THE OCULOCARDIAC REFLEX DURING STRABISMUS SURGERY*

GEORGE T MOONIE, M D ,† DONALD L REES, M D ,‡ AND
DENZIL ELTON, M R C S , D A †

A VARIETY of ocular stimuli produce variation in the heart rate with or without alteration in rhythm These changes are commonly referred to as the oculocardiac reflex Cardiac slowing after pressure on the eyeballs was independently reported by Aschner[1] and Dagnini[2] in 1908 Aschner[1] carried out a thorough investigation by animal experimentation, he divided the oculomotor, trochlear, abducens, facial, auditory, and trigeminal nerves and found that an intact trigeminal nerve was necessary for this reflex to occur He stated that the afferent impulse is carried by the ophthalmic branch of the trigeminal nerve and from here it passes to the vagal centres The efferent impulses to the heart are transmitted by the vagus nerve

The importance of the oculocardiac reflex was realized when Sorenson and Gilmore[3] in 1956 reported that traction on the medial rectus muscle caused cardiac arrest in a patient, which reverted to normal upon release of the muscle Bradycardia or cardiac arrhythmias occurred in each of 16 additional cases whenever the extraocular muscles were stretched Kirsh[4] in 1958 reported a fatal cardiac arrest which followed manipulation of the extraocular muscles This prompted him to investigate the oculocardiac reflex more fully Utilizing serial electrocardiography in a study of 50 cases, he found that a variety of ocular stimuli could produce significant electrocardiographic changes and that the depth and type of general anaesthetic or the use of local anaesthesia did not affect the sensitivity of the reflex Conversely, Reed and McCaughey[5] in 1962 noted that light anaesthesia in conjunction with controlled or assisted respiration favoured the production of the oculocardiac reflex As reported in a paper by Schamroth[6] in 1958, the application of pressure to the eyeballs of 28 conscious patients without local anaesthesia caused sinus bradycardia in all instances, plus a shift of the primary pacemaker in 18 Reid[7] in 1952 stated that in a normal heart, vagal stimulation produces no serious consequences, but that anaesthesia renders the heart as vulnerable to increased vagal tone as does the presence of diffuse organic lesions

Berler,[8] in a survey of the literature, reports that electrocardiographic changes occurred in 139 out of 265 patients, an incidence of 50 per cent However, only four patients of his own series of 25 demonstrated an oculocardiac reflex following ocular manipulation According to Rhode[9] the reflex is positive in 90 per cent of children under 15 years of age Fulton[10] states that vagal tone is at a

*Presented at the Annual Meeting of the Western Division of the Canadian Anaesthetists' Society at Vancouver, British Columbia, April 26–30, 1964

†Department of Anaesthesia, University of Alberta Hospital, Edmonton, Alberta
‡Department of Surgery, Division of Ophthalmology, University of Alberta Hospital, Edmonton, Alberta

maximum during adolescence and early adulthood. The physiological vagotonia probably accounts for the high incidence of bradycardia observed in children during strabismus surgery

The vagal effects on the heart have varied from simple sinus bradycardia described by Reed[5] in his series of 77 patients, to S A block, sinus arrest, wandering pacemaker, nodal and bigeminal rhythms reported by others [4 6 8]

There is considerable difference of opinion regarding the importance of this reflex Berler[8] and Taylor[11] consider it transient and of little significance However, a survey by Kirsh[4] of eight major hospitals in the United States led him to the conclusion that fatal cardiac arrest occurs once in 3500 cases of ophthalmic surgery Gartner and Billet[12] estimate a total of 45 deaths a year in the United States during or following eye surgery They mention that the majority of these were due to intubation or as a result of respiratory complications

Two methods have been advocated in the prevention of this reflex Kirsh,[4] Mallinson,[13] and Berler[8] find retrobulbar block to be completely effective This was not substantiated in reports by Reed[5] and Bosomworth,[14] who found intravenous atropine to be most effective Routine premedication with atropine has little effect in preventing the oculocardiac reflex according to several authors [5 8,14] The dose of intramuscular atropine necessary to produce almost complete vagal blocking in an adult is 2 0 milligrams, which is three times the usual premedicating dose [15 16] In addition Walton,[17] Deacock,[18] and Reed[5] have found that gallamine gives partial protection from the oculocardiac reflex

