THE MISMANAGEMENT OF SUXAMETHONIUM APNOEA

BY

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SUMMARY

A case of prolonged apnoea after suxamethonium is described in which misleading evidence was obtained from studies of neuromuscular conduction with a Medilec nerve stimulator. Estimations of esterase activity, dibucaine number and fluoride number were within the range expected in individuals possessing only "atypical" serum cholinesterase. Electromyographic studies showed a normal response to decamethonium, but a severe and prolonged neuromuscular block after suxamethonium. The block was apparently depolarizing for at least 1½ hours. After a review of other prolonged apnoeas the role of neostigmine in the prolongation of the apnoea is highlighted, and it is suggested that the demonstration of post-tetanic facilitation does not necessarily imply that the block will be improved by anticholinesterase drugs. Two types of theory are outlined to explain this type of case, and precautions against inadvertently making matters worse are suggested.

"Suxamethonium apnoea" is no longer news. Recent papers by Churchill-Davidson and Wise (1960) and Bush (1961) give the impression that it is not even a clinical problem, provided that a logical and scientific approach to diagnosis and treatment is adopted. The purpose in recording another case of this genetically determined abnormality is to demonstrate that even under favourable circumstances, when the requisite diagnostic apparatus is available, difficulties can still be encountered and even possibly engendered, and to present some electromyographic findings in this case which may help to explain these difficulties.

CASE REPORT

A male patient, R.M., aged 36, was admitted for submucous resection of the nasal septum. There was no significant previous medical history. Premedication consisted of pethidine 100 mg and hyoscine 0.4 mg, and the patient arrived in the anaesthetic room drowsy, but easily rousable and co-operative. Anaesthesia was induced with thiopentone 400 mg and was followed by suxamethonium 100 mg. After passing a cuffed orotracheal tube the patient was ventilated with nitrous oxide (6 l/min) and oxygen (2 l/min) with 0.5 per cent halothane (Fluotec). After 20 minutes, when respiration had not recommenced, it was assumed that apnoea was due to suxamethonium sensitivity. Halothane was discontinued, and the operation was performed while ventilation was continued with nitrous oxide and oxygen.

At the end of the operation, approximately 1 hour after the suxamethonium had been given, neuromuscular conduction was tested with a Medilec nerve stimulator applied to the ulnar nerve (Christie and Churchill-Davidson, 1958). There was pronounced weakness but post-tetanic facilitation was easily demonstrated. Atropine 0.6 mg and neostigmine 1.25 mg were given. The only detectable effect of this was a worsening of the neuromuscular conduction and abolition of post-tetanic facilitation.

Ventilation was continued for another hour. At this time the mixed venous Pco₂ was 43 mm Hg (using the rebreathing method of Campbell and Howell (1960)) and post-tetanic facilitation could again be demonstrated, although not so markedly. In view of the previous experience, the short-acting anticholinesterase drug, edrophonium, was administered in a dose of 10 mg instead of neostigmine. There was a brief improvement and a few random movements of limbs took place. Within a few minutes the neuromuscular block was no better than before.

Ventilation was continued for a further 1½ hours. It was now 3½ hours since the suxamethonium had been administered and the neuromuscular block was still pronounced. Post-tetanic facilitation, although not marked, was again demonstrated to the satisfaction of a fresh observer. Mixed venous Pco₂ was still normal. It was decided to administer edrophonium 10 mg again, and to reinforce this with nikethamide 5 ml on the supposition that there might be a concomitant abnormal central depression from thiopentone. Respiration of a curarized pattern commenced, but ceased within half a minute, and artificial ventilation was resumed.

