THE CARDIOVASCULAR EFFECTS OF OXYTOCIC DRUGS

MICHAEL JOHNSTONE

SUMMARY

The effects of ergometrine 0.5 mg i.v. and of synthetic oxytocin 5 units i.v. on the alpha and the beta adrenoceptive blood vessels of the forearm have been studied by means of volume-pulse plethysmography in 40 gynaecological patients. Ergometrine constricted the alpha and the beta blood vessels. It is thought that the widespread vasoconstriction may precipitate post-partum hypertension or pulmonary oedema in obstetric patients when other alpha vasoconstrictive factors are present. Synthetic oxytocin caused transient dilatation of the alpha and the beta adrenoceptive blood vessels and predisposed to severe postural hypotension. The hypotensive effect of the oxytocin was much less when the drug was given to patients who were in the lithotomy position. It was concluded that oxytocin is preferable to ergometrine in obstetric patients in whom an alpha vasoconstrictive tendency has been induced by one or other of the several factors described.

Ergometrine maleate and synthetic oxytocin (Syntocinon) are used to stimulate uterine activity in parturient women. Familiarity with their effectiveness has increased the dosage and the indications for them. The intravenous dose of ergometrine originally recommended was 0.05 mg (Dudley and Moir, 1935). Intravenous doses of 0.5 mg are now commonplace. Oxytocin is injected intravenously in single doses up to 10 units. The mixture of ergometrine 0.5 mg and oxytocin 5 units has been advised to expedite the onset of uterine activity when the drugs are given intramuscularly (Embrey, 1961). The oxytocin acts in about 2 minutes and is very short acting—about 3 minutes—whereas ergometrine has a latent period of about 7 minutes after intramuscular injection and is long-acting.

The actions of ergometrine and of oxytocin are not confined to the parturient uterus. Brooke and Robinson (1970) observed that ergometrine 0.25 mg intravenously constricted the systemic venous bed and caused a 41 per cent decrease in the venous compliance of the forearm veins. The venoconstriction was associated with rises in the mean arterial and central venous pressures. They considered that the peripheral vascular reaction to ergometrine may provoke pulmonary oedema and other cardiovascular problems in obstetric patients with heart disease. Baillie (1963) reported rises in systolic and diastolic blood pressures after ergometrine 0.5 mg intravenously during operative delivery under general anaesthesia in obstetric patients with pre-eclamptic toxaemia. He suggested that the post-partum hypertension caused by ergometrine may provoke an encephalopathy similar to post-partum eclampsia. Clinical and animal studies suggest that ergometrine constricts coronary arteries afflicted with atheromatous degeneration (Garattini and Shore, 1964; Stein, 1949). The evidence includes electrocardiographic signs of coronary insufficiency which occurred in the absence of blood pressure changes after ergometrine 0.2 mg intravenously.

Nakano and Fisher (1963) described the cardiovascular effects of oxytocin in animals. They observed that it decreases the cardiac output and blood pressure by reducing the calculated total peripheral resistance. The fall in blood pressure was rapidly followed by increases in the heart rate through reflex stimulation of the sympathetic baroreceptor system. The depressant effect was very transient. Venous occlusion plethysmographic studies in man showed that oxytocin causes peripheral vasodilatation by a directly depressant effect on the smooth muscle of the peripheral blood vessels (Deis, Kitchin and Pickford, 1963). Apparently minor and fleeting changes in the T-wave of the electrocardiogram

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after intravenous doses of oxytocin 10 units have been reported (Bergquist and Kaiser, 1959).

The purpose of this study was to examine by means of volume-pulse plethysmography the effects of ergometrine and of oxytocin on the blood vessels of the finger (alpha adrenoceptive) and the skeletal musculature (beta adrenoceptive) in gynaecological patients undergoing spontaneous or therapeutic abortion. An extensive review of the literature concerning the adrenoceptive features of the major systemic vascular beds has been presented by Green and Kepchar (1959). They describe the blood vessels of the kidney, skin, splanchnic and meningeal circulations as being alpha adrenoceptive. These vessels constric in response to adrenergic stress and are dilated by blockade of their sympathetic innervation. The blood vessels of the skeletal muscles contain mainly beta adrenergic receptors and are dilated by adrenergic stimuli and constricted by sympathetic blockade. There is no clear evidence of the presence of either alpha or beta adrenergic receptors in the brain or pulmonary circulations. These appear to be controlled mainly by a metabolic autoregulatory mechanism which may be independent of sympathetic influence. Intense sympathetic activity such as that provoked by the injection of large doses of catecholamines causes widespread constriction of both the alpha and beta blood vessels and shifts the blood volume to the relatively passive brain and pulmonary vascular beds, thereby predisposing to pulmonary oedema or cerebral oedema (Adriani et al., 1967; Ringrose, 1962).

