: Ibid., 1929, xliv, 531.
Boas, E. P., and Muíson, I.: Journ. Lab. and Clin. Med., 1923, ix,
    152.
Bordley, J., and Baker, B. M.: Bull. Johns Hopkins Hosp., 1926, xxxvi, 320.
Boyd, W.: Textbook of Pathology, 1932, p. 563.
Broadbent, W. H.: The Pulse, 1890.
Bright, R.: Guy's Hosp. Rep., 1836, i, 340.
Cannon, W. B., and de la Paz, D.: Amer. Journ. Physiol., 1911, xxviii, 64.
Cannon, W. B., and de la Paz, D.: Amer. Journ. Physiol., 1911, xxviii, 64.

Castex, M. R.: Hypertension Artérielle Paroxystique, Paris, 1933, p. 23.

Cohen, M. B., Fineberg, M. H., and Rudolph, J. A.: Amer. Journ. Med. Sci., 1933, clxxxvi, 35.

Cowdry, E. V.: Arteriosclerosis, New York, 1933.

Cushing, H.: Bull. Johns Hopkins Hosp., 19.1, xii, 290.

Idem: Papers Relating to the Pituitary Body, Hypothalamus, and Parasympathetic Nervous System, 1932, 113.

Cutler, O. I.: Arch. Path., 1928, v, 365.

Draper, G.: Disease and the Man, 1930, p. 89.

Evans, G.: British Medical Journal, 1923, i, 549.

Fahr, T.: Deut. Arch. f. klin. Med., 1920, cxxxiv, 366.

Frey, E. K., Kraut, H., and Schultz, F.: Arch. f. exper. Path. u. Pharm., 1930, clviii, 334.

Gley, P., and Kisthinios, N.: Presse Méd., 1929, xxxvii, 1279.

Goldzieher, M. A.: The Adrenals, 1929, p. 234.

Gray, J.: Medical Research Council, Special Report Series, No. 178, 1933.

Gull, W. W., and Sutton, H. G.: Med.-Chir. Trans., 1872, lv, 273.

Huchard, H.: Traité Clinique du Cœur et des Vaisseaux, 1893.

Izquierdo, J. J., and Cannon, W. B.: Amer. Journ. Physiol., 1928, 1xxxiv, 545.

Janeway, T. C.: Amer. Journ. Med. Sci., 1913, cxlv, 625.

Idem: Arch. Int. Med., 1913, xii, 755.

Idem: Bull. Johns Hopkins Hosp., 1915, xxvi, 341.

Johnson, G.: Med.-Chir. Trans., 1868, li, 37.

Josué, O.: Bull. et Mém. Soc. Méd. des Hôp. de Paris, 1904, 3 sér., xxi, 139.

Keith, N. M., Wagener, H. P., and Kernohan, J. W.: Arch. Int.
  Josué, O.: Bull. et Mém. Soc. Méd. des Hôp. de Paris, 1904, 3 sér., xxi, 139.

Keith, N. M., Wagener, H. P., and Kernohan, J. W.: Arch. Int. Med., 1928, xli, 141.

Kerppola, W.: Acta Med. Scand., 1922-3, lvii, 515.

Kylin, E.: Ibid., 1921, lv, 368.

Idem: Klin. Woch., 1925, iv, 806.

Kroelz, C.: Quoted by Grollman, The Cardiac Output of Man in Health and Disease, 1932, p. 233.

Labbé, M., Tinel, J., and Doumer: Bull. et Mém. Soc. Méd. des Hôp. de Paris, 1922, 3 sér., xlvi, 982.

Labbé, M., Violle, P. L., and Azérad, E.: Presse Méd., 1930, xxxviii, 553.

Lamson, P. D.: Journ. Pharm. and Exper. Therap., 1920-1, xvi, 125.
  125.

Langeron, L.: Questions Cliniques d'Actualité, 1932, 3 sér., 131.

Langeron, L., and Lohéac, P.: Presse Méd., 1929, xxxvii, 1153.