The unwisdom of administering two drugs in close succession was now apparent. However, since a further dose of nikethamide failed to initiate respiration, it was concluded that the anticholinesterase had been the beneficial agent and the patient was given atro-
Suxamethonium 2 mg was injected intravenously and the e.m.g. recorded every half-minute. After 2 minutes there had been no detectable change in the e.m.g. and the patient had no abnormal subjective sensations. Suxamethonium 4 mg was then given and a progressive paralysis developed over the next few minutes, reaching a maximum clinically in about 4 minutes, and electromyographically in about 8 minutes, when the trace was virtually flat, as in figure 2n. The paralysis developed in the order customarily described as typical of increasing doses of non-depolarizing neuromuscular blocking agents, and without muscle fasciculation; that is to say, he first complained of diplopia, then of being unable to move his eyes, and speech became slurred. Difficulty in swallowing was experienced and then heaviness of the arms and legs; he then became unable to speak and failed to move his limbs at all when requested. Fortunately, the diaphragm was little affected, and he remained apparently fully oxygenated breathing air. Eight minutes after the second injection of suxamethonium, edrophonium 10 mg was given. There was no clinical improvement and no change on the electromyograph, thus confirming that, despite the slow and differential nature of the block and the absence of muscle fasciculation, this was a depolarizing type of block. (Unfortunately, the electromyograph stimulator available to us was not able to deliver a tetanic rate of stimulus.) During the course of the next 8 to 10 minutes the intensity of the neuromuscular block diminished, commencing with the facial muscles, quickly followed by the laryngeal and the extra-ocular eye muscles. At this time, about 20 minutes after the suxamethonium, the patient presented the picture of total paralysis of the limbs, with a virtually flat e.m.g. trace from the thenar muscles (fig. 2e), yet able to converse freely and with full recovery of the eye muscles. He persisted in this state for a further 20 minutes, when some recovery was noticeable in the proximal muscle groups of the limbs. On using the stimulator a muscle twitch occurred in the upper arm under the neutral lead, although there was still complete absence of neuromuscular conduction in the hand.

After a further 20 minutes, making now 1 hour in all, action potentials began to be recordable from the small muscles of the hand (fig. 2f), although the patient could not perform any voluntary action with them. Voluntary movements reappeared 15 minutes later, but the e.m.g. trace
A. Normal trace.
B. After 2 mg decamethonium.
C. After 2.5 mg decamethonium (total dose).
D. After 10 mg edrophonium.
E. 30 minutes later.

A. Normal trace.
B. 4 minutes after suxamethonium (6 mg).
C. 6 " " "
D. 10 " " "
E. 40 " " "
F. 1 hour " " "
G. 1½ hours " "
H. 3 minutes later after edrophonium (10 mg).
after 1½ hours (figure 2c) still had an amplitude barely one-fifth of that recorded prior to the suxamethonium, and there was marked muscle weakness. At this time edrophonium 10 mg was again given, and failed to produce any change in either the weakness or the e.m.g. (fig. 2d); thus demonstrating that the block still had a depolarizing pattern—in the hand at least. Unfortunately, for reasons unconnected with this experiment, it was not possible to continue recording the e.m.g. any longer. At this time the patient described his feelings graphically, “as though I was moving my arms through water”, and it was another 1½ hours before he felt completely normal. (It may be noted in passing that the failure to respond to edrophonium was not due to deterioration of the drug, since there was increased salivation for approximately 5 minutes each time it was given.)

### BIOCHEMICAL INVESTIGATION

Esterase activity, dibucaine (cinchocaine) number and fluoride number of the patient’s serum were estimated for us by Professor H. Harris and Dr. M. Whittaker, of King’s College, London. The results are set out in the first line of table I, and are those expected in an individual with only atypical serum cholinesterase (Kalow and Staron, 1957; Harris and Whittaker, 1961).

None of the patient’s blood relations had ever had an anaesthetic and, in the interests of preventive medicine, serum from the patient’s mother and from both of his children was also investigated. The results are shown in table I also and place all of them in the range expected of heterozygous individuals, who have both typical and atypical serum cholinesterase. Should they receive suxamethonium it is quite probable that they would react normally, or at worst, have an apnoea of only a few minutes duration.

### DISCUSSION

The investigation of the serum cholinesterase activity in this patient and his relatives reveals the pattern most frequently found with this biochemical abnormality. It adds nothing new to the genetic aspect of suxamethonium apnoea, which subject has been recently reviewed by Harris and Whittaker (1962). It is unnecessary, therefore, to discuss it in detail in the present context, except to say that the results are consistent with this patient being homozygous for the atypical gene and, therefore, possessing only an atypical serum cholinesterase, unable to metabolize suxamethonium in clinical doses.

Several aspects of the management of the clinical problem call for further comment. Firstly, it is necessary to examine the possibility that the actual method of treatment adopted may itself have magnified the problem and prolonged the apnoea. From various case reports the impression is gained that the “natural” duration of apnoea in patients possessing this genetic abnormality varies between about 45 and 90 minutes. It will be determined largely by the total dose administered.