**METHOD**

Forty patients between the ages of 17 and 32 years were selected for study. Each had clinically normal cardiovascular, respiratory, renal and other systems and was admitted to the hospital for gynaecological surgery, mostly dilatation and curettage of the uterus for the removal of retained products of conception. All were normotensive, normovolaemic, afebrile and free from pain. The effects of ergometrine 0.5 mg intravenously were studied in 20 patients and those of oxytocin in a second group of 20 patients.

Preoperative sedation was needed to eliminate the alpha vasoconstrictive reaction to fear in patients not afflicted with pain (Johnstone, 1971). The mixture has no direct effect on vasomotor activity as the vasoconstrictive reactions to pain, cold, surgical trauma and to methoxamine are unimpaired by it. Ergometrine 0.5 mg was given intravenously to each patient on arrival in the anaesthetic room about 15 minutes before the induction of anaesthesia.

No premedication was given to the patients of group 2. It was anticipated that the expected vaso-dilative effect of oxytocin might be concealed by the alpha vasodilatation of sedation. This group was divided into two sub-groups of 10 patients to determine the influence of posture on the hypotensive effect of oxytocin. Each patient in the first sub-group received oxytocin 5 units intravenously before the induction of anaesthesia, whilst resting supine in a slightly head-up position. A similar dose of oxytocin was given intravenously to the patients of the second sub-group when they had been placed in the lithotomy position and a slight head-down tilt after the induction of anaesthesia.

Crystal plethysmographic transducers and platinum-foil electrocardiographic electrodes were used. The crystal plethysmographic transducer is suitable for detecting changes in the calibre of the alpha and beta adrenoceptive blood vessels. It consists of a 10 mm length of piezo-electric element embedded in a disc or ring of acrylic resin (Horsfall, 1968). Finger (alpha) plethysmograms are obtained by applying a ring sensor to a finger. Muscle (beta) plethysmograms are displayed by placing a disc sensor on the skin covering the muscle mass of either the forearm or the calf (fig. 1). Transmission of the electrical analogues of the pulse-waves and the electrocardiogram was achieved with tiny, very short range radio transmitters (Horsfall, 1968; Johnstone, 1967).

Tiny radio transmitters of different frequencies were used for the finger and muscle plethysmographic transducers. A third transmitter was used for single-lead electrocardiography (Lead 4) in the patients treated with oxytocin. One or other of the cardiovascular signals was displayed continuously on an oscilloscope and permanent recordings were made at appropriate times with a single-channel Mingograf writer fed by a conventional radio tuner. The sensitivity of the amplifying circuits of the receiving and display systems was set with a 1 mV pseugenator to give a deflection of 3 mm per 1 mV for the plethysmography (Horsfall, 1968).
FIG. 1. The finger and muscle plethysmographic transducers with their transmitters in position for use. Alpha plethysmograms are obtained by placing the ring transducer on the finger. To obtain beta plethysmograms a strip of metallic lead 3 cm wide and 5 mm thick is moulded round the muscle mass of the forearm leaving a space of about 4 cm between the ends. The crystal transducer is placed on the skin between the ends of the metallic strip. The assembly is held firmly in position with a bandage of non-elastic adhesive tape.

standard sensitization of 1 cm per 1 mV was used for the electrocardiography.

The finger and muscle transducers were applied to each patient on arrival in the anaesthetic room. A brachial cuff was placed in position for the measurement of systolic and diastolic blood pressures by sphygmomanometry. The e.c.g. electrodes were applied to the patients who were to be treated with oxytocin. A plastic cannula was then inserted into a forearm vein before the injection of the oxytocic drug. It should be noted that the pain of venepuncture causes reflex alpha vasoconstriction and beta vasodilatation in sedated patients (fig. 2). The effect is very transient and disappears when the patient is left undisturbed. Ergometrine was injected when the alpha vasodilatation reappeared.