Langeron, L., and Delcour: Arch. des Mal. du Cœur, 1928, xxi, 13.

Lenaz, L.: Presse Méd., 1922, xxx, 974.

Lewis, T.: Diseases of the Heart, 1933, p. 227.

Livon, C.: C. R. Soc. de Biol., 1898, 1, 135.

Lohéac, P.: Thèse de Lille, 1928.

Lomaz, L.: Klin. Woch., 1926, v, 1028.

Lucas, W. S.: Arch. Int. Med., 1912, x, 597.

MacWilliam, J. A.: British Medical Journal, 1927, i, 125.

Mahomed, F. A.: Med.-Chir. Trans., 1874, 1vii, 197.

Idem: Gw's Hosb. Reb., 1879, 3 s., xxiv., 363.
                                125.
    Manomed, F. A.: Mea.-Chiv. 17ans., 1874, 191, 197. Idem: Guy's Hosp. Rep., 1879, 3 s., xxiv, 363. Idem: Ibid., 1881, 3 s., xxv, 295. Major, R. H.: Journ. Amer. Med. Assoc., 1924, lxxxiii, 81. Idem: Minnesota Med., 1932, xv, 797. Mosenthal and Short: Amer. Journ. Med. Sci., 1923, clxv, 531. O'Hare, J. P., Osler's Modern Medicine (McCrae), 1927, v, 593.
```

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xxxiv, 631.
Osler, W.: British Medical Journal, 1912, ii, 11.
Patek, A., and Weiss, S.: New England Journ. Med., 1931, ccv,
    Paul, F.: Virchow's Arch., 1931, cclxxxii, 256.
Pfiffner, J. J., and Myers, V. C.: Journ. Biol. Chem., 1930, lxxxvii,
Phitner, J. J., and Myers, V. C.: Journ. Biol. Chem., 1930, 18xxvii, 345.

Pincoffs, M. C.: Trans. Assoc. Amer. Phys., 1929, xliv, 295.
Reid, C.: Quart. Journ. Med., 1925-6, xix, 411.
Ryle, J. A.: Guy's Hosp. Rep., 1927, 1xxvii, 307.
Santenoise, D.: C. R. Soc. de Biol., 1930, civ, 765.
Idem: Progrès Méd., 1932, 1, 2170.
Schröder, K.: Virchow's Arch., 1928, cclxviii, 291.
Starling, E. H.: British Medical Journal, 1925, ii, 1163.
Starling, E. H., and Anrep, G. V.: Proc. Roy. Soc., B, 1925, xcvii, 463.
Tuthill, C. R.: Arch. Path., 1931, xi, 760.
Vaquez, H.: Bull. et Mém. Soc. Méd. des Hôp. de Paris, 1904, 3 sér., xxi, 120.
Idem: Cong. Franç. de Méd., Paris, 1904, p. 338.
Idem: Maladies du Cœur, 1921, p. 501.
Volhard, F., and Fahr, T.: Die Brightsche Nierenkrankheit, Berlin, 1914.
Waldbott, G. L.: Journ. Amer. Med. Assoc., 1920, xciv, 1390.
Weber, F. P.: Polycythaemia, Erythrocytosis and Erythraemia, 1921.
Weiss, S.: New England Journ. Med., 1932, ccvii, 165.
With, W.: Leit Med. 1923, xcvii, 151.
   Weiss, S.: New England Journ. Med., 1932, ccvii, 165.
Weitz, W.: Zeit. f. klin. Med., 1923, xcvi, 151.
Wollheim, E., and Lange, K.: Deut. med. Woch., 1932, lviii, 572.
Wong, K. C.: Chinese Med. Journ., 1928, xlii, 855; repeated in
History of Chinese Medicine, Tientsin Press, 1932, p. 39.
```

Oppenheimer, B. S., and Fishberg, A. M.: Arch. Int. Med., 1924,

FIELDS OF VISION IN CONNEXION WITH INTRACRANIAL LESIONS *

ADAMS A. McCONNELL, F.R.C.S.I.

