surgeons carry out the radical operation on cancers of the breast associated with enlarged supraclavicular glands provided the glands are mobile and not fixed to form a supraclavicular mass. All the evidence points to the danger of any form of surgical treatment in these patients. Operative intervention at this advanced stage can only hasten the patient's end.

Stage IV.—This group comprises cases with distant metastases in bones, viscera, or lungs. These patients are unsuited for any form of active treatment other than hormonal. X rays, however, may help in the relief of pain caused by secondary deposits in bone, particularly when the metastatic process is confined to a solitary deposit—for example, a single vertebra. Although the ultimate prognosis is grave, some of these patients survive for long periods with well-controlled x-ray treatment. The pain of brachial neuritis caused by a supraclavicular mass of glands is similarly relieved.

The Operative Mortality of Radical Mastectomy

The operation of radical mastectomy is not yet obsolete. Until it is, operative mortality and morbidity must be reduced to a minimum. Cade (1948) has collected the figures of 22 British surgeons. There were 11,014 radical mastectomies, with 182 deaths—a mortality of 1.65%. In my series of 170 consecutive radical mastectomies there was no operative death—that is, no patient died within one month of operation or while still in hospital.

Summary and Conclusion

It is more than half a century since Halsted first practised his operation for cancer of the breast. While much has been achieved since then by individual effort it is well to remember that a busy surgeon cannot expect to see many more than 500 patients with cancer of the breast. Spread as they must be over 30 to 40 years, conclusions arising from this experience can come only slowly.

It would seem that we have now reached a stage in the history of this disease when it would be of great advantage to be able to test different combinations of surgery and radiotherapy and hormone methods of treatment upon groups of patients on a scale large enough to make the trials of value. Collaboration between individual surgeons as well as between surgeons and radiotherapy centres would make such investigations possible. Collaboration before treatment and the pooling of results afterwards would provide the volume of material which is necessary if expert independent statistical analysis is to be of value, as it can be, in determining future lines of treatment and in determining them within a reasonable span of time.

Early diagnosis is the key to the problem of treatment of breast cancer.

No significant improvement in treatment need be expected until there is wider knowledge of the basic clinical fact that *there is no outward visible change in the appearance of the breast in early malignant disease.*

REFERENCES

- Adair, F. E. (1943). *J. Amer. med. Ass.*, **121**, 553.
Cade, Sir S. (1948). *Proc. R. Soc. Med.*, **41**, 129.
Gordon-Taylor, G. (1938). *British Medical Journal*, **2**, 1071.
Greenwood, M. (1926). Ministry of Health Report No. 33, p. 6.
Haagensen, C. D., and Stout, A. P. (1942). *Ann. Surg.*, **116**, 801.
Keynes, G. (1937). *British Medical Journal*, **2**, 643.
Kraemer, W. H. (1940). *Surg. Gynec. Obstet.*, **70**, 570.
McWhirter, R. (1948). *Proc. R. Soc. Med.*, **41**, 122.
Marshall, S. F., and Hare, H. F. (1947). *Ann. Surg.*, **125**, 688.
Ministry of Health Report (1926). No. 34, p. 20 (Leeds Series).
Richards, G. E. (1948). *Brit. J. Radiol.*, **21**, 109, 249.
Truscott, B. McN. (1947). *Brit. J. Cancer*, **1**, 129.

PAPILLOEDEMA IN EMPHYSEMA*

BY

THOMAS SIMPSON, M.Sc., M.D., M.R.C.P.

Physician, Chase Farm Hospital, Enfield

Papilloedema rarely occurs in emphysema. Cameron (1933) seems to have been the first to record it. Friedfeld and Fishberg (1934) mention the occurrence of papilloedema in a case of severe right heart failure secondary to pulmonary disease but do not give the cerebrospinal fluid pressure. Meadows (1947) records a case and suggests that the papilloedema was secondary to chronic increased venous pressure and polycythaemia, these in turn being due to the chronic chest condition. Beaumont and Hearn (1948) report a case under the title "Reversible papilloedema due to heart failure" and suggest that the cause is increased pressure in the venous cranial sinuses interfering with the filtration processes of the arachnoid villi.

