ELECTROCARDIOGRAPHY DURING ANAESTHESIA

BY

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INTRODUCTION

As research in anaesthesia develops, the attention of the anaesthetist is being directed more and more to a closer study of the cardiovascular system under the varying conditions of surgery and anaesthesia. The immediate success of all surgical and anaesthetic procedures depends primarily on the patient's cardiovascular system and its ability to withstand the direct and indirect stresses to which it is subjected by the surgeon and the anaesthetist. This would appear to be a self-evident fact, yet, in the assessment of new techniques and manoeuvres, it is surprising how often it has been overlooked. The past few years have seen the introduction of several new and revolutionary anaesthetic techniques; the published reports of these often contain only the briefest and most indefinite references to the cardiovascular states of the patients before, during, and after anaesthesia.

In attempting to assess the value of an anaesthetic technique it is advisable to apply a system of measurement which is reasonably accurate and acceptable to every anaesthetist. In the estimation of cardiovascular function during anaesthesia, and indeed in the estimation of practically every vital function, clinicians have a restricted choice of instruments which can be conveniently used for obtaining accurate records. Pulse palpation can be inaccurate and misleading, and blood pressure estimations are not a reliable index of myocardial function. The electrocardiogram provides us with a record of the heart rate, its rhythm, the site and number of the pacemakers, the efficiency of the conducting tissue, and, to a certain extent, it may depict changes in the position and size of the heart. The electrocardiogram also provides a convenient method for recording fluctuations of autonomic tone occasioned by the numerous drugs now at our disposal, and it is a valuable index of the electrolytic balance of the blood; it is indispensable in deciding what is the cause of cardiovascular collapse in the anaesthetized patient (Johnstone, 1955).

The electrocardiogram is essentially a picture of the site of origin of the stimulus potential and the speed and direction in which it travels to initiate the myocardial contraction—in other words, it depicts the activity of the Purkinje fibres. It provides no indication of the strength of the myocardial contraction. The Purkinje conducting tissue and the myocardium derive nutriment from a common blood supply—the coronary arteries; therefore, if it is observed that certain drugs or manoeuvres are causing electrocardiographic evidence of impairment of conductivity, I feel it is reasonable to assume that the myocardium is involved in the deterioration. The classic signs of diminished conductivity are widening of the QRS complex with displacement of the ST segment and alterations in the shape of the T wave. At this stage in the development of the electrocardiology of anaesthesia I do not propose to enlarge on these changes, as our knowledge is by no means complete.

EQUIPMENT

The most suitable type of electrocardiographic equipment for theatre use is the valve amplifier which combines a direct-writing galvanometer monitored by a cathode ray oscilloscope. The galvanometer and the monitor circuits should be wired in parallel to enable them to be used independently: the direct writer is used intermittently for control checks and for obtaining permanent records of important changes, whilst the monitor is used continuously throughout the operation. Electrodes and leads are positioned so...
as not to interfere with the operation site, and, when single lead electrocardiography is in use, it is generally advisable to attach a third electrode and lead to the patient to "earth" the currents induced from neighbouring electrical equipment.

The recording galvanometer should be sensitized according to the Einthoven convention of 1 cm deflection for 1 millivolt. The amplitude of the deflections on the monitor is adjusted to any sensitivity which permits easy observation when it is placed in a relatively remote part of the theatre.

RECOGNITION OF EXTRACARDIAC POTENTIALS (ARTEFACT)

It has long been known that fallacies may arise in the interpretation of electrocardiograms taken during surgical operations: fibrillary and ectopic auricular contractions are closely simulated by tremors in skeletal muscle; large steel retractors may induce static discharges or may short-circuit the cardiac potentials; and interference from electrical equipment is also a frequent source of disturbance.

Figure 1 illustrates a series of electrocardiograms distorted by various types of extracardiac interference:

A shows the superimposition of the 50-cycle alternating current (AC) interference on the cardiogram and its elimination by "earthing" the patient. Usually this interference is eliminated by the "earth" terminal on the power plug and it is seldom necessary to use a separate "earth" lead. Endoscopes in situ are the worst offenders in this respect, and AC interference from them is very difficult to eliminate. It is still possible to identify the cardiac potentials through the AC "hum."

B illustrates the effect of diathermy on the cardiogram. This interference is impossible to eliminate and it completely obliterates the cardiac potentials. It does not constitute a serious drawback as its use is transient. The diathermy current does not damage the amplifying or monitoring circuits, but its high frequency vibrations may shatter the galvanometer pen; for this reason it is advisable to have the galvanometer on an independent circuit.