Kalow and Gunn (1957) measured the duration of apnoea after various doses of suxamethonium ranging from 25 mg to 1000 mg given to patients having courses of electroconvulsive therapy. They found a linear relationship between the logarithm of the dose of suxamethonium and the logarithm of the duration of the apnoea, and the slope of this line was characteristic for each individual. From this line they determined the duration of apnoea that would have followed a dose of 100 mg of suxamethonium. In the patients with low dibucaine number this ranged from 50 to 64 minutes.

Suxamethonium sensitivity has occurred in two other patients in this hospital within the last 6 months, the durations of apnoea being 60 and 75

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**Table I**

<table>
<thead>
<tr>
<th>Esterase activity number</th>
<th>Dibucaine number</th>
<th>Fluoride number</th>
</tr>
</thead>
<tbody>
<tr>
<td>R. M. (Patient)</td>
<td>48</td>
<td>26</td>
</tr>
<tr>
<td>C. M. (Daughter)</td>
<td>92</td>
<td>64</td>
</tr>
<tr>
<td>M. M. (Daughter)</td>
<td>102</td>
<td>64</td>
</tr>
<tr>
<td>D. I. M. (Mother)</td>
<td>87</td>
<td>63</td>
</tr>
</tbody>
</table>

Typical average values:

1. Normal (usual phenotype) 105 80 61
2. Atypical (homozygous atypical phenotype) — 22 23
3. Intermediate (heterozygous) — 62 48

Esterase activities were determined spectrophotometrically with benzoyl-choline as substrate (Kalow and Genest, 1957), and are expressed in arbitrary units.

Dibucaine numbers were determined by the method of Kalow and Genest (1957) and fluoride numbers by the method of Harris and Whittaker (1961). The typical average values are taken from data in Harris and Whittaker (1961).
minutes. The neuromuscular block was reversed on each occasion with neostigmine. Of the seven cases described by Bush (1961), all of which were atypical homozygous, five conform to this clinical pattern, as do six of the seven cases described by Argent, Dinnick and Hobbiger (1955).

An examination of cases that failed to conform to this pattern reveals interesting parallels. In one of the two really prolonged apnoeas that occurred among Bush's seven cases, edrophonium was given comparatively soon after the suxamethonium, while the patient was apnoeic. Apnoea persisted for 3 hours. In the other, neostigmine failed to maintain an initial improvement, even though a non-depolarizing type of block had been previously demonstrated, and the child had to be re-intubated and ventilated for a further 60 minutes. Amongst the cases reported by Argent, Dinnick and Hobbiger (1955), occurred one in which 6 hours passed before ventilation was judged to be normal. Although the picture was complicated by a probable concurrent carbon dioxide narcosis, this patient also received neostigmine whilst apnoeic, only 35 minutes after suxamethonium. Harper (1952) reported an instance of apnoea lasting 3½ hours. His patient too received neostigmine, whilst still apnoeic, 45 minutes after the suxamethonium.

Abrams and Ginsberg (1960) reported another case which fits into the same pattern, the first dose of an anticholinesterase being given about ¾ hours after the suxamethonium, again while the patient was still apnoeic. Three and a half hours later, after a blood transfusion, the block was on clinical evidence typically non-depolarizing, and edrophonium and neostigmine reversed it quite satisfactorily. Porteous (personal communication, 1962) observed apnoea in a man aged 66 who received suxamethonium 100 mg for bronchoscopy. After 75 minutes of apnoea, random muscle twitches occurred, associated with weak diaphragmatic movements. Edrophonium 10 mg improved ventilation for 5 to 10 minutes but it remained of a very curarized pattern. Thirty minutes later the muscle movements were stronger but this time administration of edrophonium had no clinically detectable effect. Atropine and neostigmine likewise caused no improvement, and it was a further 4 hours before he ceased to require assisted ventilation.

Unfortunately, there was available no nerve stimulator or means of estimating Pco₂. The patient's serum showed an activity of 20 units, and dibucaine and fluoride numbers were also 20. (Methods and standards as in table I.)

It would seem, then, that some patients with a low esterase activity and a low dibucaine number do not follow the clinical pattern of recovery usually associated with this condition. Two possibilities exist: either these patients are even more atypical, in some way as yet undetected, or else their abnormal clinical courses are related to the treatment they receive. What may be of particular significance is the fact that all the patients who had very prolonged block received an anticholinesterase drug early in the course of resuscitation, either before any spontaneous muscle activity had commenced, or soon after it was detected. In the latter case, after a short initial improvement, the neuromuscular block worsened, and persisted for an hour or more. In contrast, the patients whose apnoea was of "normal" duration either received no anticholinesterase drug, or received it some time after the neuromuscular block had shown evidence of improving spontaneously. In these cases there was an immediate and lasting improvement in the neuromuscular block when the anticholinesterases were given.