Although I have studied only three cases, the rarity of the condition prompts me to evaluate the causes of papilloedema in emphysema and to suggest their mechanism.

Case Reports

Case 1.—A motor driver aged 54 was admitted to Redhill County Hospital on April 2, 1946. He had had bronchitis for 28 years and orthopnoea for six months. Seven days before admission oedema had developed. On examination he was seen to be extremely cyanosed, orthopnoeic, and oedematous, and ascites was present. The veins of his face were dilated, his eyes were prominent, the conjunctivae were congested, and he complained of poor vision. Bilateral papilloedema and engorged retinal veins only were seen on fundal examination. The venous pressure was raised to 4 cm. above the sternal angle. Clinical and x-ray examination revealed emphysema, numerous basal rales, and cor pulmonale. The blood pressure was 140/100. The red cells numbered 5,620,000 per c.mm., and the haemoglobin was 120%. Lumbar puncture could not be done owing to his general condition. On April 27 the papilloedema had receded, the retinal veins were less engorged, and he could see better. Clinically the venous pressure was now at the sternal-angle level, the oedema had gone, his vital capacity was 2 litres, and the blood pressure was 135/90. Also the eyes were less prominent and his head was less congested. He was readmitted on Aug. 7, 1946, in semi-coma, with massive oedema and twitching of the arms. The neck veins were not visibly distended and the blood pressure was 180/90. He was discharged on Sept. 25 with no oedema and minimal cyanosis. He was readmitted on Nov. 25, 1946, with gross oedema, gallop rhythm, and a venous pressure of 7 cm. above the sternal angle. He died four days later. The fundi were not commented on in the last two admissions. At necropsy the lungs showed gross emphysema, and although the medium branches of the pulmonary artery showed a well-marked degree of atheroma the smaller branches were normal microscopically. The heart weighed 500 g., the right ventricular wall being 1.5 cm. thick and the left normal. The brain was not examined.

Case 2.—A builder's labourer aged 46 years was admitted to St. Mary's Hospital, Paddington, on Jan. 5, 1948, with congestive failure. Since 1925 he had had winter bronchitis, and in December, 1947, he was orthopnoeic. Nocturnal ankle oedema started in May, 1945, but it troubled him only towards the end of 1947. On examination he was deeply cyanosed, his eyes were prominent, and oedema and ascites were present. Emphysema, numerous basal rales, and cor pulmonale were present. The blood pressure was 140/80. On Jan. 14 the fundi showed bilateral papilloedema, with extreme engorgement and tortuosity of the retinal veins, normal retinal arteries, and one hard white exudate in the right fundus. At this time the venous pressure measured 2 cm. in the neck, the oedema had gone (mersalyl), but the deep cyanosis remained and he was extremely breathless on movement in and out of bed. The red cells

*Paper read before the Middlesex County Medical Society on March 18, 1948.

Amending regulations have been made which relax certain prohibitions in the existing orders affecting the importation and sale of mutton. Caseous lymphadenitis is not regarded as communicable to humans, and the amendments bring the regulations into line with present-day practice in the exporting countries concerned.

numbered 5,700,000 per c.mm. and the haemoglobin was 120%. Renal investigations were normal, and the vital capacity never exceeded 1.5 litres. On Jan. 16 the C.S.F. pressure was 215 mm. of water and the venous pressure 9.8 cm. above the sternal angle. He was discharged on Feb. 13 with the fundal appearances unchanged.

Case 3.—A summary of the clinical notes of this patient is contained in the appendix to the paper by Howarth *et al.* (1947). I quote it in full and add further post-mortem details (Lennox, 1948, personal communication) relevant to my discussion.

"Male, aged 42. Chronic bronchitis and asthma 10 years. Cyanosed, irritable, and forgetful 1 year. Deep cyanosis, oedema, and ascites. Vital capacity 1,100 ml. E.C.G.: S.R. Right axis shift. ST₃ depressed. P₂ 0.25 mv. X-ray: pulmonary arteries enlarged. Heart: moderate enlargement. Blood: R.B.C., 5,300,000 per c.mm.; haemoglobin, 93%. Numerous fundal haemorrhages. Papilloedema developing later. C.S.F. pressure greater than 300 mm. No improvement from oxygen and thiouracil. Death after two months. Post mortem: heart weight 440 g. Left ventricle normal. Lungs: emphysema, slight atheroma of pulmonary artery, intimal thickening of small branches of pulmonary artery."