The finger plethysmogram, the muscle plethysmogram, the electrocardiogram and the blood pressure were recorded immediately before the injection of the oxytocic drug and at 30 second intervals for 10 minutes thereafter in each patient. Anaesthesia was then induced in each patient with an intravenous injection of propanidid mixed with atropine 0.5 mg and maintained with nitrous oxide, oxygen and halothane. Plethysmograms, electrocardiograms and blood pressures were recorded throughout anaesthesia and for 10 minutes after the operation in each case. A similar monitoring procedure was followed in the 10 patients to whom oxytocin was given when they were placed in the lithotomy position after the induction of anaesthesia.

RESULTS

Prior to the injection of ergometrine the range of amplitudes of the finger pulse waves was 10 to 18 mm (mean 13). The range of amplitudes of the muscle waves was 8 to 30 mm (mean 17). The pulse rates were between 62 and 85 (mean 71) beats/min. The systolic pressures were from 85 to 120 mm Hg (mean 103) and the diastolic pressures were 40 to 80 mm Hg (mean 61).

Ten minutes after the injection of ergometrine the range of amplitudes of the finger pulse waves was 2 to 14 mm (mean 4). The amplitudes of the muscle pulsations were 1 to 19 mm (mean 8). The
systolic pressures were between 85 and 150 mm Hg (mean 114) and the diastolic pressures were between 55 and 110 mm Hg (mean 76). The pulse rates were unchanged after ergometrine. Anaesthesia produced dilatation of the finger blood vessels, the range of amplitudes being 12 to 30 mm (mean 14) but the muscle vessels remained constricted with an amplitude range of 2 to 12 mm (mean 3). Both finger and muscle blood vessels constricted immediately after withdrawal of the anaesthetic. The sequence of events in a patient of this group is illustrated in figure 3 and the mean amplitudes of the plethysmograms of the group are shown in figure 4.

Prior to the administration of oxytocin to the 10 conscious patients in the supine, slightly head-up position the range of amplitudes of their finger pulse waves was 2 to 10 mm (mean 6). The amplitude range of the muscle waves was 10 to 30 mm (mean 16). The pulse rates were between 60 and 110 (mean 95) beats min. The systolic pressures were between 105 and 130 mm Hg (mean 115) and the diastolic pressures were between 70 and 85 mm Hg (mean 76). One minute after the injection of oxytocin the amplitudes of the finger pulse waves had increased to a range of 10 to 25 mm (mean 16) and the muscle amplitudes also increased to a range of 12 to 35 mm (mean 19). The pulse rates increased to a range of 110 to 140 (mean 127) beats/min. The systolic blood pressures fell to a range of 40 to 85 mm Hg (mean 76), and the diastolic range dropped to 45 to 0 mm Hg. Shortly after the onset of the tachycardia
the electrocardiogram showed sinus rhythm in all patients, a deep inversion of the T-wave in 8 patients (fig. 5) and flattening of the T-wave in 2. Three minutes after the injection of oxytocin all measurements had returned to their pre-injection values. The sequence of plethysmographic changes in one of the patients in illustrated in figure 6 and the mean amplitude changes for the group are shown in figure 7.

The administration of oxytocin to the 10 anaesthetized patients in the lithotomy position caused comparatively little change in cardiovascular behaviour. The electrocardiograms were normal prior to the injections. The range of finger pulse wave amplitudes was 12 to 25 mm (mean 16). The range of amplitudes of the muscle pulsations was 5 to 10 mm (mean 6). The pulse rates were between 75 and 120 (mean 95) beats/min. The systolic pressures were between 85 and 125 mm Hg (mean 106) and the diastolic pressures were 55 to 90 mm Hg (mean 76). After the injection of oxytocin the T-waves of the electrocardiogram remained unchanged in 8 patients, and 2 showed a very slight and transient decrease in amplitude. The range of pulse rates was 100 to 120 (mean 112) beats/min. The amplitudes of the finger pulse waves were slightly diminished, the range being 11 to 19 mm (mean 13) and the amplitudes of the muscle waves were transiently increased, the range being 8 to 15 mm (mean 11). The systolic pressures were unchanged in 7 patients, the remainder showing very transient falls not exceeding 15 mm Hg. The sequence of events in a patient of this group is illustrated in figure 8 and the mean amplitudes of the various plethysmograms are shown in figure 9.