Since the anticholinesterase drugs act as suxamethonium extenders in normal individuals (McCaul and Robinson, 1962) it is natural to suppose that they could be responsible for producing a very prolonged neuromuscular block in suxamethonium-sensitive individuals. Indeed, this was suggested by Bush (1961) in one of his cases and by Abrams and Ginsberg (1960) in theirs. If this is true, it would seem that it is in no way connected with any effect on the breakdown of suxamethonium by serum cholinesterase. In suxamethonium-sensitive individuals, atypical cholinesterase probably plays no part in the elimination of the drug, since there is virtually no hydrolysis of suxamethonium by this enzyme at the normal pH of blood and in the concentration at which it would be present (Kalow, 1959). Inhibiting the enzyme with an anticholinesterase drug could, therefore, have little effect.

In support of this view one may instance the patient reported by Hart and Mitchell (1962), in whom no cholinesterase activity was ever detected. Yet weak, spontaneous respiration commenced after 75 minutes, which is comparable with the
THE MISMANAGEMENT OF SUXAMETHONIUM APNOEA

duration of apnoea in other "typical" cases. It is of particular interest in this context that, despite a short period of improvement after edrophonium 10 mg, the neuromuscular block in this patient, also, worsened when neostigmine was given, and persisted for 4.5 hours. The parallel with the case reported here is again very striking. Although it was 1.5 hours after the suxamethonium when neostigmine was given, there was only a minimal flicker of activity from the diaphragm at that time. The peripheral nerve stimulator was used and showed a severe neuromuscular block, but unfortunately no mention is made of the presence or absence of post-tetanic facilitation.

That there should be two basically different responses to anticholinesterases after prolonged exposure to suxamethonium is no more than one expects. Churchill-Davidson and Christie (1959) have shown that when suxamethonium is given in repeated doses, or continuously over a long period, the endplate region undergoes a change, and the initial depolarizing block evolves into one behaving in some respects like a non-depolarizing one (the phase II block). However, this change must take place over a period of time, and different muscle fibres could be in different stages at any one moment. There must be, in fact, a stage in which a "mixed" block is giving way to a "dual" block. The clinical picture at this time would be dependent on which muscles were most advanced in this evolution. It has been stated before (Wylie and Churchill-Davidson, 1960), and is confirmed by the present case, that some muscles can be paralyzed when others have clinically recovered. The electromyographs in the case described here show, furthermore, that a considerable degree of recovery can take place, and yet the block may remain apparently typically depolarizing in type, even as long as 3 hours after a dose of only 6 mg of suxamethonium.

The clinical problem would thus appear to resolve itself into the need to determine what stage in the evolution of the neuromuscular block has been reached, and when termination of the apnoea is required, to avoid giving anticholinesterase drugs while any depolarizing block is still present. This was the approach of Churchill-Davidson and Wise (1960) who based this critical decision on the satisfactory demonstration of an improvement in neuromuscular conduction after tetanic stimulation of a motor nerve (post-tetanic facilitation). A simple apparatus has been described by Christie and Churchill-Davidson (1958) for this purpose. They stated that the presence of this phenomenon was definitely indicative of a non-depolarizing neuromuscular block, and that the injection of anticholinesterases at this time must be beneficial. When such a block has been caused by a curare-like drug, this is undoubtedly a logical corollary. However, there is some evidence (Taylor, 1959) that the non-depolarizing type of block which develops in these cases is not the same as that which follows the administration of a curare-like drug, and the perverse response described here should perhaps have been less unexpected than it was. Several instances of "dual block" due to suxamethonium have been promptly terminated by neostigmine after the satisfactory demonstration of post-tetanic facilitation, and anticholinesterase drugs were confidently given in the present instance because of that evidence. The worsening of the block in these circumstances was an unpleasant surprise, and largely prompted this communication. For, if our observations are correct, this test can seriously mislead in this situation.