The study reported here was initiated by the high incidence of bradycardia which we have noted during strabismus surgery, and we decided to test the effectiveness of retrobulbar blocks in its prevention While observing one of our earlier patients we noted complete recovery from marked bradycardia when tension was inadvertently maintained on the medial rectus muscle for a period of 40 seconds The muscle hook was removed without further alteration in the rate A survey of the literature revealed that after precipitation of an oculocardiac reflex, normal rate and rhythm occurred following the release of muscle tension or pressure on the globe There were, however, three reports indicating that this reflex was self-limiting [11 17 19] We then directed our investigation towards the effect of continued extraocular muscle traction on the oculocardiac reflex

Method

The investigation was carried out on 66 healthy patients during surgical correction of strabismus They ranged in age from 1 to 32 years with a mean of 6 4 years This is an age group where reflex activity can be expected to be relatively high

Premedication was given one hour preoperatively and consisted of intramuscular meperidine, one milligram per pound, and atropine sulphate 0 2 to 0 4 milligrams depending on the weight and age of the child Patients over six years of age were induced with intravenous thiopentone, 2 5 milligrams per pound, followed by succinylcholine, 0 5 milligrams per pound After ventilation with oxygen, and when muscle relaxation was satisfactory, an endotracheal tube was

inserted Anaesthesia was maintained with a mixture of nitrous oxide 1 litre and oxygen 1 litre with 1 per cent halothane Ventilation was controlled using a semi closed circle absorber technique Children under the age of six were induced with nitrous oxide, oxygen, and halothane until they fell asleep At this time thiopentone was given intravenously and the remainder of the technique was the same as for patients over six years of age

Intrabronchial carbon dioxide concentration was monitored continually by means of a 3 8 F nylon catheter threaded through the endotracheal tube, and leading to a Beckman, Spinco model LB-1 medical gas analyser Random intrabronchial oxygen samples were measured by a Clarke electrode Arterial blood samples were taken where feasible and analysed for oxygen and carbon dioxide content using the Astrup technique These results corresponded closely to those obtained for the intrabronchial gas samples

A standard lead 2 electrocardiographic tracing was recorded throughout various manipulations, which included deliberate pressure on the eyeballs of 26 patients, intubation of 9 patients, and traction on the extraocular muscles of all patients The heart rate was calculated from the tracing before, during, and after the stimulus, on the basis of any two consecutive beats The incidence of reflex disturbances would have been much lower had the rate been measured over a 10-second interval, because slowing would have been much less apparent Cardiac arrest, if it had occurred, would in all probability have been preceded by only a few slowed beats [5 20] Therefore, any slowing of the pulse rate, if even for a few beats, is significant The oculocardiac reflex was considered to be present when there was either a conduction defect, or an abrupt decrease in heart rate by more than 10 per cent The time of onset and the duration of the reflex, as well as the time of maximum slowing of rate, were measured The types of arrhythmias and conduction defects were recorded, and the effect of repeated traction on the muscles was noted

Results

Pressure on the eyes of 8 conscious patients provoked a bradycardia in 2 During anaesthesia 5 of these patients, including the original 2, responded to extraocular muscle traction with a sinus bradycardia Bradycardia following orbital pressure developed in 5 of 18 anaesthetized patients Subsequently, extraocular muscle traction produced bradycardia in 10 of these patients, 5 of whom had already responded to orbital pressure Continuous electrocardiographic recording during intubation of 9 patients did not demonstrate any changes in cardiac rhythm, yet 6 of these patients developed an oculocardiac reflex following eye muscle traction

Table I summarizes the incidence of oculocardiac reflex Forty-four of the 66 patients developed changes in cardiac rhythm following extraocular muscle traction Fifteen of these were simple sinus bradycardia The remaining 29 patients developed, in addition to initial sinus bradycardia, various abnormalities including S A block, wandering pacemaker, nodal and bigeminal rhythm The mean time of onset of cardiac abnormalities was 1 9 seconds with a range of 1 to 5

TABLE I

CHANGES IN CARDIAC RHYTHM FOLLOWING EXTRAOCULAR MUSCLE TRACTION

Total number of cases	66	
Number of cases showing O C R	44	
Types of O C R observed		
Sinus bradycardia	44	100 %
S A block	2	4 5
Wandering pacemaker	15	36 4
Nodal rhythms	23	52
Bigeminal rhythm	2	4 5

seconds The mean maximum slowing of rate occurred at 5 3 seconds with a range of 2 to 15 seconds