The surface veins of the brain were engorged to an exceptional degree and the convolutions were slightly flattened. Serial section of the brain showed prominent veins. The authors had found the venous pressure to be 5 cm. above the sternal angle; the blood pressure was 118/69, and the arterial O₂ saturation was 42%.

Discussion

Bilateral papilloedema is generally associated with and attributed to raised C.S.F. pressure in the cranial cavity. Patients with papilloedema usually have a higher C.S.F. pressure than those without, in cerebral tumour (Ayer, 1929), for example, or hypertension (Pickering, 1933-4). Of the three cases reported here two showed a raised C.S.F.

TABLE I

Case	Sex	Vital Capacity in Litres	C.S.F. Pressure in mm. of Water	Venous Pressure in cm. above the Sternal Angle (S.A.)
A	M	1.1	50	1
B	M (died)	Too dyspnoeic	160	S.A. +
C	M (died)	"	100	5
D	F	0.7	70	0
E	M	1.3	45	11
F	F	1.5	80	0
G	M	1.5	135	0

pressure, as did Meadows's (1947) case. In seven cases of emphysema and cor pulmonale without papilloedema I found the C.S.F. pressures to lie between 45 and 160 mm. of water (Table I). It seems probable, therefore, that the papilloedema in emphysema is likewise due to a raised C.S.F. pressure.

In congestive cardiac failure and obstruction of the superior vena cava very high venous pressure may be present without papilloedema. In one case of congestive failure the venous pressure measured 35 cm. above the sternal angle, yet only haziness of the disk margins and congestion of the retinal veins were seen by our ophthalmologist,

Mr. C. B. Goulden. Owing to the oedema the C.S.F. pressure could not be determined. In two other cases of congestive failure the C.S.F. pressure measured 30 and 22.5 cm. and the corresponding venous pressures were 21.5 and 15 cm. In neither case was papilloedema seen.

Pickering (1942) quotes a case of bronchial carcinoma obstructing the superior vena cava in which the venous pressure was raised by 30 cm. and the C.S.F. pressure was 42 cm., yet papilloedema was absent. It is uncertain why these venous and C.S.F. pressures should be unassociated with papilloedema. These cases with high C.S.F. pressure and no papilloedema differ from the cases of emphysema I have described with high C.S.F. pressure and papilloedema in that in the former venous pressure is generally raised in the head and neck, while in the latter the general venous pressure is more or less normal. For instance, the highest venous pressure recorded was 9.8 cm. above the sternal angle (Case 2) and in this case papilloedema was seen when the venous pressure measured only 2 cm. in the neck.

In the three cases of emphysema, therefore, the raised C.S.F. pressure cannot be ascribed to raised venous pressure, for this on measurement was normal or nearly so. A different cause must be found, and this cause may be sought in the essential functional disturbance of emphysema—namely, deficient haemo-respiratory exchange. In emphysema the O₂ saturation of the arterial blood is low and its CO₂ content high (Meakins and Davies, 1925). Both these changes affect the intracranial vessels, that of CO₂ excess being much more conspicuous. In five patients with emphysema and cor pulmonale without papilloedema who died at this hospital the dilatation of the cerebral veins seen at necropsy was a striking feature (see also Case 3).

Wolff and Lennox (1930) observed the pial vessels of cats directly through a trephine hole in the skull. In two experiments the animals inhaled a mixture of room air and approximately 5% CO₂. Although the CO₂ content of the blood was increased by only 2% there was a 17% increase in the size of the observed pial artery. At the same time the C.S.F. pressure, measured in millimetres of Ringer's solution, rose from 40 to 60. However, Dumke and Schmidt (1942-3), in experiments on the cerebral blood flow in 3 to 6 kg. monkeys, cast doubt on the validity of these observations, finding that anoxaemia was a more powerful dilator of the cerebral vessels than CO₂. They, however, draw attention to the fact that none of their animals showed appreciable hyperpnoea during anoxaemia. Had they done so the reduction in the arterial CO₂ tension might have caused constriction of the cerebral vessels, and this sufficient to counteract the dilator effect of anoxaemia. The increase in cerebral blood flow resulting from anoxaemia may therefore have been considerably greater than it would have been under more nearly normal circumstances.