SURGEON, RICHMOND HOSPITAL, DUBLIN

It is of scientific interest only to determine the site of a lesion when the latter cannot be treated, but to localize it becomes of urgent practical importance when a chance of radical cure exists. It was not so important to make a diagnosis of appendicitis a hundred years ago as it is to-day. To invite a surgeon to join in introducing this subject is, I think, an indication of the impression that something definite may be done for many intracranial lesions, and that a visual defect must be looked for as carefully as one looks for rigidity and tenderness in the right iliac fossa.

Within recent years much experimental work has been done on the anatomy of the visual paths, and it is not out of place to summarize the results as briefly as possible. Impulses from the left visual field cross to the right halves of the retinae. The fibres from the right sides of both retinae come together at the optic chiasma, and pass in the right optic tract to the external geniculate body. It is accepted that this is the only subcortical centre for vision, and that no visual fibres pass, as formerly described, to the pulvinar of the optic thalamus or the superior quadrigeminal body. From the external geniculate body the secondary neurones of the visual path pass in the optic radiation to the striate area of the occipital lobe. The optic radiation or geniculo-calcarine pathway is identical with the external sagittal stratum of the occipital lobe.

Such is the usual arrangement of the visual pathway, and the general conclusion is that the left side of the field of vision has its centre in the right cerebral hemisphere, just as the left hand has its centre in the right side of the brain, the visual impulses crossing in the air, those from the hand crossing in the spinal cord. Gross lesions of the right visual pathway behind the chiasma will give rise to left homonymous hemianopia; a lesion of the chiasma will give rise to bitemporal hemianopia, but further anatomical details are necessary if we are to distinguish between lesions above and below the chiasma, or to deter-

^{*} Read in opening a discussion in the Section of Ophthalmology at the Annual Meeting of the British Medical Association, Dublin.

mine at which point between chiasma and cortex the long pathway has been interrupted.

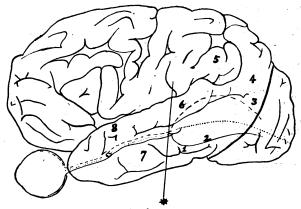
In general, the fibres from the upper half of the retina lie dorsal from the eye to the cortex, from the lower half ventral, while the macular fibres lie between. In the right optic tract the fibres from the upper nasal quadrant of the left retina and from the upper temporal quadrant of the right retina lie together on the inner and upper surface; those from the lower quadrants lie on the outer and under surface, while the macular fibres occupy most of the upper outer segment. When the fibres reach the external geniculate body they preserve a somewhat similar relation, the superior quadrants of the right halves of the retinae being projected upon the internal segment of that body, the inferior quadrants upon the external segment, while the macular fibres end in the intermediate dorsal segment (Brouwer).1 This localization suggests that homologous fibres from the retinae have come together in the external geniculate body, and that homologous fibres run together in the visual radiation.

From the external geniculate body the visual radiation spreads, its dorsal fibres curving upwards and backwards from the internal segment, its ventral fibres downwards and backwards from the external segment, and its intermediate fibres more directly backwards. Experimental work does not seem to reveal any pronounced "temporal loop "directed towards the temporal pole. In Poliak's2 experiments, however, the ventral branch of the visual radiation was not injured, and its complete course was therefore not marked by degeneration. In a coronal section through the right occipital lobe the visual radiation appears like a crescent embracing the lateral aspect of the posterior horn of the lateral ventricle. It consists of three parts: (1) a dorsal horizontal branch from the internal segment of the external geniculate body-which thus carries impulses from the dorsal segments of the right halves of the retinae-and ending in the upper lip of the calcarine fissure; (2) a vertical branch containing the macular fibres and ending in the most posterior part of the striate cortex; its upper half carries the impulses from the dorsal quadrants of the homonymous hemimaculae, while its lower carries those from the ventral; (3) a ventral horizontal branch from the external segment of the external geniculate body, terminating in the lower lip of the calcarine fissure, and conveying impulses from the ventral segments of the right halves of the retinae.