Continuous eye muscle traction caused fatigue of the oculocardiac reflex with a return to the normal rate and rhythm in all cases Two of these are illustrated in Figure 1 The mean duration of cardiac disturbances was 48 4 seconds with a range of 8 to 288 seconds

The effect of repeated muscle traction on the duration of the oculocardiac reflex was observed It was noted that the shorter the period between the first and second muscle tractions, the first having been maintained until the reflex was fatigued, the shorter would be the duration of the oculocardiac reflex produced by the second Conversely, the longer the delay before the second traction was applied, the more likely the reflex was to become fully active once again This is illustrated in Figure 2, which represents the findings in 20 patients. The recovery of the reflex is shown here as a percentage This figure was obtained by measuring the duration of reflex changes after initial traction, and comparing this with the duration of reflex changes after a second traction which followed a measured rest interval These observations are not statistically of value because of the small number of patients involved, but they do give an indication of the effect of repeated extraocular muscle traction after varying rest periods

DISCUSSION

The sinoatrial node manifests the highest degree of rhythmicity in the heart and is responsible for the initiation of the heart beat The rate is normally modified by antagonistic tonic impulses mediated by the vagus and sympathetic nerves Impulses from the cardio-inhibitory centre of the brain stem reach the heart through the vagus nerve The vagal fibres enter the wall of the atrium and synapse with ganglion cells, the postganglionic fibres then supply both the S A and A V nodes and the base of the ventricles

The normal cardiac impulse originates in the S A node and is propagated through the wall of the atrium and reaches the A V node, from here it passes through the bundle of His, the right and left branches, into the ventricular muscle in a regular sequence Reflex stimulation of the cardiac vagi due to ocular manipulation causes an inhibitory effect on the heart, which is most marked on the upper portion of the S A node and the atrium This results in a decrease in rate or cessation of the heart beat, which may return to normal,

