longus appeared to be normal. A large piece of the thickened inferior retinaculum was excised, and this immediately allowed the tendon to move more easily (Fig. 2). No abnormality of the tendons, as is often seen in tendovaginitis at the wrist, was present. The skin was

FIG. 1.—Showing swelling of peroneus brevis and thickening of retinaculum.

FIG. 2.—After excision of thickened area of retinaculum.

sutured and a firm dressing applied. The biopsy report stated: "Chronic inflammatory tissue with no evidence of tuberculosis."

The operation gave complete relief of pain, and the swelling subsided.

Comment

Diagnosis of this condition is not difficult to make if the possibility of its occurrence is remembered.

The operation produced as satisfactory a result in the ankle as it does in the wrist.

REFERENCE

Parvin, R. W., and Ford, L. T. (1956). *J. Bone Jt Surg.*, **38A**, 1352.

In the preface to the eleventh edition of his *Clinical Examination of the Nervous System* Professor G. H. Monrad-Krohn writes that it will probably be the last from his hand. He retired recently from the chair of neurology in the University of Oslo, and the present edition of this well-known textbook has been revised with the assistance of Professor S. Revsum. Although some account of electroencephalographic, radiographic, and other technical methods of diagnosis is included, the main emphasis of the book is on clinical examination, which the author regards as one of the chief pillars of neurology. The book is published by H. K. Lewis and Co. Ltd., London (pp. 466 +xx; illustrated. 1958. 40s.).

NITROUS OXIDE ANAESTHESIA FOR BRONCHOSCOPY

A METHOD SUITABLE FOR OUT-PATIENTS

BY

DAVID BROWN, M.B., F.F.A.R.C.S.

Senior Anaesthetic Registrar, Middlesex Hospital and
London Chest Hospital

AND

J. V. I. YOUNG, M.B., F.F.A.R.C.S.

Anaesthetic Registrar, London Chest Hospital

This paper is based on our experience in giving general anaesthesia for over 700 bronchoscopic examinations, mainly on out-patients.

Bronchoscopy is a challenge for the anaesthetist. Whatever method of anaesthesia is used there is a danger of asphyxia. There are several reasons for this. The patients are often in poor general health, and in particular may suffer from a diminished pulmonary reserve, excessive sputum, hyperinflation, and obstructive airway disease, or cardiovascular insufficiency. The area of lung available for gas exchange may be reduced by occlusion or kinking of some of the more proximal air passages when the bronchoscope is passed deep into the bronchial tree. Severe bleeding after bronchial biopsy sometimes floods the airway. The anaesthetist has to share the whole airway with the bronchoscopist, who often prefers to work in a darkened room.

Most anaesthetic agents present difficulties. If anaesthesia is maintained by drugs given intravenously the effects outlast the examination. Explosive volatile agents are not safe. Chloroform, halothane, and trichlorethylene, apart from other factors, are objectionable to the operator unless the proximal end of the bronchoscope is closed (Herron, 1950; Mathewson *et al.*, 1956; Kovacs, 1957). We use nitrous oxide as our principal agent, adding a small dose of thiopentone for adults to provide a more comfortable and speedy induction. As in other published methods, muscle relaxation is obtained with suxamethonium (Churchill-Davidson, 1953; Kelsall, 1954; Cheatle and Chambers, 1955; Buck, 1955; Green and Coleman, 1955; Pinkerton, 1957).

Preliminary Assessment of Patient

The case histories and chest radiographs are examined. Superior vena caval obstruction is regarded as an absolute contraindication because the action of drugs given intravenously is apt to be delayed, and abolition of the negative inspiratory intrathoracic pressure increases the venous congestion. Cardiovascular disease is a relative contraindication: each patient is assessed individually. We have preferred topical analgesia for two patients with coronary artery insufficiency and for three patients who had cerebral vascular disease. Including five patients with mediastinal venous obstruction, our rejection rate for this type of general anaesthesia has been about 2%.