**FIG. 6.** Finger (alpha) and muscle (beta) plethysmograms from a patient of 26 years. Supine, slightly head-up position. No premedication. Recorder speed 25 mm/1. sec. (a) On arrival in the anaesthetic room, (b) 1 minute after oxytocin 5 units intravenously, (c) 2 minutes later, (d) during anaesthesia.

**FIG. 7.** Mean amplitudes of the finger and muscle pulse waves in the pre-anæsthetic oxytocin cases with additional values for tracings taken during and after anaesthesia.

**FIG. 8.** Finger (alpha) and muscle (beta) plethysmograms from a patient of 28 years. Lithotomy position. No premedication. Recorder speed 25 mm/1. sec. (a) During surgery under halothane anaesthesia, (b) 1 minute later, after oxytocin 5 units intravenously. (c) 1 minute later.

**DISCUSSION**

Ergometrine 0.5 mg intravenously constricted the alpha adrenoceptive blood vessels in 19 of 20 patients. Constriction of the beta adrenoceptive vessels occurred in all patients and appeared about three minutes after the injection in each case. The alpha vasoconstriction was relatively slower in onset and was not complete until 5 minutes after the injection. Increases in the diastolic blood pressures
CARDIOVASCULAR EFFECTS OF OXYTOCIC DRUGS

FIG. 9. Mean amplitudes of the finger and muscle plethysmograms taken before and after oxytocin 5 units intravenously in 10 patients in the lithotomy position.

occurred in all patients. Moderate increases occurred in the systolic pressures of 12 patients, 6 were unchanged and 2 were decreased. Halothane anaesthesia reduced the alpha vasoconstrictive effect of ergometrine but had no effect on the beta constriction. The alpha constriction returned immediately after the withdrawal of anaesthetic and persisted for at least 2 hours as judged by the appearance of the superficial veins of the forearms.

The vasoconstrictive effect of ergometrine is of much wider distribution than that associated with sympathetic activity or sympathomimetic drugs. The effect of the latter is probably confined to the alpha adrenoceptive vessels, the beta vessels being either unaffected or dilated. The widespread vasoconstriction caused by ergometrine increases the total peripheral resistance and raises the central venous and mean arterial pressures (Brooke and Robinson, 1970; Keddie, Provan and Austen, 1966). The consequences of widespread peripheral constriction may be pulmonary oedema (Adriani et al., 1967; Sarnoff, Berglund and Sarnoff, 1953) and heart failure (Brown et al., 1947) or cerebral haemorrhage.

Post-partum hypertension with cerebral haemorrhage has been observed in relation to ergometrine (Ringrose, 1962; Casady, Moore and Bridenbaugh, 1960). The combination of ergometrine and a pressor drug such as methoxamine or methylamphetamine may precipitate the syndrome (Scott, 1968). The pressor drugs act mainly on the alpha adrenoceptive vessels, the beta vessels being relatively unaffected. The addition of ergometrine causes constriction of both alpha and beta adrenoceptive vascular beds and may provoke arterial hypertension and pulmonary oedema in obstetric patients with normal cardiovascular systems. Cardiac disease, the toxaemias of pregnancy and chronic anaemia (Korner, 1959) increase the hazard.

The development of pulmonary oedema in parturient women after the administration of vasoconstrictive drugs may be prevented to some extent by general anaesthesia. Inhalational anaesthesia partly prevents the alpha vasoconstrictive effect of ergometrine, but only for the duration of anaesthesia as the vasoconstriction returns immediately after anaesthesia. It is interesting to note in many publications that pulmonary oedema in obstetric patients usually occurs after delivery or during the recovery from anaesthesia.

It is questionable whether ergometrine alone in therapeutic dosage will precipitate pulmonary oedema in the absence of predisposing causes. Heart disease, chronic anaemia and the toxaemias of pregnancy have been mentioned. Latent phaeochromocytoma has caused many maternal deaths from pulmonary oedema which often occurred soon after confinement (Gemmell, 1955; Walker, 1964; Martins, 1969). Latent or untreated thyrotoxicosis is possibly another predisposing factor.