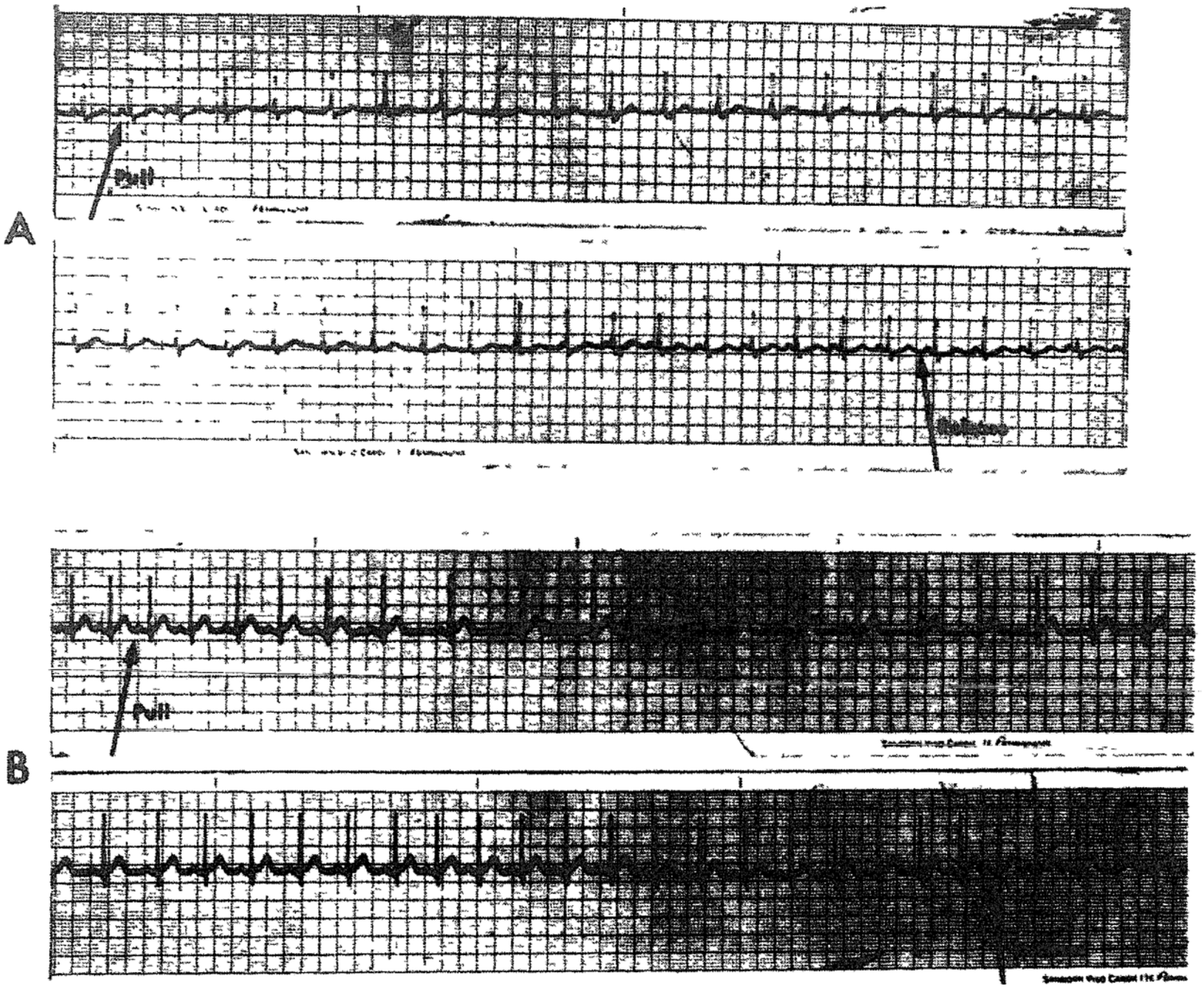

FIGURE 1 Fatigue of the oculocardiac reflex Continuous electrocardiographic recording of two patients who demonstrated an oculocardiac reflex following extraocular muscle traction, sustained traction causes a return to normal rate and rhythm

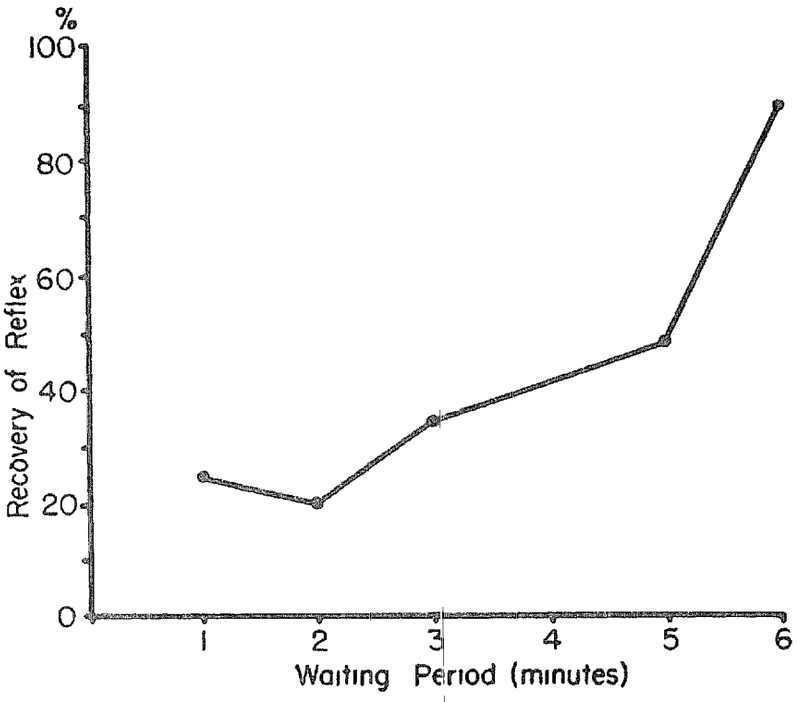

FIGURE 2 Recovery of the oculocardiac reflex after fatigue

or to a downward shift of the pacemaker to the tail of the S A node, or to the A V node Examples of these arrhythmias are shown in Figure 3

Sinus Bradycardia

Since the effect of increased vagal tone is most marked on the upper regions of the S A node and on the conduction time through the atria, the most common arrhythmia noted is sinus bradycardia This series showed that sinus bradycardia was present in all cases in which an oculocardiac reflex occurred Fifteen of these returned to normal and the remainder progressed to arrhythmias due to further depression of the pacemaker

S A Block

Sinoatrial block is due to an actual physiological block within the S A node There is a sudden decrease in rate, usually to half the previous rate, although, three, four, or more beats may be dropped, resulting in atrial standstill However, the P R interval is normal This is demonstrated in Figure 4