A simple explanation of the procedure is given to each patient. They are told that they will be in a semiconscious, dream-like state for the examination, which they will not find unpleasant. They are not told that they will go right off to sleep. The majority

remember nothing, but just occasionally a patient will hear and recall what has been said during the bronchoscopy. It is important that everyone in the team should realize this.

Technique

Adults are given 1/75 gr. (0.8 mg.) of atropine sulphate intramuscularly one hour before the examination. A Gordh needle is fixed in position in a left forearm vein. The flowmeters of the gas machine are set to deliver 5 litres of oxygen and 10 litres of nitrous oxide a minute, and the patient is asked to breathe this from a facepiece. Then 1 ml. of 2½% thiopentone solution for each two stone (0.08 ml. per kg.) of body weight is rapidly injected intravenously. To avoid the muscle fasciculation of end-plate depolarization, this solution contains about 1.5 mg. of gallamine triethiodide per ml., prepared by adding 160 mg. to a 100-ml. bottle of 2½% thiopentone solution. After this has taken effect it is followed by suxamethonium chloride, 5 mg. for each stone (0.8 mg. per kg.) of body weight.

When breathing stops the lungs are ventilated with the nitrous oxide and oxygen mixture for three minutes. This reduces the amount of nitrogen and carbon dioxide in the lungs, increases alveolar oxygen tension, and provides an effective concentration of nitrous oxide in the brain. We regard this step as the essential part of the technique. To ventilate the lungs efficiently we use an anaesthetic assembly incorporating a non-rebreathing valve.

The Negus bronchoscope is passed with the gas mixture flowing into its proximal end through tubing attached to the nipple provided. The instrument should be introduced without delay because the absorption of nitrous oxide and oxygen from the alveoli into the blood is rapid. A significant replacement of the mixture of gases in the lungs by room air can occur in a short time if the airway is clear, even if there are no respiratory movements. If delay is anticipated—for example, for teaching purposes—we pass an insufflation catheter through the larynx to within an inch (2.5 cm.) of the main carina to deliver the nitrous oxide and oxygen (Cheatle and Chambers, 1955; Buck, 1955).

Apnoea and full muscle relaxation are kept up during the bronchoscopy by intermittent doses of suxamethonium. The second dose is usually required before passing the bronchoscope. The chest is compressed rhythmically to lessen the accumulation of carbon dioxide (Toker, 1955; Green and Coleman, 1955; Bayuk, 1957). We have found that the Pinkerton (1957) cuirass is more convenient than the hands for this purpose.

Should cyanosis occur it is corrected without delay. The bronchoscope is withdrawn until its tip lies above the main carina. The nitrous oxide and oxygen mixture is passed directly down the lumen of the bronchoscope using a No. 8 Magill (32 F.C.G.) endotracheal tube and connexions (Churchill-Davidson, 1953). Additional oxygen may be added by using the bypass. Loss of gases around the side of the bronchoscope can be reduced by depressing the chin. If the hypoxia is caused by profuse haemorrhage following bronchial biopsy, suction must alternate with inflation. We keep a bronchus blocker available in case the haemorrhage is not controllable by suction.

Usually the bronchoscopy proceeds without interruption. When the examination is over, the

bronchoscope is removed and the lungs are again ventilated with the nitrous oxide and oxygen mixture, using a facepiece and oropharyngeal airway until muscle power has returned. The patient is then sat up, regains unconsciousness quickly, and is encouraged to cough. Out-patients are kept resting for at least one hour.

Children have not been bronchoscoped as out-patients. They are premedicated with an intramuscular injection of papaveretum and scopolamine, to which is added atropine 1/300–1/150 gr. (0.2–0.4 mg.). For each 5 lb. of body weight 1 minim (0.06 ml.)—that is 0.026 ml. per kg.—of the standard solution containing papaveretum 1/3 gr. (20 mg.) and scopolamine 1/150 gr. (0.4 mg.) per ml. is given (Doughty, 1957). Nitrous oxide and oxygen are used to induce anaesthesia, and the intravenous needle is inserted when the child is unconscious.