These experimental results, taken in conjunction with the clinical observations of Gordon Holmes³ and others, indicate that there is a fixed geometric projection of the retinac upon the cortex of the striate area. No segment, not even the foveá centralis, has a diffuse or bilateral representation in the cortex.

The striate area is that part of the occipital cortex which forms both lips of the calcarine fissure in its whole extent, and extends for a short distance on to the lateral aspect of the occipital lobe. On the right side the fibres which come via the visual radiation from the most nasal part of the left retina terminate in the extreme anterior part of the fissure; those which come from the right halves of the retinae terminate more posteriorly, and the most posterior area receives the fibres from both homonymous hemimaculae. To state these facts in terms of the visual fields, the peripheral part of the left visual field is projected on the anterior part of the right visual cortex, the central part on the posterior. The floor of the fissure represents the horizontal meridian of the retina.

We may divide the visual pathway in so far as it is commonly involved by intracranial tumours into five sections: (1) the intracranial part of the optic nerves; (2) the optic chiasma; (3) the optic tract; (4) the visual radiation; and (5) the striate area of the occipital lobe. I shall confine my remarks to my personal experiences or perimetry in cases of tumours affecting the optic tract and the optic radiation. These sections of the visual pathway lie particularly in relation to the temporal lobe of the brain. It would simplify description and give a more accurate basis of comparison between series of cases if we divided the temporal lobe into a pre-geniculate and a postgeniculate area by a coronal plane at the anterior portion of the external geniculate body (see Diagram).

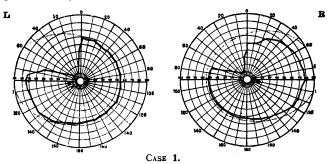


The asterisk indicates plane through anterior end of external geniculate body. Broken line marks fibres from upper half of retina, and continuous line those from lower half. Dots indicate macular fibres; numbers refer to sites of tumours in the cases. Both right- and left-sided tumours are represented as left.

pre-geniculate corresponds almost exactly to the anterior half of the lobe; the post-geniculate to the posterior half. Then if a definite "tract" defect of the visual fields can be established in contradistinction to a "radiation' defect each part of the temporal lobe has its own burglar alarm, provided that the temporal "loop" of the optic radiation described by Meyer does not exist. I have selected the cases with partial visual defects, for obviously a complete homonymous hemianopia might be caused by interruption of the visual pathway anywhere from chiasma to cortex.

Tumours of, or Involving, the Post-geniculate Part of the Temporal Lobe

Case 1.—M.D., aged 13. Admitted May, 1931. Petit mal since age of 4. Two major epileptic attacks at 12. Recent development of double papilloedema. Left upper quadrantic hemianopia. Slight lower facial weakness left side. Operation. Haemangiomatous cyst, size of orange. The mural nodule was situated (1, on Diagram) at anterior inferior portion of cyst.



Case 2.-D. O'B., aged 39. Admitted April, 1933. Attacks of unconsciousness for three months. Occasional dizziness. Gradual deterioration of memory and slips in writing and speech. Sensory aphasia. No headache, no vomiting. Left anosmia. Bilateral papilloedema. Paresis right side of mouth. Right upper quadrantic defect. Operation. with small cyst immediately below the root of the descending horn of the left ventricle (2, on Diagram).

Case 3.-E. M., aged 28. Admitted August, 1932. Two months' history of headache and vomiting; fourteen days diplopia. Double papilloedema. Partial right hemianopia. No other localizing symptom. Operation. Angioblastoma left side of tentorium invaginating occipital lobe up to the point marked 3 in Diagram.