Wandering Pacemaker

Increased vagal tone depresses the rhythmicity of the S A node, resulting in a shift of the pacemaker to lower portions of the S A node or to various levels in the A V node There is a return to normal with decrease in vagal tone Shift of the pacemaker causes an alteration in the size, shape, and direction of the P waves, the P R interval, and the rate The P waves become flat as the pace maker moves into the tail of the S A node When the pacemaker shifts to the A V node the P R interval shortens, because of the proximity of the pacemaker to the ventricles This progressive shortening of the P R interval can continue until the P wave is buried in the QRS complex or shows up in a retrograde fashion between the QRS and T complexes Alteration in rate is due to suppression of one pacemaker while another takes over

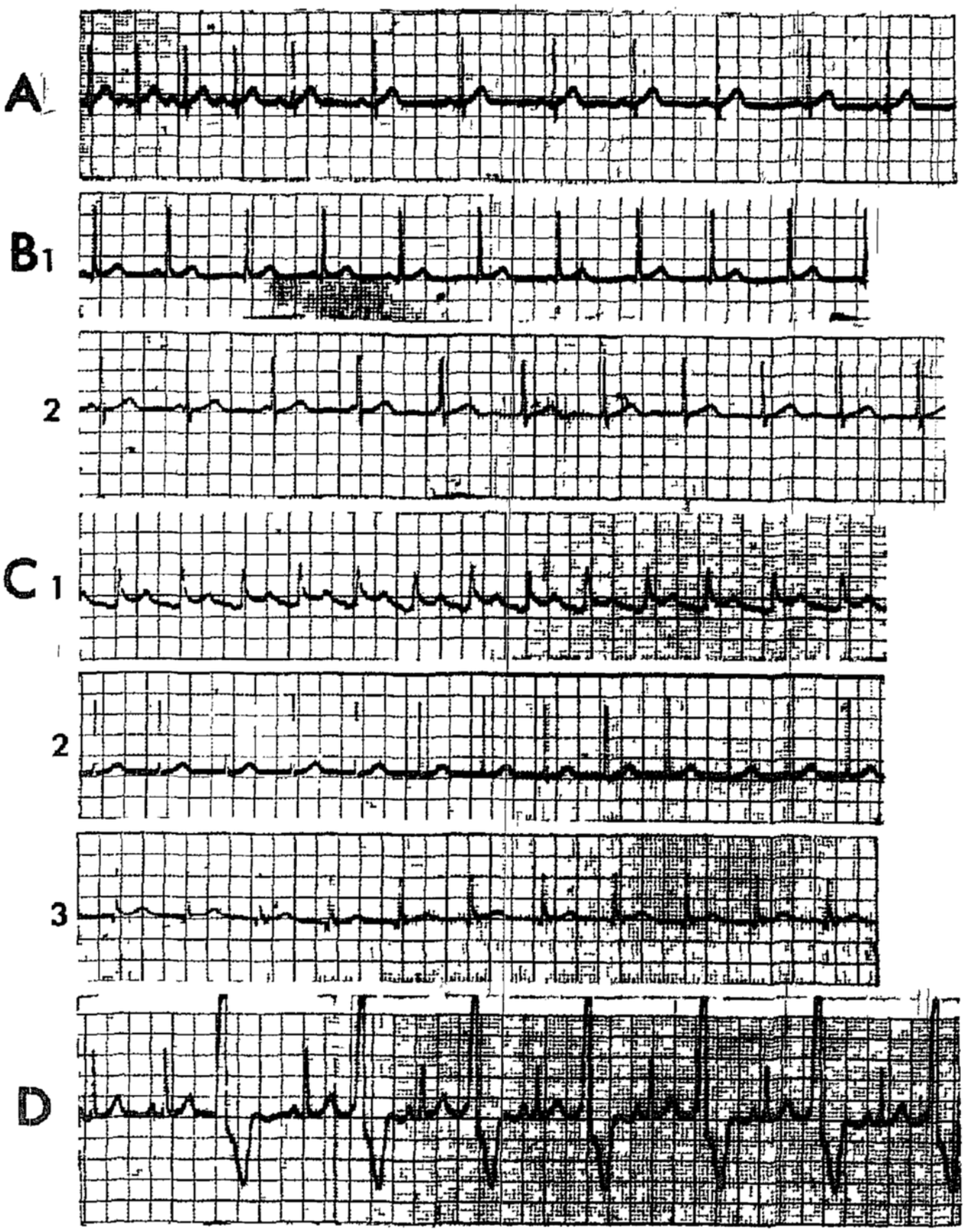

FIGURE 3 Cardiac arrhythmias due to vagal stimulation A sinus bradycardia B wandering pacemaker (1) in sinoatrial node (2) in atrioventricular node C A–V nodal rhythms (1) upper nodal (2) mid nodal (3) lower nodal D ventricular bigeminy

