- Crutchfield, W. G. (1937). *Amer. J. Surg.*, **33**, 592.
 — (1938). *J. Bone Jt. Surg.*, **20**, 696.
 Davis, A. G. (1929). *Ibid.*, **11**, 133.
 — (1938). *Ibid.*, **20**, 429.
 Davis, K. S. (1937). *Radiology*, **29**, 695.
 Dickson, J. A., and Eddy, A. W. (1936). *Surg. Clin. N. Amer.*, **16**, 1001.
 Doub, H. P. (1934). *Radiology*, **23**, 267.
 Drummond, R. (1934). *Guy's Hosp. Rep.*, **84**, 510.
 Dunlop, J. (1937a). *West. J. Surg.*, **45**, 600.
 — (1937b). *Amer. J. Surg.*, **38**, 568.
 Edey, B. T. (1937). *Med. J. Austral.*, **2**, 371.
 Gallie, W. E. (1937). *Ann. Surg.*, **106**, 770.
 Giles, R. G. (1936). *Radiology*, **27**, 182.
 Glissan, D. J. (1937). *Med. J. Austral.*, **2**, 376.
 Gordin, A. E. (1937). *Amer. J. Surg.*, **38**, 374.
 Griffiths, H. E. (1935). *Lancet*, **2**, 439.
 Griswold, R. A. (1936). *J. Bone Jt. Surg.*, **18**, 784.
 Hadaway, H. (1934). *British Medical Journal*, **1**, 986.
 Hatch, E. S. (1936). *New Orleans med. surg. J.*, **88**, 702.
 Hauser, E. (1935). *Illinois med. J.*, **68**, 320.
 Heller, E. P. (1936). *J. Missouri med. Ass.*, **33**, 439.
 Henry, M. O. (1936). *Nebraska med. J.*, **21**, 289.
 Hoen, T. I. (1936). *Arch. Neurol. Psychiat.*, Chicago, **35**, 158.
 Hoets, J. (1937). *Austral. New Zeal. J. Surg.*, **7**, 170.
 Holmblad, E. C. (1934). *Int. J. Med.*, **47**, 20.
 Hosford, J. P. (1936). *Lancet*, **1**, 249.
 Humphries, S. V. (1937). *S. Afr. med. J.*, **11**, 159.
 Irwin, S. T. (1936). *British Medical Journal*, **1**, 1.
 Jaeger, J. R. (1938). *Rocky Mountain med. J.*, **35**, 205.
 Jefferson, G. (1933). *Practitioner*, **130**, 332.
 Jewett, E. L. (1937). *J. Bone Jt. Surg.*, **19**, 1128.
 Johnson, H. F. (1936). *Nebraska med. J.*, **21**, 175.
 Jones, F. S. (1937). *J. Connecticut med. Soc.*, **1**, 159.
 Jones, R. W. (1931). *British Medical Journal*, **1**, 300.
 — (1934). *J. Bone Jt. Surg.*, **16**, 30.
 — (1938). *Ibid.*, **20**, 567.
 King, T. (1937). *Austral. New Zeal. J. Surg.*, **6**, 380.
 Kleinberg, S. (1936). *Amer. J. Surg.*, **33**, 85.
 Koudienko, J. M. (1936). *Amer. J. Roentgenol.*, **35**, 468.
 Koven, B., and Koven, M. T. (1938). *Indust. Med.*, **7**, 315.
 La Ferte, A. D. (1936). *J. Michigan med. Soc.*, **35**, 366.
 Lambrinudi, C. (1936). *Guy's Hosp. Gaz.*, **50**, 39.
 Laszlo, A. F. (1935). *Ann. Oto-Rhino-Laryngol.*, **46**, 226.
 Latham, O. (1938). *Med. J. Austral.*, **2**, 24.
 Lind, J. E. (1937). *Med. Rec.*, **146**, 65.
 Little, N. (1938). *Med. J. Austral.*, **2**, 89.
 Love, J. G. (1937). *Surg. Clin. N. Amer.*, **17**, 1103.
 Lowman, C. L. (1935). *Amer. J. Surg.*, **29**, 420.
 Lowman, K. E. (1934). *Nav. med. Bull.*, Wash., **32**, 330.