Obstetric patients with latent phaeochromocytoma or thyrotoxicosis present problems especially when anaesthesia is required. The first sign of such a problem is usually a rapid ventricular or supraventricular tachycardia appearing immediately after the induction of anaesthesia. The cardiac involvement can be controlled by the immediate use of a beta adrenoceptor blocker (Johnstone, 1970). The use of a beta blocker may be dangerous if treatment is delayed until the appearance of pulmonary oedema: it may precipitate cardiac arrest. The immediate use of vasodilator drugs like phentolamine is indicated if pulmonary oedema is to be avoided (Fox et al.,
1969). Sympathetic blockade at other levels may be equally effective (Sarnoff, Goodall and Sarnoff, 1952). The sympathetic blocking action of chlorpromazine has been used successfully in the management of ergometrine-vasopressor induced hypertension (Casady, Moore and Bridenbaugh, 1960). Other drugs are available for this purpose (Green and Kepchar, 1959).

It is possible that the post-partum pulmonary oedema which follows the aspiration of vomit during anaesthesia (Mendelson, 1946) may be caused by a combination of factors and not by aspiration alone. It is strange that the lethal form of the syndrome occurs after delivery in obstetric patients with previously normal cardiovascular and respiratory systems (Parker, 1956). The aspiration of gastric secretion is not an obvious feature in all the reported cases. In many of the cases reported by Mendelson (1946) there was no clear evidence that vomiting occurred during anaesthesia and the pulmonary oedema was transient in all his patients.

Vomiting or regurgitation during anaesthesia may cause respiratory acidosis and hypoxia by partially obstructing the air passages. This provokes brisk activity of the sympathetic nervous system with constriction of the alpha adrenoceptive blood vessels and dilatation of the beta vessels. This is a cardiovascular crisis similar to that caused by the administration of excessive doses of vasopressor drugs (Adriani et al., 1967). When ergometrine is given in these circumstances the vasoconstriction becomes widespread. The conditions are set for the appearance of pulmonary oedema when the anaesthetic is withdrawn.

The use of ergometrine in obstetric patients seems to be undesirable in certain circumstances. These include the presence of cardiovascular, respiratory, and renal diseases, chronic anaemia and the toxaemias of pregnancy. Asphyxial episodes during anaesthesia, the simultaneous use of vasopressor drugs and other factors which increase sympathetic nervous activity in the alpha adrenoceptive vasculature like extreme fear, pain and physical exhaustion (Brod et al., 1959). The use of synthetic oxytocin is preferable to ergometrine in these circumstances.

The cardiovascular effects of synthetic oxytocin are the opposite of those of ergometrine. Oxytocin causes widespread dilatation of the alpha and beta adrenoceptive blood vessels. It predisposes to profound postural hypotension and sinus tachycardia when it is given to patients lying in a slightly head-up position. The postural hypotension is potentially dangerous because it is associated with electrocardiographic evidence of acute myocardial ischaemia presumably from transient failure of the coronary circulation (fig. 5, B). The effect of oxytocin does not last for more than 2 minutes. The hypertensive hazard of oxytocin is eliminated almost completely by ensuring that the patient is in the lithotomy position or lying supine with a slightly head-down tilt when the drug is injected intravenously in a single dose of 2 units or more. These postures ensure the venous return to the heart and the maintenance of the cardiac output and the blood pressure during the period of vasodilatation. The continuous intravenous infusion of relatively small doses of dilute oxytocin—0.5 unit a minute—is not associated with risk to the cardiovascular system (Ribot et al., 1964).

It is not improbable that oxytocin may have a mildly depressant effect on the myocardium. Katz (1964) observed that it suppresses the cardiac dysrhythmias associated with inhalational anaesthesia. The slight and very transient decreases in the amplitude of the finger volume-pulse and the disappearance of the dicrotic notch from the finger pulse-wave (fig. 8, B) when a dose of 5 units was given intravenously to patients anaesthetized with halothane may indicate a transient weakening of the myocardial contractile force. Another explanation may be a diminution in the venous return to the heart caused by the dilatation of the beta adrenoceptive vasculature which appears transiently after the intravenous injection. The effect is too brief to be important in patients with normal cardiovascular systems. If may be significant in patients with advanced heart disease (Robinson et al., 1967) and is avoidable by the continuous intravenous infusion of dilute solutions.