A V Nodal Rhythm

The A V node possesses a high degree of rhythmicity only slightly less than that of the S A node and will become the pacemaker of the heart if the rhythmicity of the S A node is depressed below that of the A V node

FIGURE 4 Sinoatrial block with A V nodal escape Note S A block following fifth QRS complex which is equal to two cycle lengths S A block equal to three cycle lengths occurs aft r the sixth QRS com plex this is terminated by nodal escape beats

The A V node is a relatively long structure and the electrocardiographic changes appear to depend on the level at which the impulse arises When the focus is at the atrial end the impulses travel down into the ventricle as well as up into the atria The distance to the atria is shorter so the impulse arrives there first causing the P wave to precede the QRS complex Since it is spreading in a retrograde fashion into the atria the P wave is inverted The focus is closer to the ventricles than is the S A node so the P R interval is shorter than normal this is upper nodal or coronary sinus rhythm In lower nodal rhythm the focus is at the ventricular end of the A V node and the ventricles are activated and contract before the atria The P wave will follow the QRS or ventricular complex but will be inverted because of the retrograde spread through the atria The P wave appears to be absent when the impulse arises in the centre of the node because it is buried in the QRS complex

Ventricular Bigeminy

Ventricular bigeminy occurs when a normal beat is followed regularly by a ventricular premature systole The premature ventricular systole is not preceded by an atrial contraction and is usually followed by a relatively long pause called the compensatory pause This pause may not be present if the heart rate is slow because the ventricles will have recovered from their refractory period by the time the next atrial impulse arrives

The mechanism of this phenomenon is debatable although the re entry theory receives most support An increase of vagotonia may cause depression of ventricular contractility [1] According to Bellet a given area of heart muscle may fail to respond to the normally conducted impulse but by the time the impulse has reached the terminal portion of the ventricle the depressed area is responsive and forms a new ectopic impulse This impulse excites the normal myocardium and forms an ectopic beat Schmoth reports that ventricular bigeminy following eyeball compression occurs most frequently in patients with heart disease receiving digitalis Lamb [3] reports that this arrhythmia may occur in patients following the release of breath after a prolonged inspiration

A V Nodal Escape

According to Bellet depression of the S A nodal resulting in periods of sinus arrest is a potentially dangerous situation The inherent rhythmicity of the A V node asserts itself following a period of atrial standstill and produces a single nodal escape beat which may be repeated [Fig. 4] The QRS complex

in nodal escape is normal, and P waves are absent This is a very important homoeostatic defence mechanism because in its absence a pacemaker in the ventricle would take control of cardiac rhythm at a much slower rate Failure of this ventricular escape would result in cardiac arrest Our observations indicated that the time of maximum slowing of rate is 5 3 seconds following muscle pull, and after this time the rate and rhythm return to normal slowly

It is the contention of many writers that hypercarbia,[24] hypoxia,[5,9] or both[11] make the heart more susceptible to vagal stimuli In 66 6 per cent of our patients, however, the oculocardiac reflex was noted while pCO_2 and pO_2 levels were within normal limits The effect of the anaesthetic agent in the production of arrhythmias must be taken into account Laver[25] reports an altered sequence of A V activity resulting in nodal rhythm during halothane anaesthesia We had four patients, not in this series, who exhibited nodal rhythm during halothane anaesthesia The rhythm reverted to normal when the concentration of halothane was decreased We must therefore appreciate the possible role of halothane in the production of the oculocardiac reflex

Fatigue

The heart rate and rhythm in patients exhibiting the oculocardiac reflex always returned to normal despite continued traction on the extraocular muscles The average time for this return to normal was 48 8 seconds with a minimum of 8 seconds and a maximum of 288 seconds Cessation of muscle tension following return to normal did not modify the rate or rhythm

Repeated muscle traction in these patients resulted in the reflex being sustained for shorter periods providing the rest intervals were short Arrhythmias also tended to be less marked than those that occurred after the original muscle traction The reflex approached the original as the rest interval lengthened This would indicate that fatigue of the reflex arc follows prolonged stimulation These observations agree with those of Planten,[19] who described the exhaustion of the oculocardiac reflex in a patient in whom repeated muscle traction caused a distinct vagal effect on three occasions and no effect was noted the fourth time