 Mensör, M. C. (1936). *J. Bone Jt. Surg.*, **18**, 153.
 — (1937). *Ibid.*, **19**, 381.
 Mixer, W. J. (1937). *New Engl. J. Med.*, **217**, 899.
 Morrison, W. A., and Flammson, R. J. (1935). *Calif. West. Med.*, **43**, 416.
 Muir, J. B. G. (1935). *Chinese med. J.*, **49**, 1092.
 Munger, A. D. (1937). *J. Urol.*, **37**, 54.
 Munro, A. H. G., and Irwin, C. G. (1938). *Brit. J. Surg.*, **25**, 621.
 Nauth, W. W., and Mattison, P. A. (1938). *Minnesota Med.*, **21**, 179.
 Newell, E. T. (1937). *South. med. J.*, **30**, 799.
 Oldberg, E. (1936). *Surg. Clin. N. Amer.*, **16**, 291.
 Olin, H. A. (1937). *Indust. Med.*, **6**, 401.
 Padula, R. D. (1938). *J. Bone Jt. Surg.*, **20**, 507.
 Pease, C. N. (1935). *Amer. J. Dis. Child.*, **49**, 849.
 Pettit, R. T. (1935). *Illinois med. J.*, **68**, 318.
 Phillips, H. B. (1938). *J. Amer. med. Ass.*, **110**, 1266.
 Plaut, H. F. (1938). *Ohio State med. J.*, **34**, 760.
 Pybus, F. C. (1936). *British Medical Journal*, **2**, 860.
 Reich, R. S. (1938). *Surgery*, **3**, 416.
 Roberts, S. M. (1937a). *J. Bone Jt. Surg.*, **19**, 199.
 — (1937b). *Ibid.*, **19**, 477.
 Rogers, W. A. (1937). *Amer. J. Surg.*, **38**, 599.
 — (1938). *J. Bone Jt. Surg.*, **20**, 689.
 Ruth, V. A. (1936). *J. Iowa med. Soc.*, **25**, 620.
 — (1938). *Ibid.*, **28**, 275.
 Ryerson, E. W. (1937). *Indust. Med.*, **6**, 646.
 — and Christopher, F. (1937). *J. Amer. med. Ass.*, **108**, 468.
 Sadek, E. (1936). *J. Egypt. med. Ass.*, **19**, 173.
 Scarff, J. E. (1937). *N.Y. St. J. Med.*, **37**, 461.
 Siris, I. E. (1938). *Amer. J. Surg.*, **40**, 611.
 Soto-Hall, R., and Haldeman, K. O. (1935). *Surg. Gynec. Obstet.*, **61**, 827.
 — (1936). *Calif. West. Med.*, **45**, 238.
 Speed, K. (1935). *Ann. Surg.*, **102**, 102.
 — (1938). *Arch. Surg.*, **37**, 175.
 Steiner, G. (1938). *Amer. J. Roentgenol.*, **39**, 43.
 Stephen, R. A. (1938). *J. R.A.M.C.*, **70**, 395.
 Stooky, B. (1937). *Surg. Gynec. Obstet.*, **64**, 407.
 Sucha, W. L. (1938). *Nebraska med. J.*, **23**, 47.
 Thomson, J. E. M. (1937). *Surg. Gynec. Obstet.*, **64**, 440.
 — (1938). *Nebraska med. J.*, **23**, 45.
 Tibbets, M. H. (1936). *Minnesota Med.*, **19**, 702.
 Travers, M. P. (1937). *J. Florida med. Ass.*, **23**, 574.
 — (1938). *Ibid.*, **24**, 541.
 Voris, H. C. (1937). *Surg. Clin. N. Amer.*, **17**, 543.
 Wakeley, C. P. G. (1936). *Maingot's Postgraduate Surgery*, **2**, 1861, London.
 Walshe, F. M. R., and Ross, J. (1936). *Brain*, **49**, 277.
 Williams, P. C. (1937a). *J. Bone Jt. Surg.*, **19**, 343.
 — (1937b). *Ibid.*, **19**, 690.
 Williamson, G. A. (1938). *Lancet*, **48**, 16.