C shows interference due to tense somatic muscles in a patient awaiting the induction of anaesthesia. The P waves have been obscured and it is impossible to determine the site of the pacemaker.

D shows, in the same patient as C, the abolition of somatic interference by the administration of thiopentone. The P waves become clear as soon as the patient loses consciousness.

E illustrates interference encountered when scissors—metal friction—or large retractors are used during the operation. These deflections
often closely simulate ectopic contractions (extrasystoles) and activate certain types of electronic cardiotachometers, causing these instruments to give erroneously high recordings of the pulse rate.

F shows somatic interference caused by the intravenous administration of suxamethonium. G shows the disappearance of the somatic potentials with the onset of suxamethonium paralysis.

H was taken during trichlorethylene tachypnoea. The deflections marked “Dp” are due to diaphragmatic contractions and closely resemble auricular ectopic beats.

I shows distortion of the cardiogram by hiccupping during upper abdominal surgery in an inadequately anaesthetized adult.

J shows considerable alternations in the electrical potential of the cardiac deflections during anaesthesia in a patient under controlled respiration. During pulmonary inflation the R wave becomes very small. This diminution of voltage suggests a change in the position of the heart relative to the skin electrodes and does not indicate myocardial deterioration. Change in the voltage of the cardiac deflections (electrical axis) may be induced by the Trendelenburg and lateral positions, by complete curarization, and by thoracotomy. These factors should be taken into account when assessing the effects of drugs or operations on the cardiogram.

ELECTROCARDIOGRAPHIC ABNORMALITIES DUE TO AUTONOMIC IMBALANCE

The commonest electrocardiographic abnormality encountered in the anaesthetized patient is a change either in the position or number of the pacemakers. Widening of the QRS complex, shift of the ST segment, and alterations in the T wave have not been fully investigated and their significance is not yet clear. Pacemaker changes, however, have been well documented and their significance is more fully understood; this paper will be concerned mainly with those abnormalities which, depending on the aetiology, fall into three main groups: those due to vagal activity, those secondary to sympathetic stimulation, and the fibrillary phenomena.

Figure 2 illustrates diagrammatically the myocardium with its conducting system and autonomic innervation; superimposed are examples of cardiograms obtained from the particular areas of the heart to which they are related on the diagram. The sinus node and the atrioventricular node are richly innervated by both sympathetic and parasympathetic fibres and their activity is governed by these nerves, the sympathetic being stimulant and the parasympathetic inhibitory. The ventricular myocardium and the peripheral Purkinje network have a well defined sympathetic innervation but the parasympathetic supply of these structures has not been demonstrated anatomically, though its influence can be observed by physiological means (Johnstone, 1953).

ELECTROCARDIOGRAPHIC DISTURBANCES DUE TO VAGAL STIMULATION

Increase in vagal tone may be induced by a variety of methods in nonatropinized patients: inhalation of irritant anaesthetic vapours may cause reflex vagal inhibition of the heart (pulmonary cardiac reflex); administration of parasympathetic stimulants such as neostigmine or suxamethonium; high spinal anaesthesia by abolishing the normal sympathetic countereffect; by surgical stimulation either of the parasympathetic centre in the hypothalamus or of the vagus nerve peripherally; and sometimes by overdistension of the lungs during controlled respiration.

Vagal stimulation causes slowing of the sinus rate with flattening of the P wave (fig. 2A and 2B). More intense stimulation may inactivate the sinus node causing cardiac arrest (fig. 2G). The efficiency of the atrioventricular node and the bundle of His may be impaired by vagal stimulation, leading to partial heart block (fig. 2c: note prolonged PR interval), or to complete heart block with ventricular standstill (fig. 2h); rarely the vagal stimulus may block one of the branches of the bundle of His, producing the electrocardiogram typical of intraventricular (bundle-branch) block (fig. 2r).

More often, however, when the sinus node is inactivated by vagal stimulation, the atrioventricular node escapes and takes over the role of pacemaker with the appearance of characteristic electrocardiograms, the form of which is related...
to the site of the pacemaker within the atrioventricular node and bundle. When the pacemaker is within the atrioventricular node the stimulus spreads backward to the auricles (retrograde spread) and an inverted P wave precedes the ventricular complex (fig. 2c); a pacemaker within the bundle causes synchronous contractions of the auricle and ventricle, the P wave being buried in the ventricular complex and therefore invisible (fig. 2d); a pacemaker situated low in the bundle near its bifurcation causes ventricular contraction to precede the auricular contraction, when an inverted P wave follows the R wave (fig. 2e). Vagal stimulation not
infrequently causes the auricles and ventricles to beat independently (atrioventricular dissociation), the auricles being controlled by the sinus node and the ventricles by the atrophicventricular node; sometimes an occasional sinus impulse will be conducted to the ventricles, causing a pulse irregularity, referred to as atrophicventricular dissociation with interference (fig. 3c).