The site of fatigue is most probably in the central synapses of the brain It is generally accepted that repeated electrical stimulation of certain areas of the brain rapidly produces fatigue, which is manifested by fading of an induced response This response cannot be obtained until these areas of the brain regain their initial excitability, after a period of recovery[26] Delgado,[27] studying the circulatory effects of electrical stimulation of the brain, noted that prolonging the stimulation beyond 12–20 seconds was unnecessary because the effects disappeared despite the continuation of the stimulus Strom[28] observed similar results during electrical stimulation of portions of the hypothalamus He demonstrated fatigue when stimulation was prolonged beyond 30 seconds, or if it was applied repeatedly with only 10- to 15-second intervals of rest The signs of fatigue were not present when these intervals were extended to 20 to 30 seconds These observations are in keeping with our results and may explain to some extent the excellent results achieved with retrobulbar blocks Since only those patients who demonstrated an oculocardiac reflex were concerned, it may well

have been that the reflex was fatigued after the first muscle traction, and this was the reason for the apparent beneficial effect of the retrobulbar block

Conclusions

The oculocardiac reflex was noted in 44 of 66 patients undergoing strabismus surgery Disturbances occurred at the time of extraocular muscle traction in the absence of hypercarbia and hypoxia The oculocardiac reflex proved to be much more active than the vago-vagal reflex associated with intubation Arrhythmias were not produced during intubation of a group of 9 patients, but later 6 of these developed arrhythmias following extraocular muscle traction

Fatigue of the oculocardiac reflex following extraocular muscle traction was confirmed Repeated muscle traction with short rest periods either abolished or produced a modified response When the rest periods were lengthened, this response approached that produced by the original traction

The danger period is during the first few seconds of muscle traction when slowing of the heart rate is at a maximum Atrial standstill followed by cardiac arrest could occur at this time This critical period may have passed by the time an arrhythmia could have been detected by usual clinical methods Therefore, prevention of the reflex should be instituted before surgery Since a retrobulbar block could precipitate an oculocardiac reflex,[8] and its effectiveness is in doubt, perhaps the administration of atropine intravenously a few minutes before ophthalmic surgery would be the method of choice

Résumé

Toute une variété de stimuli oculaires peuvent produire une variation de la vitesse cardiaque avec ou sans modification du rythme Un tel réflexe peut conduire à l'arrêt cardiaque Les auteurs ont étudié ce réflexe au cours de la chirurgie correctrice du strabisme chez 66 malades en bon état. Chez 44 de ces malades, ce réflexe oculocardiaque a été observé Les troubles sont apparus au moment de la traction sur les muscles extraoculaires et, cela, alors qu'il n'existait ni hypoxie ni hypercarbie Le réflexe oculocardiaque nous a semblé beaucoup plus actif que le réflexe vago-vagal observé au cours de l'intubation Au cours de l'intubation chez neuf malades, nous n'avons pas observé d'arythmie mais, plus tard, six d'entre eux, à la suite de la traction sur les muscles extraoculaires, ont présenté de l'arythmie

Nous avons observé une fatigue, un épuisement du réflexe oculocardiaque produit par traction sur les muscles extraoculaires Des tractions répétées, après de cortes périodes de repos, produisaient soit des réponses modifiées, soit une abolition du réflexe Si les périodes de repos étaient prolongées, la réponse au réflexe redevenait semblable à celle qui était observée à la suite de la traction originale

La période dangereuse est au cours des quelques secondes de traction sur les muscles, alors que le ralentissement du cœur est à son maximum C'est à ce moment que l'arrêt cardiaque peut survenir après l'arrêt auriculaire Cette

période critique peut déjà être terminée avant qu'une arythmie puisse être dépistée par les méthodes cliniques courantes En conséquence, c'est avant la chirurgie qu'il faut prendre des précautions contre les réflexes

Etant donné qu'un blocage rétrobulbaire peut provoquer un réflexe oculocardiaque[8] et que, de plus, ses effets demeurent douteux, il est probable que l'atropine administrée par voie endoveineuse, quelques instants avant l'opération pour la chirurgie oculaire, serait la méthode de choix

ACKNOWLEDGMENTS

We wish to express our appreciation to Dr R E Rossall, Assistant Professor, Division of Cardiology, Department of Medicine, Dr E A Gain, Director, Department of Anaesthesia, and Dr J W R McIntyre, Assistant Professor, Department of Anaesthesia, for their help in the preparation of this manuscript, and to Mr Donald Quinn for his technical assistance