CONVULSIONS OCCURRING UNDER VINESTHENE ANAESTHESIA

BY

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Convulsions occurring under vinesthene anaesthesia have not, so far, been recorded in this country, although, according to a personal communication from Professor Rovenstine of New York University, they have been observed in America. During the last two years I have given vinesthene on 2,406 occasions, and there have been nine cases of convulsions, none of them fatal. These cases may be divided into two groups: 196 in-patient operations with four convulsions and 2,210 dental out-patient extractions with five convulsions—incidences of 1 in 49 and 1 in 442 respectively. The nature of the convulsions in the in-patient group was, however, quite different from that of the out-patient group.

Convulsions in the In-patient Group

The convulsions which occurred in the in-patients came on towards the close of the operation, and were very similar to those met with under deep ether anaesthesia, with the exception that each case had an evipan induction. A search of the literature of ether convulsions has not revealed a single case coming after premedication with a barbiturate drug. The intravenous injection of a barbiturate will immediately abort a case of ether convulsions, so an entirely different factor must be present in vinesthene convulsions, as these come on after a barbiturate has been given. Detailed case records follow.

Case 1.—Acute appendicitis in a nurse aged 22. Evipan induction was followed by nitrous oxide-oxygen-vinesthene with carbon dioxide absorption, using Connell's machine. The anaesthesia was normal until sewing up of the peritoneum, then respiration became irregular and convulsions started in the throat muscles, rapidly spreading all over the body and becoming most violent in character. Respiration ceased for two minutes and the pulse failed for a few seconds. Respiration then restarted with inspiratory stridor and gradually returned to normal, the convulsions gradually ceasing. Treatment consisted in stopping the anaesthetic and raising the head. The rectal temperature was 99° and the theatre temperature 75°. Recovery was uneventful. There was no previous history of fits. The soda lime was tested and found not to have lost its absorptive power: it was thought that the condition might have been due to excess carbon dioxide.

Case 2.—Resection of colon in a man aged 72. The anaesthetic sequence was the same as in the first case. Convulsions started while the peritoneum was being sewn up. There was slight cyanosis but no respiratory arrest; the head was raised without effect, but convulsions ceased after five minutes, only to recur later when anaesthesia became too light. The rectal temperature was 99° and the theatre temperature 74°, and a thunderstorm was in progress. Recovery was uneventful. There was no history of fits. 55 c.cm. of vinesthene was used before the convulsions started.

Case 3.—A plastic operation to a kidney in a man aged 24. Vinesthene was used as he had bronchiectasis. The anaesthetic sequence was as in the first case. Convulsions started after twenty minutes' anaesthesia; the head was raised without effect, and 2 c.cm. of evipan was then given intravenously, with immediate cessation of convulsions. Later, as the respiration was shallow, 3 c.cm. of coramine was given. The rectal temperature was 99.6° and the theatre temperature 78°; the weather was dull and oppressive. Recovery was uneventful.

Impaired Memory after Convulsions.—Nine out of sixteen patients (mostly schizophrenics) who recovered or improved after treatment by induced convulsions complained of impaired memory; and in eight there was impairment of intellect. Caution is advised in using this treatment in those whose work depends on memory and intellect.—Disturbance of Memory after Convulsion Treatment. G. Tooth and J. M. Blackburn.—*Lancet*, July 1, 1939, **2**, 17.

Case 4.—Gastrectomy in a woman aged 60. The anaesthetic sequence was as in the first case. Convulsions developed after thirty-five minutes, and there was pronounced cyanosis. 2 c.cm. of evipan was given intravenously, and convulsions ceased. The rectal temperature was 100.2° and the theatre temperature 74°. The patient died from a pulmonary embolism ten days after the operation. At the post-mortem examination special attention was paid to possible abnormalities due to the vinesthene, but nothing was found. Sections of the liver examined microscopically showed no degeneration.

It will be noted that anaesthesia was deep in each case, that sepsis was present in two cases, and that the carbon dioxide absorption technique was used. No cases of convulsions occurred in the in-patient series where carbon dioxide absorption was not employed; but, on the other hand, the cases which received closed-circuit anaesthesia were those which required deep anaesthesia, and for motives of economy the closed circuit was chosen. Taking all things into consideration the convulsions occurring in this group appear to be similar to those met with under ether anaesthesia.