Results

At the time of writing we had used this method of anaesthesia for 723 bronchoscopies. The age distribution of the patients is given in Table I. The

TABLE I

Age (years):	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90
Total No.	13	1	15	48	140	258	207	39	2
Percentage of total (approx.)	1.8	0.14	2.1	6.6	19.3	35.7	27.2	5.4	0.28

duration of the examination has varied between 3 and 30 minutes, but usually five patients can be anaesthetized and bronchoscoped in an hour. Each patient is conscious and talking rationally before the anaesthetist starts the induction of anaesthesia on the next patient.

There have been no fatalities.

Complications

Before we used gallamine to stop the muscle fasciculations caused by suxamethonium severe pain in the trunk and limbs was common during the first few days following the examination (Morris and Dunn, 1957). Prolonged apnoea occurred once.

Case 1.—A man aged 61 and weighing 9 stone (57 kg.) had a two-months history of breathlessness, cough, blood-streaked sputum, and right-sided chest pain. Radiography showed a right hilar opacity with peripheral shadowing at the right apex. He was premedicated with atropine, 1/75 gr. (0.8 mg.), and given thiopentone, 112 mg., with gallamine 8 mg. This was followed by suxamethonium, 50 mg. Bronchoscopy showed a carcinoma of the right main bronchus. A biopsy was taken. Apnoea persisted for two hours. During this time his lungs were ventilated with nitrous oxide and oxygen. Three pints (1.7 litres) of 5% dextrose in water was given intravenously to help get rid of the paralytic drugs. When spontaneous respiration began it was inadequate. Atropine, 1/75 gr. (0.8 mg.), was given intravenously; after 10 minutes this was followed by neostigmine, 0.5 mg. Improvement occurred, and muscle power and respiration became adequate after a further 2 mg. of neostigmine. He had no after-effects.

Histological examination of the biopsy showed squamous-cell carcinoma, and he was admitted for further treatment. A curare-sensitivity test was performed and no objective signs were noted. For his thoracotomy, general anaesthesia was induced and maintained with nitrous oxide and oxygen.

Relaxation was obtained with the dimethyl ether of tubocurarine (total dose 24 mg.). A Carlens catheter was used to control the airway. After prolonged dissection the growth was found to be inoperable. Atropine, 1/75 gr. (0.8 mg.), with neostigmine, 2.5 mg., was given at the termination of anaesthesia. Recovery was uneventful.

The patient's serum was found to have a pseudocholinesterase level which was low (23 units).

Anticholinesterase administration was also required for another patient.

Case 2.—This patient was a diabetic man aged 43. A total dose of 450 mg. of suxamethonium was required for the bronchoscopy, which lasted 10 minutes. Partial neuromuscular block persisted after the examination, which we judged to be antidepolarizing in nature. Atropine and neostigmine administration was followed by recovery.

Discussion

It was solely out of consideration for the patients themselves that we decided to abandon topical analgesia alone as a routine for bronchoscopy. It is true that people can be bronchoscoped satisfactorily with sedation and surface analgesia only, but there is no doubt that many do suffer considerable distress. We did not regard this suffering as an unavoidable necessity: general anaesthesia might be preferable for routine use. In order to justify the change in an out-patient clinic the method used must not increase the risk for the patient, hamper the operator, give more work to the nurses, or use more hospital beds. We did not find that any of the published methods would fulfil these requirements. Some of the more recent British methods are set out in Table II for comparison.

Our attempt has been satisfactory both for patients and for staff, and we consider it worth recording. Other advantages have become apparent. The surgeons have assured us that the examination is made easier and allows a better view of the less accessible parts of the bronchial tree. No sedative is included in the premedication, and surface analgesics are not used. The patients therefore have the benefit of full protective reflexes immediately after the examination.