**ELECTROCARDIOGRAPHIC DISTURBANCES DUE TO SYMPATHETIC OVERACTIVITY**

The effects of sympathetic stimulation are seen mainly in the sinus node and in the peripheral parts of the Purkinje network and are essentially those of increased irritability of the cells in these structures. Moderate stimulation causes sinus tachycardia with increase in the amplitude of the P wave and the appearance of occasional ventricular extrasystoles, due to activation of cells in the ventricular portions of the Purkinje system. More intense stimulation produces more frequent ventricular extrasystoles which may eventually dominate the cardiac activity, either in the form of a multifocal ventricular tachycardia (fig. 2L), bifocal ventricular tachycardia (fig. 2K), or a unifocal ventricular rhythm (fig. 2J). Note the similarity in the electrocardiographic form of a unifocal rhythm and an intraventricular block.

Sympathetic overactivity may be precipitated by a variety of factors:

1. The induction of anaesthesia by the inhalation of gases or vapours, particularly if it is associated with struggling and partial asphyxia.
2. The use of agents which are known to activate the sympathetico-adrenal mechanisms, e.g. chloroform, cyclopropane and trichlorethylene.
3. By the administration of adrenaline or nor-adrenaline.
4. By the retention of carbon dioxide which has pharmacological properties not unlike those of adrenaline.
5. By the surgical manipulation of suprarenal tumours.

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**Fig. 3**

The irregular pulse during anaesthesia. See text for explanation.
The hypertensive effect of sympathetic stimulation may produce reflex vagal inhibition of the heart through activation of the baroceptors in the aortic arch and carotid sinuses; the true effects of sympathetic stimulation on the heart will therefore be rendered more obvious by the administration of atropine.

THE IRREGULAR PULSE DURING ANAESTHESIA

The nature of a pulse irregularity is best determined by means of the electrocardiograph. Some of the causes of the completely irregular pulse are illustrated on figure 3; sinus arrhythmia and the rarer forms of sinus block are not included. Tracing 3A shows a sinus rhythm interrupted by isolated ventricular extrasystoles, and tracing 3B shows a multifocal ventricular rhythm in which the sinus beats have been completely replaced by ventricular extrasystoles. Tracing 3C illustrates an atrioventricular dissociation with interference. This abnormality is invariably due to vagal overactivity and is best treated by the intravenous administration of atropine.

Tracing 3E is one of auricular flutter with an irregular ventricular response. This is an uncommon complication of anaesthesia but it occurs in experimental work on auricular fibrillation in which auricular flutter is seen during the transition from sinus rhythm to auricular fibrillation. Tracing 3B is of auricular fibrillation. Tracing 3E is of auricular fibrillation with frequent ventricular extrasystoles.

THE FIBRILLARY PHENOMENA

Fibrillation, which may affect either the auricles or the ventricles, does not appear to be directly related to autonomic imbalance as are the other forms of arrhythmia encountered during anaesthesia. Important work on the aetiology of auricular fibrillation has recently been reported by Professor Burn and his colleagues (1956). It would appear that in fibrillation the abnormality lies in the myocardial cells rather than in the cells of the conducting tissue.

Ventricular fibrillation is often alleged to be the cause of cardiovascular collapse during anaesthesia, but unfortunately the exact circumstances in which it occurs and its electrocardiographic confirmation are seldom presented. As yet, comparatively little is known of its aetiology, though it is almost certain that it does not occur without preceding electrocardiographic changes. There is little doubt that it may be caused by several factors, all of which probably have myocardial ischaemia as a common predisposing effect. Every effort should be made to identify the predisposing causes and I feel sure that it will be much more satisfactory to prevent the occurrence of ventricular fibrillation than to devise complex and dangerous methods of treatment for it.

Auricular fibrillation is a rare complication of anaesthesia although frequently encountered in the pre-operative and postoperative phases. In most cases established auricular fibrillation is uninfluenced by anaesthesia, but the atrioventricular node, the bundle of His, and the peripheral Purkinje network are still subject to autonomic effects; figure 3E shows a pre-operative auricular fibrillation with frequent ventricular extrasystoles precipitated by the induction of anaesthesia with trichlorethylene.