To explain the difference between this work and that of previous workers Schmidt (1944) says that either the response of the cortex is not representative of the great

TABLE II

Case	Age	Diagnosis	C.S.F. Pressure in mm. of Water					Hyperpnoea During the Inhalation
			During Inhalation of CO ₂		After Cessation of Inhalation of CO ₂		After Voluntary Hyperpnoea — 6 Breaths	
			Initial	Final	Max. Rise	Final		
A. B.	43	Fibrositis	75	145 (60)	155 (70)	75 (142)	—	Nil
C. D.	65	Chronic bronchitis ..	85	165 (70)	173 (100)	85 (290)	—	Slight
E. F.	50	Orthostatic albuminuria ..	80	165 (45)	240 (65)	90 (165)	—	+
G. H.	24	Neurosis	78	165 (90)	180 (120)	78 (205)	63	Nil
I. J.	48	Headaches	75	190 (105)	—	—	50	Nil
K. L.	34	Pleural effusion	120	200 (40)	245	120 (95)	75	+

The numbers in parentheses show the time in seconds after the beginning of the experiment that the pressures were recorded.

mass of brain substance or else species differences exist. (Note that in Case 3 serial section of the brain showed prominent veins.) He concludes that at the present time it seems probable that both may be true. If these observations of Dumke and Schmidt (1942-3) are correct, then priority must be given to O₂ lack over CO₂ excess as a cerebral vascular dilator, but this in no way invalidates my conclusions, since both are present in the cases under discussion.

Dixon and Halliburton (1913-14) studied the effects of various substances on the secretion of C.S.F. from a wide cannula placed in the subcerebellar cisterna of dogs. They found that the inhalation of CO₂ increased the secretion markedly. Further, they (1914) studied the effect on the C.S.F. pressure of inhalation of concentrations of CO₂ varying from 3 to 10%. In a few seconds after inhalation of the CO₂ mixture the C.S.F. pressure rose steeply and after stopping the inhalation it fell, but more slowly than it rose. They concluded that of all the conditions which influenced C.S.F. secretion the most important were a deficiency of O₂ and an excess of CO₂ in the blood.

In view of these findings it was decided to observe the effect of inhaling CO₂ on the C.S.F. pressure in human subjects. In six patients not suffering from intracranial or vascular disease the effect on the C.S.F. pressure of inhaling a mixture of 7% CO₂ in 93% O₂ through an oronasal type B.L.B. mask, to which they had been previously accustomed, was observed.

The C.S.F. pressure was recorded after it had become stabilized during natural respiration; CO₂ mixture was then inhaled for a varying period of time and then discontinued. It was found that after cessation of inhalation the C.S.F. pressure continued to rise to a maximum. The C.S.F. pressure and times were recorded at the end of the period of inhalation, at the point of maximum rise, and finally when the C.S.F. pressure had returned to its original level. In three patients the effect of voluntary overbreathing (six breaths) was determined. The results are shown in Table II.

These observations show clearly that a rise of blood CO₂ raises the C.S.F. pressure in the human subject. They thus support the hypothesis that raised C.S.F. pressure in emphysema is due to CO₂ retention in the blood.

A further possibility must be mentioned. Polycythaemia was suggested by Meadows (1947) as a partial explanation of the papilloedema in his case. Sir Charles Symonds supported this suggestion and said that he had seen similar fundal changes in a case of primary polycythaemia. Loman and Dameshek (1944) describe papilloedema in a man aged 41 with a five-year history of polycythaemia. The general venous pressure was but 8 cm. and the C.S.F. pressure 380 mm. The red cells numbered 8.1 to 9 million per c.mm. and the haemoglobin was 136 to 142%. After venesection the papilloedema subsided, leaving a slight degree of optic atrophy in one disk. They were convinced by intra-jugular-pressure studies that venous sinus thrombosis had not occurred. However, none of the cases I have described had a degree of polycythaemia comparable to these cases.