REFERENCES

1 ASCHNER, B Ueber einen Bisher noch nicht beschriebenen Reflex vom Auge auf Kreislauf und Atmurg Verschwinden des Radialispulses bei Druck auf das Auge Wien-klin Wchnschr *21* 1529 (1908)
2 DAGNINI, G Intorno ad un Riflesso Provocato in Alcuni Emplegici Collo Stimulo Della Cornea e colla Pressione Sul Bulbo Oculare Bull Sci Med *8* 380 (1908)
3 SORENSON, E J & GILMORE, J E Cardiac Arrest during Strabismus Surgery Am J Ophth *41* 748 (1956)
4 KIRSH, R E , SAMET, P , KUGEL, V , & AXELROD, S Electrocardiographic Changes during Ocular Surgery and Their Prevention by Retrobulbar Injection A M A Arch Ophth *58* 348 (1957)
5 REED, H & MCCAUGHEY, T Cardiac Slowing during Strabismus Surgery Brit J Ophth *46* 112 (1962)
6 SCHAMROTH, L Electrocardiographic Effects of Eyeball Compression Am J Cardiol *2* 321 (1958)
7 REID, L C , STEPHENSON, H E JR , & HINTON, J W Cardiac Arrest Arch Surg *64* 409 (1952)
8 BERLER, D K The Oculocardiac Reflex Am J Ophth *56* 954 (1963)
9 RHODE, J , GROM, E , BAJARES, C , ANSELMI, A , CAPRILES, M , & RIVAS, C A Study of Electrocardiographic Alterations Occurring during Operations on the Extraocular Muscles Am J Opth *46* 367 (1958)
10 FULTON, J F A Textbook of Physiology, 17th ed , p 688 Philadelphia, Saunders (1955)
11 TAYLOR, C , WILSON, F , ROESCH, R , & STROELTING, V K Prevention of Oculocardiac Reflex Anesthesiology *24* 646 (1963)
12 GAFTNER, S & BILLET, E A Study of Mortality Rates during General Anaesthesia for Ophthalmic Surgery Am J Ophth *45* 847 (1958)
13 MALLINSON, F B & COOMBES, S K A Hazard of Anaesthesia in Ophthalmic Surgery Lancet i 574 (1960)
14 BOSOMWORTH, P P , ZIEGLER, C H , & JOCOBY, J The Oculocardiac Reflex in Eye Muscle Surgery Anesthesiology *19* 7 (1958)
15 CRAIG, F N Effects of Atropine, Work and Heat on Heart Rate and Sweat Production in Man J Appl Physiol *4* 826 (1952)
16 CULLUMBINE, H , MCKEE, W H , & CREASEY, N H Effects of Atropine Sulphate upon Healthy Male Subjects Quart J Exptl Physiol *40* 309 (1955)
17 WALTON, F A Reflex Bradycardia in Ophthalmic and Oropharyngeal Surgery Canad Anaesth Soc J *4* 414 (1957)
18 DEACOCK, A R DE C & OXER, H F The Prevention of Reflex Bradycardia during Ophthalmic Surgery Brit J Anaesth *34* 451 (1962)

19 PLANTEN, J TH Oculocardiac Reflexes in Operations for Strabismus Ophthalmologica *135* 287 (1958)
20 BELLET, S Clinical Disorders of the Heart Beat, 2nd ed, p 128 Philadelphia Lea and Febiger (1963)
21 *Ibid*, p 66
22 *Ibid*, p 226
23 LAMB, L E, DERMKSIAN, G, & SARNOFF, C A Significant Cardiac Arrhythmias Induced by Common Respiratory Maneuver Am J Cardiol *2* 563 (1958)
24 YOUNG, W G JR, SEALY, W C, HARRIS, J, & BOTWIN, A The Effects of Hypercapnia and Hypoxia on the Response of the Heart to Vagal Stimulation Surg Gynec & Obst *93* 51 (1951)
25 LAVER, M B & TURNDORF, H Atrial Activity during Halothane Anaesthesia in Man Anesthesiology *24* 133 (1963)
26 DELGADO, J M R Prolonged Stimulation of the Brain in Awake Monkeys J Neurophysiol 22 458 (1959)
27 —— Circulatory Effects of Cortical Stimulation Physiol Rev Supp 4, *40* 146 (1960)
28 STROM, G Vasomotor Responses to Thermal and Electrical Stimulation of the Frontal Lobe and Hypothalamus Acta Physiol Scand *20*, Supp 70 83 (1950)