Auricular fibrillation may sometimes be abolished by general anaesthesia, as the following illustrates.

CASE 1. Male, 72 years. Hemicolectomy for carcinoma of the colon. Hypertensive heart disease with auricular fibrillation of at least six weeks duration. Premedicated with atropine 0.5 mg. Anaesthesia induced with thiopentone 250 mg and maintained with cyclopropane and oxygen in a closed circuit with assisted respiration. After twenty minutes of cyclopropane anaesthesia the auricular fibrillation reverted to auricular flutter (fig. 4C) and five minutes later a sinus rhythm was present (fig. 4D). Sinus rhythm persisted throughout the remainder of the operation and during convalescence.

In rare instances patients subject to paroxysmal auricular fibrillation may develop the arrhythmia at any stage of the operation.

CASE 2. Male, 63 years. For suprapubic prostatectomy. Fit and well. Heart and lungs normal. History of severe palpitations precipitated by emotional stress; each attack of palpitations lasted several hours. Premedicated with atropine 0.6 mg and pethidine 50 mg. On arrival at the operating theatre a sinus rhythm was present (fig. 5A). A few minutes later, whilst a lumbar puncture was being performed for the induction of spinal analgesia, auricular fibrillation suddenly developed (fig. 5B). 1.2 ml of heavy cinchocaine were injected into the subarachnoid space and the patient placed in about 10 degrees of Trendelenburg position. Five minutes later spinal analgesia was present and
information it would be difficult to select the specific therapy required for the correction of the irregularity. Regularity of the pulse does not imply normality of the rhythm, for ventricular extrasystoles may occur when the ventricle is empty, and therefore fail to produce a palpable pulse, but nevertheless block the following sinus beats and cause what appears on palpation to be a bradycardia. This point is illustrated in the following example.

**Case 3.** Female, 54 years. Poorly nourished. Six months history of loss of weight, increasing lassitude and weariness, loss of appetite, indigestion, and

the systolic pressure had dropped to 90 mm Hg; auricular fibrillation was still present. Thiopentone 250 mg were injected intravenously to eliminate any emotional factors which may have been related to the fibrillation; the systolic pressure then dropped to 60 mm Hg, the fibrillation remained unchanged and the ventricular response rose to 160 beats a minute (fig. 5c). Methoxamine, 10 mg intravenously, restored the systolic pressure to 120 mm Hg, but failed to influence the fibrillation. Digoxin 0.5 mg was then injected intravenously and sinus rhythm returned five minutes later (fig. 5d). A suprapubic prostatectomy was performed without difficulty and the patient made an uneventful recovery without any further paroxysms of auricular fibrillation.

These tracings are presented in order to illustrate the use of electrocardiography in determining the cause of a pulse irregularity; without this

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**FIG. 4**

Case 1. All lead 2. Male, 72 years. Hemicolecotomy.
A. Before induction. Auricular fibrillation.
B. After thiopentone. Auricular fibrillation.
C. After cyclopropane for 20 minutes. Auricular flutter.
D. After cyclopropane for 25 minutes. Sinus rhythm.
E. During recovery of consciousness.

**FIG. 5**

See text for explanation.
occasional vomiting. Radiological examination revealed a doubtful filling defect in the stomach, necessitating an exploratory laparotomy. Premedication was with atropine 0.6 mg and pethidine 50 mg. Anaesthesia was induced with 400 mg of thiopentone, an endotracheal tube inserted and a nitrous oxide-oxygen mixture administered. Prior to opening the peritoneal cavity 20 mg of laudexium methylsulphate (Laudolissin) were injected intravenously and respiration controlled. Nothing abnormal was found in the gastro-intestinal tract and the abdomen was closed. Neostigmine 2.5 mg with atropine 1 mg were then administered intravenously, but failed to restore spontaneous respiration. During the next twenty minutes a further 7.5 mg of neostigmine were given with atropine 0.5 mg to keep the pulse rate within reasonable limits, but complete apnoea persisted. Controlled respiration was continued, using a 50 per cent oxygen-nitrous oxide mixture with a total gas flow of 10 litres per minute from a Boyle's apparatus. Thirty minutes later the patient was found to be in a collapsed state, pale, sweating, radial pulse rate 40 per minute, systolic pressure 65 mm Hg, and still completely paralysed; a cholinergic crisis was suspected in view of the very slow radial pulse and the sweating. An electrocardiogram, however, revealed ventricular extrasystoles (fig. 6A). Carbon dioxide retention due to inadequate ventilation was suspected. Vigorous ventilatory efforts, with about 20 litres per minute from the Boyle's machine, failed to abolish the arrhythmia and the blood pressure dropped still lower. The endotracheal tube was removed and auffed tube reinserted and the patient ventilated with a Coxeter-Mushin circuit with soda lime and a 10-litre-per-minute gas flow. Three minutes later the extrasystoles disappeared and a steady sinus rhythm of 75 beats per minute returned (fig. 6B): systolic pressure rose to 85 mm Hg and the sweating ceased. Seven hours later spontaneous respiration returned and the patient made an uneventful recovery. Postoperative investigations revealed the presence of myasthenia gravis, affecting mainly the muscles of the upper limbs; neostigmine therapy abolished the patient's symptoms.