Summary

From a review of the literature and the study of three patients the hypothesis is put forward that papilloedema in emphysema results from a raising of the intracranial pressure.

A rise in general venous pressure does not provide a sufficient explanation, and an intracranial factor is postulated.

It is suggested both from a review of animal experiments and from observation on man that O₂ lack and CO₂ accumulation in the arterial blood in emphysema may produce cerebral vasodilatation adequate to raise the C.S.F. pressure to above the level producing papilloedema.

While polycythaemia may be a contributory factor in certain cases I do not think that it played a part in the cases under discussion.

I studied the cases through the kindness of Dr. G. H. Jennings, Redhill County Hospital, Edgware, Professor Pickering, St. Mary's Hospital, and Dr. R. Harrison, British Postgraduate Medical School. For the necropsy findings in Case 1, I am indebted to Dr. H. Paterson, Redhill County Hospital, Edgware, and for those in Case 3 to Dr. Bernard Lennox, British Postgraduate Medical School. I wish to thank Professors Pickering and McMichael for their help and guidance in the preparation of this paper.

REFERENCES

- Ayer, W. (1929). "The Intracranial Pressure in Health and Disease," Association for Research in Nervous and Mental Diseases, Baltimore, **8**, 189.
 Beaumont, G. E., and Hearn, J. B. (1948). *British Medical Journal*, **1**, 50.
 Cameron, A. J. (1933). *Brit. J. Ophthalmol.*, **17**, 167.
 Dixon, W. E., and Halliburton, W. D. (1913-14). *J. Physiol.*, **47**, 215.
 — (1914). *Ibid.*, **48**, 128.
 Dumke, P. R., and Schmidt, C. F. (1942-3). *Amer. J. Physiol.*, **138**, 421.
 Friedfeld, L., and Fishberg, A. M. (1934). *J. clin. Invest.*, **13**, 495.
 Howarth, S., McMichael, J., and Sharpey-Schafer, E. P. (1947). *Clin. Sci.*, **6**, 187.
 Loman, J., and Dameshek, W. (1944). *Trans. Amer. neur. Ass.*, **84**, 70.
 Meadows, S. P. (1947). *Proc. R. Soc. Med.*, **40**, 555.
 Meakins, J. C., and Davies, H. W. (1925). *Respiratory Function in Disease*, p. 242. Oliver & Boyd.
 Pickering, G. W. (1933-4). *Clin. Sci.*, **1**, 397.
 — (1942). *J. Mt Sinai Hosp.*, **8**, 916.
 Schmidt, C. F. (1944). *Fed. Proc.*, **3**, 131.
 Wolff, H. G., and Lennox, W. G. (1930). *Arch. Neurol. Psychiat.*, **23**, 1097.

OUT-PATIENT ELECTRIC CONVULSION TREATMENT

BY

Sir W. P. MALLINSON, Bt., M.A., B.M., M.R.C.P.
 Honorary Assistant Psychiatrist, St. George's Hospital

Electric convulsion treatment has many applications in psychiatry, and the indications for its use in severe mental illness, particularly in the depressive psychoses, are generally clear and well known. It is proposed in this paper to report upon its use as out-patient treatment for less severe and for earlier cases and to attempt to define the indications for and the limitations of such treatment.

In 1733, when the founders of St. George's Hospital met, they were influenced in their choice of the site at Hyde Park Corner by the fame of the locality as a place "where is good air for cure of consumptions, melancholy, and other infirmities." In the light of this pioneering approach to the treatment of melancholy by physical methods it was perhaps not inappropriate that the hospital should provide one of the first services, if not the first, of out-patient E.C.T. clinics. These clinics have been held regularly since November, 1941, and Table I shows the growth of the service. The clinics were initiated by Dr. William Sargent, and

TABLE I.—Total Number of Patients Treated

1942	1943	1944	1945	1946	1947
37	48	32	30	112	180

during the war years Dr. Sargent and Dr. E. T. O. Slater were in charge. Up to the spring of 1946 there was one session each week; now there are three—on Monday, Wednesday, and Friday. The increased frequency of the sessions not only allows more patients to be treated but enables more intensive treatment to be given where necessary, so that more rapid improvement can be obtained and more seriously ill patients treated.