Case 4.—A. O., aged 23. Admitted January, 1921. For four years suffered from sudden "lapses" or loss of memory, ushered in by circles of light before the left eye. For six months attacks of "biliousness." Recent onset of double papilloedema. Left homonymous hemianopia. No other localizing sign. Ventriculography showed occlusion of posterior horn of right ventricle. Operation. Astrocytoma size of plum (4, on Diagram) in medial wall of large cyst, which extended downwards and forwards.

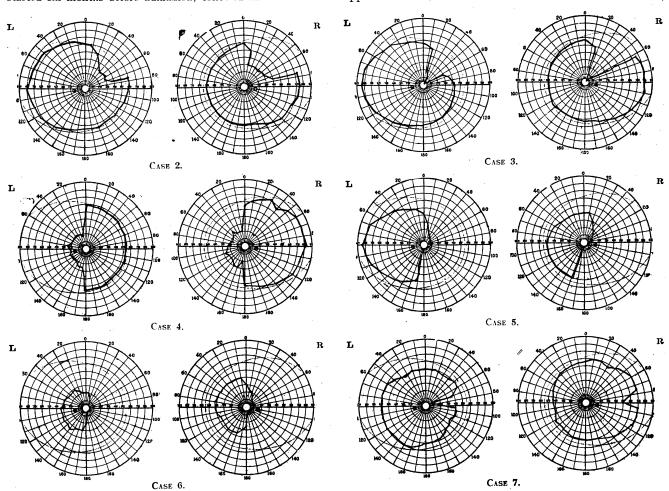
Case 5.-M. L., aged 22. Admitted April, 1926. Headache started six months before admission, followed in two months

occasion there was a suggestion of the defect being homonymous. No other localizing symptom. Operation revealed soft meningioma, size of walnut, indenting the middle of the second temporal convolution on the left side (7, on Diagram).

We have had several tumours involving the anterior part of the temporal lobe, but they either had complete homonymous hemianopia, or perimetry was impossible because of lack of co-operation. One of these (8, on Diagram) had full fields as tested by the confrontation method.

Discussion

I need not discuss these cases in detail; the charts considered with the sites marked in the diagram speak for themselves. I would draw attention to the presence of upper visual defects in the first three cases. The defects



by vomiting in the morning. Sensory aphasia, astereognosis right hand. No motor symptoms. Right homonymous hemianopia congruous incomplete in upper quadrants. Operation. Glioblastoma size of cricket ball in left parietal lobe extending downwards into upper part of left temporal lobe (5, on Diagram).

Case 6.-J. G., aged 16. Admitted July, 1922. Long history of attacks of severe headache and vomiting latterly associated with dimness of vision. Double papilloedema. Right homonymous hemianopia complete in lower quadrant. No other localizing sign. Operation. Large gliomatous cyst in posterior half of left temporal lobe with soft tumour in anterior wall (6, on Diagram).

Tumours of the Pre-geniculate Portion of the Temporal Lobe

Case 7.—A. K., aged 23. Admitted October, 1926. Attacks of giddiness for eight years. Double vision three months, then headaches and vomiting in the morning. Occasional tenderness of scalp over left ear. Slight choking both disks. Sector defect right visual field of left eye; on one were not always strictly congruous, but congruity depends on the condition of the patient as well as on the skill of the perimetrist. In the six post-geniculate cases a diagnosis was made not of a tumour of the temporal lobe, but of a tumour involving the visual radiation, and operation was planned accordingly. In each case the growth was radically removed. One of the cases of glioblastoma (Case 5) survived two years; the rest are alive to-day-one twelve years, one eleven, one two, one nine months, one three months. Case 7 has been perfectly well since her operation six and a half years ago. In each case perimetry gave essential information, and perimetry is justified of her results.

REFERENCES

Brouwer and Zeeman: Brain, 1926, xlix, 1.
 Poliak: "The Main Afferent Fibre Systems of the Cerebral Cortex in Primates, University of California Publications in Anatomy, vol. ii. University of California Press, 1932.
 Holmes: British Medical Journal, 1919, ii, 193; Brain, 1931, liv,

4 Meyer: Trans. Assoc. Amer. Phys., 1907.