The technique is not without its disadvantages. There are the dangers attendant upon the administration of any general anaesthetic, and there is the possibility of prolonged apnoea following suxamethonium. We would like especially to stress the importance of co-operation between surgeon, anaesthetist, and nursing staff. Very close attention should be paid to the finer points of technique.

We found 1/100 gr. (0.6 mg.) of atropine insufficient to antagonize the muscarinic effects of suxamethonium; 1/75 gr. (0.8 mg.) is more satisfactory. Sufficient narcosis is given by 12.5 mg. of thiopentone for every stone (2 mg. per kg.) of body weight. Our intention is to produce in the patient a state in which apnoea and positive-pressure ventilation are tolerated. Full surgical anaesthesia is not intended; a dose of thiopentone sufficient to do this would delay the recovery of consciousness.

Summary

A method is described of giving general anaesthesia for bronchoscopy, using nitrous oxide as the principal narcotic agent. It has been used satisfactorily in over 700 patients.

TABLE II.—General Anaesthetic Methods for Bronchoscopy

Author	Premedication	Induction	Relaxant	Additional Procedures	Respiration During Bronchoscopy	Maintenance	Reversal of Paralysis
Churchill-Davidson (1952)	"Omponon" + scopolamine or "Omponon" + atropine	250-500 mg. thiopentone	80-120 mg. gallamine	Inflation with oxygen. Lignocaine to cords, trachea, etc., followed by inflation with oxygen and TCE*	Spontaneous but weak. If cyanosis occurs, oxygen given by Magill tube inserted into bronchoscope	100-200 mg. thiopentone if necessary	Atropine 1 mg. Neostigmine 2.5 mg.
Churchill-Davidson (1953)	Papaveretum + hyoscine or Papaveretum + atropine	300-500 mg. thiopentone	50 mg. suxamethonium	Hyperventilation with oxygen. Lignocaine to cords, etc.	Complete apnoea. Oxygen via Magill tube in bronchoscope if cyanosis occurs	Additional suxamethonium as necessary. TCE in oxygen if more than 500 mg. thiopentone required to maintain sleep	
MacIntosh (1954)	Morphine + atropine	300 mg. thiopentone	80 mg. suxamethonium	Inflation with oxygen or air. 4% cocaine to pyriform fossa, cords, trachea, etc.	Spontaneous and adequate	Thiopentone as necessary	
Kelsall (1954)	(a) Papaveretum + scopolamine or (b) Morphine + atropine	0.5-1 g. thial barbitone	50 mg. suxamethonium	Inflation with oxygen. 1% amethocaine to cords, etc. Re-inflation with oxygen	Complete apnoea. Artificial respiration by manual compression of chest	Oxygen down side tube of bronchoscope. Suxamethonium as required	Atropine. Neostigmine
			60-120 mg. gallamine	As above, but re-inflation with oxygen + TCE	Spontaneous, but assisted if necessary	Oxygen and TCE down side tube of bronchoscope. Thiopentone + gallamine as required	
Cheate and Chambers (1955)	Papaveretum + scopolamine	Sleep dose of thiopentone	50 mg. suxamethonium	Inflation with oxygen. Insertion of "portex" tube through larynx to level of carina	Complete apnoea	Oxygen 3 l. minute through "portex" tube. Suxamethonium as required	
Buck (1955)	"	Thiopentone	Suxamethonium	Endobronchial suction catheter through larynx to carina	" "	Nitrous oxide 10 l. Oxygen 5 l. minute through catheter	
Green and Coleman (1955)	Pethidine or morphine with atropine, or scopolamine or chlorpromazine	Thiopentone, up to 500 mg.	"	Monaghan type cuirass respirator fitted to patient. Pump turned on. Lignocaine to cords, etc.	Complete apnoea. Artificial respiration by means of cuirass	Thiopentone and suxamethonium as required	
Pinkerton (1957)	Papaveretum + atropine	" "	"	Pinkerton cuirass. Lignocaine + adrenaline to cords, etc.	" " "	Oxygen into suction tube of bronchoscope. Thiopentone + suxamethonium as required	
Present series	Atropine	Thiopentone 12.5 mg. stone; gallamine 0.75 mg./stone	50 mg. suxamethonium	Hyperventilation with 10 l. nitrous oxide and 5 l. oxygen, using a non-rebreathing technique	Complete apnoea. Manual compression of chest	Nitrous oxide and oxygen mixture (10:5)	

* TCE = Trichlorethylene.