CONCLUSION

This paper by no means covers the many aspects of the electrocardiology of anaesthesia. The subject is at an early stage of development, but it is becoming more useful as an aid in reducing the risks of surgery and anaesthesia. It is a subject which can best be appreciated by practical experience, as the range of both normal and abnormal cardiograms is wide. The significance of electrocardiography has been ably described in the words of David Scherf and Adolph Schott (1953 a, b): "At first sight this topic may appear to be a very limited one, but closer study proves the view to be erroneous and reveals not only its considerable scope and complexity but also the numerous links which relate it to other biological problems, physiological as well as clinical."

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REFERENCES


SOCIETY NEWS

GLASGOW AND WEST OF SCOTLAND SOCIETY OF ANAESTHETISTS

On Saturday, October 20, 1956, at Adam House, Edinburgh, a Joint Meeting was held with the Association of Anaesthetists of Edinburgh. Dr. McCallum Miller, of Edinburgh, presided, and the speaker was Dr. W. N. Rollason, of Hull. The subject was “The Role of the Anaesthetist in the Treatment of Facial Neuralgias.”

Dr. Rollason said that the treatment of facial neuralgias involved dentist, neurologist, neurosurgeon, and anaesthetist whose place in treatment was increasing in importance. The speaker at this point paid tribute to Wilfred Harris for his work on this subject.

The patients are referred to the Anaesthetic Outpatient Department, and to date number 41, of whom 26 were female and 15 male.

Facial neuralgias are divided into the following types: dental, ocular, auricular, atypical, postherpetic, glossopharyngeal, tic douloureux, intramedullary, osteitis, tumour, and psychogenic. The diagnosis is made from the history and physical examination with X-ray investigation of skull, sinuses, temporomandibular joints, cervical spine and teeth. Blood is taken for Wasserman and Khan reactions and urea estimation. Urine analysis is done.

When the diagnosis is made, the specific type of facial neuralgia is referred to the appropriate specialist, the commonest cause being dental.

Tic douloureux occurs usually in people over 50 years of age. The constant symptom is pain—unilateral, trigeminal in distribution, agonizing, paroxysmal, precipitated by an event such as shaving, talking or washing. Certain trigger zones are present where light touch to a particular area will precipitate tic douloureux. Facial analgesia and absence of the corneal reflex are never found. The differential diagnosis is stressed as being of the greatest importance. Treatment is under the control of the specialist most concerned. The auricular type may be helped by injection of the great auricular nerve; the postherpetic by X-ray therapy; glossopharyngeal by topical anaesthesia to the areas or by nerve block in the neck. The intramedullary type is due usually to syringomyelia and is treated by analgesic drugs and chlorpromazine. The neoplastic type is usually accompanied by paralysis and anaesthesia and is referred to the neurosurgeon, while neuralgia due to psychogenic causes is precipitated by emotional trauma and may respond to psychotherapy.

Tic douloureux in the early stages may be controlled by phenobarbitone, pure trichlorethylene, nicotinic acid, radiotherapy, vitamin B12. Before commencing radical treatment its effects must be plainly explained to the patient who must elect to undergo it. The points to be stressed are that the numbness will be permanent and that the eye on the affected side may be involved.

Dr. Rollason considered that in young patients operation is the procedure of choice, injection being reserved for the elderly. With injection the death rate is nil, the failure rate high, and the technique difficult. Thirty-five cases have been injected using the anterior approach. The method of injection was described and the precautions taken during and after treatment, along with an impressive list of possible complications.

In conclusion Dr. Rollason described the conditions in a provincial hospital without readily obtainable neurosurgical aid, and stressed the necessity of accurate diagnosis.