ACKNOWLEDGEMENTS

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REFERENCES


CARDIOVASCULAR EFFECTS OF OXYTOCIC DRUGS


LES EFFETS CARDIOVASCULAIRES DES MEDICAMENTS OCYTOCIQUES

SOMMAIRE
Les effets d'ergometrine 0.5 mg i.v. et d'oxytocine synthétique 5 unités i.v. sur les vaisseaux sanguins alpha- et beta-adrénergiques chez des patientes gynécologiques. Ergometrine cause une contraction des vaisseaux sanguins alpha et beta. On croit que le vasoconstriction étendue peut précipiter l'hypertension ou oedème pulmonaire post-partum chez les patientes obstétriques. L'oxytocine synthétique cause une dilatation transitoire des vaisseaux sanguins alpha et beta-adrénergiques et prédispose à une accélération hypertensive post-partum. L'effet hypotenseur d'oxytocine était beaucoup moins prononcé lorsqu'on avait administré le médicament aux patientes en position de lithotomie. On conclut que l'oxytocine est préférable à l'ergometrine chez les patientes obstétriques, chez qui une tendance à la vasoconstriction-alpha à été suscitée par un ou plusieurs des facteurs décrits.

DIE CARDIO-VASKULÄREN WIRKUNGEN VON OXYTOCIN-MITTELN

ZUSAMMENFASSUNG
Die Wirkungen von Ergometrin, 0,5 mg i.v., und von synthetischem Oxytocin, 5 Einheiten i.v., auf die Arterien- und Venenleitbahn beim gesunden und beim herzkranken Menschen untersucht. Ergometrin bewirkte eine Kontraktion der arteriellen und venösen Gefäße. Man konnte daran denken, dass die ausgedehnte Vasoconstriction eine postpartale Hypertonie oder ein Lungenödem bei geburtshilflichen Patientinnen provoziert, wenn andere Alpha-Vaso- konstriktor-Faktoren vorhanden sind. Synthetisches Oxytocin verursachte eine übermäßige Dilatation der Alpha- und Beta-Rezeptoren und

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**RESUMEN**

Han sido estudiados los efectos de 0,5 mg de ergometrina por vía i.v. y 5 unidades de oxitocina sintética por vía i.v. sobre los vasos sanguíneos alfa y beta adrenoceptores del antebrazo por medio de la pletismografía de volumen del pulso en cuarenta pacientes ginecológicas. La ergometrina produjo contracción de los vasos sanguíneos alfa y beta. Se cree que la extensa vasoconstricción pudiera precipitar la hipertensión posparto o edema pulmonar en pacientes obstétricas cuando hay presentes otros factores alfa vasoconstrictores. La oxitocina sintética causó una dilatación transitoria de los vasos sanguíneos adrenoceptores alfa y beta y predispuso a una intensa hipotensión postural. El efecto hipotensor de la oxitocina fue mucho menor cuando este fármaco fue administrado a pacientes en posición de litotomía. Se concluyó que la oxitocina es preferible a la ergometrina en pacientes obstétricas en las cuales la tendencia vasoconstrictora alfa ha sido inducida por cualquiera de los factores descritos.

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**EDINBURGH AND EAST OF SCOTLAND SOCIETY OF ANAESTHETISTS**

**Syllabus 1972–73**

1972

**SATURDAY, OCTOBER 21.** Combined Meeting with the Glasgow and West of Scotland Society in the Large Surgical Lecture Theatre, Royal Infirmary, Edinburgh. 5.00 p.m. (1) Professor J. J. Bonica, University of Washington, Seattle: "The effects of uterine contractions and maternal hypotension on intervillous perfusion". (2) Dr F. Cockburn, University of Edinburgh: "Nutrition, neurones and neonates".

**TUESDAY, NOVEMBER 14.** Dr R. T. Brittain, Head of Pharmacology Department, Allen and Hanburys, Ltd: "New non-depolarizing muscle relaxants".

**TUESDAY, DECEMBER 12.** Dr J. Alfred Lee, Southend-on-Sea: "Fifty years of anaesthesia in the U.K."

1973

**TUESDAY, JANUARY 9.** Dr C. F. Hider: Symposium on "Coronary artery surgery".

**TUESDAY, FEBRUARY 13.** Members' Night.

**FRIDAY, FEBRUARY 23.** Annual Dinner.

**TUESDAY, MARCH 13.** Dr Mark Mehta, Norfolk and Norwich Hospital: "Chronic pain".

**TUESDAY, APRIL 24.** Annual General Meeting.

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