We thank the surgeons and anaesthetists of the London Chest Hospital for their co-operation, and in particular Mr. J. R. Belcher, who allowed us to use the method in his out-patient clinic.

REFERENCES

- Bayuk, A. J. (1957). *Anesthesiology*, **18**, 135.
 Buck, H. A. (1955). *Anaesthesia*, **10**, 313.
 Cheate, C. A., and Chambers, K. B. (1955). *Ibid.*, **10**, 171.
 Churchill-Davidson, H. C. (1952). *Ibid.*, **7**, 237.
 — (1953). *Ibid.*, **8**, 128.
 Doughty, A. (1957). *Brit. J. Anaesth.*, **29**, 407.
 Green, R. A., and Coleman, D. J. (1955). *Anaesthesia*, **10**, 369.
 Herron, R. A. C. (1950). *Ibid.*, **5**, 40.
 Kelsall, P. D. (1954). *Brit. J. Anaesth.*, **26**, 182.
 Kovacs, S. (1957). *Anesthesiology*, **18**, 335.
 Macintosh, R. R. (1954). *Anaesthesia*, **9**, 77.
 Mathewson, H. S., McIntyre, J. D., and Meriwether, D. R. (1956). *Anesthesiology*, **17**, 623.
 Morris, D. D. B., and Dunn, C. H. (1957). *Brit. med. J.*, **1**, 383.
 Pinkerton, H. H. (1957). *Brit. J. Anaesth.*, **29**, 421.
 Toker, P. (1955). *S. Afr. med. J.*, **29**, 40.

HAZARDS WITH LOCAL ANAESTHETICS DURING BRONCHOGRAPHY AND BRONCHOSCOPY

BY

SAMIR KUMAR GUPTA, M.B., M.R.C.P.Ed.

Medical Registrar, Islington Chest Clinic and Whittington Hospital, London

Since 1935 amethocaine hydrochloride, which belongs to the procaine group, has been widely used as a local anaesthetic by chest physicians and surgeons. A notable advance in 1943 was the introduction of lignocaine, a drug chemically allied to acetanilide. Lignocaine is regarded as a very safe drug (Hewer and Lee, 1957). Surprisingly, in Britain there have been few published case reports regarding the hazards of these drugs during bronchoscopy and bronchography, though reactions to amethocaine have been described (Hancock, 1939; Jackson, 1949). Here are reported five examples of untoward reactions to these drugs.

Our techniques for bronchoscopy and bronchography are as follows. For premedication we use butobarbitone, 3 gr. (0.2 g.), 90 minutes before bronchography. A subcutaneous injection of atropine sulphate, 1/100 gr. (0.65 mg.), is given one hour later, and simultaneously the patient begins to suck slowly a tablet of amethocaine (65 mg.). On arrival at the x-ray department the patient often spits out the remainder of the tablet when about 2–4 ml. of 4% lignocaine solution is slowly delivered over the back of the tongue into the tracheobronchial tree. Bronchography is usually carried out by "spoon-technique." For bronchoscopy the premedication is either morphine (or pethidine) with atropine sulphate in the usual dosage. Half an hour before the procedure the patient starts sucking amethocaine. In the operation theatre the throat is painted with 2 ml. of 4% lignocaine solution and the tracheobronchial tree is anaesthetized with 0.5% solution as spray.