The Dental Out-patient Group

The second group consisted of 2,210 dental extractions in children between the ages of 3 and 9. Anaesthesia was produced by means of a Goldman inhaler, using 3 c.cm. of vinesthene for each case. The nature of the convulsions was quite different from that of the first group. There were five cases of convulsions, and each one came on after the anaesthetic was concluded. Anaesthesia was normal in each case, and the child was then removed to the recovery room, where it recovered consciousness sufficiently to start spitting out. Twitching of the facial muscles then began, and soon all the muscles of the body were involved. The child would then become unconscious and cyanosed, and would cease breathing. At this stage death appeared imminent. Artificial respiration was employed and preparations were made to give evipan; but in each case respiration restarted before evipan was given, and the convulsions gradually ceased. The colour returned to normal and the child would then sleep for about an hour. If awakened before this it would get up and run about the room, banging its head on the walls and appearing completely incoordinated. On the employment of restraint the child would go to sleep once more and waken some time later apparently quite normal. Those who were allowed to sleep following the cessation of convulsions awoke normally in about an hour.

This "syndrome" has now been accepted by the nursing staff as a normal complication of vinesthene anaesthesia and no undue alarm is felt. A return to the use of ethyl chloride as a dental anaesthetic was contemplated, but the nursing staff was emphatic in its preference for vinesthene, despite convulsions, to the far greater dangers of ethyl chloride. Careful inquiry into the history of the children revealed no abnormality; each child was seen a week after the anaesthetic and appeared to have suffered no ill effects. Rectal temperatures taken at the time were normal and no patient was incontinent. Fresh vinesthene was used direct from a sealed ampoule. Fortunately for the sake of comparison, one epileptic child developed convulsions; but these were quite typical, and came on during the anaesthetic, not afterwards. Recovery was uneventful, and the mother admitted that she had forgotten to give him his bromide during the previous week. This case was not included in the above series.

Commentary

The cause of these convulsions which come on after anaesthesia appears quite obscure. It is well known that vinesthene deteriorates rapidly at any temperature, with the formation of aldehydes in considerable quantity. These aldehydes, on the addition of an acid or any hydrogen ion, will polarize and form resins which are definitely toxic to laboratory animals, causing convulsions. On the other hand, the mental symptoms would appear to indicate some form of cerebral damage, fortunately not of a permanent nature. So far as I am aware no death under vinesthene anaesthesia has yet been recorded, although in experimental animals an overdose has resulted in death from a condition resembling acute yellow atrophy. It is therefore recommended that anaesthesia with vinesthene should not exceed an hour in duration. I have, however, given it for three and a half hours without the occurrence of any untoward symptoms. Until a fatality occurs, which I hope may never take place, it is unlikely that the pathology of vinesthene convulsions will be determined.

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TUBERCULOUS LARYNGITIS

AN ANALYSIS OF 428 CASES

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Opinion generally in the medical profession regarding tuberculous laryngitis may be said in recent years to have veered round from a pronounced pessimism to an unjustified optimism. It is now a far cry from Morell Mackenzie's (1880) oft-quoted "The prognosis of laryngeal tuberculosis is always extremely unfavourable and it is not certain that any cases ever recover," and Blumenfeld's (1908) "The cure of tuberculosis of the larynx is still to-day an exception," to the too prevalent opinion, voiced in several recent textbooks of oto-laryngology and of medicine, that tuberculous laryngitis—the most common, and with the exception of tuberculous meningitis the most serious, complication of pulmonary tuberculosis—is a comparatively benign condition requiring no attention beyond the treatment of the pulmonary disease. One reason for this prevalent misconception is that, as the care of tuberculosis is a statutory obligation of the local authority, tuberculous laryngitis is nowadays rarely seen in the clinics of the ear, nose, and throat departments of the large teaching hospitals or even in the special ear, nose, and throat hospitals: a surprising number of the postgraduates who have visited our throat clinic at Colindale Hospital have said that they had never seen or been shown a case of tuberculous laryngitis before. Another reason is that even to-day extraordinarily few patients with suspected or actual pulmonary tuberculosis have the larynx examined before they have been admitted to a special chest hospital or sanatorium: on questioning 400 consecutive tuberculous patients we found that only thirty-two (8 per cent.) had had a