Case Reports

Case 1.—In 1954 a boy aged 16 was admitted to hospital with a chronic productive cough. Prior to bronchography he was given 65 mg. of amethocaine to suck at 1.45 p.m. At 2.15 p.m. 8 ml. of amethocaine (2%) was administered through a nasal catheter, but was immediately coughed out. A further 8 ml. was given. Within two minutes he became giddy and semicomatose. The catheter was taken out immediately. He rapidly became unconscious, with widely

dilated pupils, not reacting to light. Cyanosis and vomiting soon followed, but there was no convulsion. His pulse and blood pressure remained normal throughout. Oxygen was delivered through a mask and his pharynx was repeatedly aspirated. Half an hour later he regained consciousness, with residual confusion. An uneventful bronchoscopy was carried out later under nitrous oxide and oxygen anaesthesia.

Case 2.—A housewife aged 27 had had pulmonary tuberculosis since 1952. On September 8, 1955, a left upper lobectomy with a left apical thoracoplasty was performed. On September 10 x-ray examination showed a collapsed left lower lobe. Bronchoscopy was immediately arranged. At 3.15 p.m. the back of her throat and pyriform fossa were painted with 2 ml. of 2% amethocaine solution. She at once developed a severe headache. Unconsciousness with a convulsion rapidly followed. She became very pale and cyanosed, her pulse became thready, and her blood pressure was unrecordable. Immediate artificial respiration was begun with oxygen through a mask. Intravenous thiopentone sodium (500 mg.) was given while blocks were placed under the foot of the bed. The trachea and pharynx were frequently aspirated. Improvement quickly followed the administration of intravenous thiopentone. At 4 p.m. nikethamide (250 mg.) was administered intravenously. By 5 p.m. she was merely semicomatose. One hour later she was conscious, although very weak, was slightly confused, and had hypotension (90 mm. Hg). Next morning she was normal.

Case 3.—A 50-year-old housewife came for a bronchogram in August, 1955. One amethocaine lozenge (65 mg.) was given to suck, followed by local instillation of 2% amethocaine (4 ml.) down her air passage. She immediately complained of intense headache and giddiness. She became semicomatose, and remained so for 15 minutes. The foot of the bed was raised on blocks and a careful watch was kept on her. One hour later she was again normal. The bronchography was carried out uneventfully.

Case 4.—A housewife aged 25 was admitted to hospital with increasing dyspnoea on exertion. She had signs of a tight mitral stenosis with sinus rhythm, and also signs of left basal bronchiectasis. Three weeks later, at 2 p.m., she was given one amethocaine lozenge (65 mg.) to suck. The trachea was anaesthetized with 4 ml. of 4% lignocaine solution and within 15 minutes she appeared to be comatose. At 3.30 p.m. she was deeply unconscious, apnoeic, and cyanosed. Artificial respiration and oxygen administration were begun. Intravenous nikethamide (250 mg.) was given, and was repeated twice. She had no convulsion. Solution of adrenaline hydrochloride, B.P. (0.5 ml.), was given subcutaneously. Repeated rigors followed. After two hours of artificial respiration breathing was resumed, but she did not regain consciousness until 7 p.m. She remained confused till late at night. Next morning she was quite normal.

Case 5.—A woman aged 32 was arranged to have a bronchial lavage. Sucking of an amethocaine lozenge (25 mg.) was uneventful. Fifteen minutes later, 1 ml. of 4% lignocaine was poured over the back of the tongue. She complained of immediate palpitation, with tingling and numbness over her right hand. She became pale, sweated, and was slightly breathless. She appeared normal in about 10 minutes, but complained of numbness for another hour. Lavage was done uneventfully.

Discussion

In this series, Case 5 was undoubtedly hypersensitive. Case 2 was probably also hypersensitive, as the symptoms appeared immediately following the amethocaine paint. In Case 4 it is difficult to determine the offending drug; lignocaine might have been responsible, as the drowsiness appeared shortly after its administration, and the pattern of symptoms supports this view (Morrisset, 1957). Case 1 was a definite case of toxicity: this patient had at least four times the safe