It would be unwise to build too much on this isolated observation, even though it was demonstrated to several observers, one of whom at least had had considerable experience with this test. Although this is the first time that it has been alleged that post-tetanic facilitation could be associated with a neuromuscular block that is worsened clinically by anticholinesterases, it must be remembered that, as yet, few reports of apnoea following suxamethonium have appeared in which this test has been performed. However, consideration of one of the cases in the report by Bush (1961) leads to the supposition that it may have occurred in other hands. In case 3, a non-depolarizing block was demonstrated while the patient was apnoeic. Despite this evidence, anticholinesterase drugs were not given until spontaneous ventilation occurred some 30 minutes later. Atropine and neostigmine were then given. After a short initial improvement, the block relapsed and the patient had to be ventilated for a further 60 minutes. This is hardly the normal course of events in a straightforward non-depolarizing block.

Are these observations valid? Is it possible to visualize a state of neuromuscular block which...
exhibits post-tetanic facilitation of conduction and yet worsens after an anticholinesterase drug has been given? The changes that lead to the emergence of a non-depolarizing type of block are still inadequately understood and a subject of controversy between experts. Within these limitations two types of explanation can be suggested.

Firstly, it seems possible that the various muscle fibres within a group could be in different stages of transition. The block in some fibres within a group might have developed sufficiently to give a clinically detectable post-tetanic facilitation when a large group is tested by stimulating a motor nerve; yet at the same time the majority of the fibres might still behave as though depolarized.

Alternatively, one must postulate that the response is related to the state of the endplate region, such that a high local concentration of normal transmitter substance prolongs the period of neuromuscular block.

There is supporting evidence for this in the work of Thesleff (1955) who showed that maintaining a local concentration of depolarizing drugs, including suxamethonium and acetylcholine, in the endplate region for more than 15 minutes rendered them refractory to depolarization. Neostigmine would not restore transmission. This condition only slowly recovered. Prolonged depolarization with suxamethonium could have a similar effect, and subsequently raising the local concentration of acetylcholine with anticholinesterases would produce a similar, slowly recovering refractory state. The words of Paton (1958) on this subject are so apposite that they are worth quoting: "It seems to be implied that a depolarizing drug may produce, in occasional patients, or under particular condition, one of two states; either one in which . . . anticholinesterases may be helpful; or one in which the endplates have become so refractory that acetylcholine itself produces further neuromuscular block, in which case anticholinesterases may actually make things worse."

In the light of the case reported here, and that of Abrams and Ginsberg (1960), it is suggested that these different responses may, in fact, take place successively in the same patient, and that there is a transitional stage before the development of a typical phase II block.

The characteristics of this stage would seem to be some, or all of the following features.

An increase in local concentration of acetylcholine succeeds at first in breaking through the endplate resistance, but this is only temporary and the endplate shortly becomes more refractory, and this state persists for upwards of 45 minutes. Clinically, therefore, there may be an improvement in the block when anticholinesterase drugs are given, but this is not maintained. If edrophonium is given, this short improvement may be mistakenly equated with the short duration of action of the drug, and so apparent improvement after this drug may be followed by deterioration after neostigmine. Similarly, short tetanic bursts of stimuli to a motor nerve give a detectable temporary improvement in the force of contraction. If there is sufficient stimulus to respiration there will be a tracheal tug. This state gradually changes into the one usually described as the phase II block. Post-tetanic facilitation is then much more pronounced; there is a curarized pattern of ventilation provided that arterial Pco₂ is normal or a little raised, and provided that there is no central depression of respiration. Anticholinesterases now produce a long-lasting improvement.

What are the determinants of this process and how do they affect the time taken to progress through the stages?

Clearly, they must vary from case to case. The factors affecting the duration of apnoea in suxamethonium-sensitive individuals include total dose (Kalow and Gunn, 1957), redistribution (Argent, Dinnick and Hobbiger, 1955), and non-enzymatic alkaline hydrolysis (Foldes, 1957). Churchill-Davidson and Richardson (1952) have shown that active contraction of muscles, by increasing local muscle blood-flow, has a marked effect on the duration of action of decamethonium, and this is probably one of the most important factors in this type of case also. Although impossible during the stage of complete depolarization, it becomes progressively possible as the block changes in character, and it may be of significance that the longest periods of apnoea have occurred when neostigmine was given before any muscles had undergone active contraction. Thus, the advice of Churchill-Davidson and Wise (1960), that it is unnecessary to wait for spontaneous ventilation before giving neostigmine, is probably more technically correct than practically expedient in this situation. The caution shown by Bush (1961) in waiting for ven-
tilation to commence, even though post-tetanic facilitation could be demonstrated, may well have saved him some hours of unnecessary artificial ventilation.

To sum up: from the author’s experience and investigation of this patient, and from a review of the recorded experiences of others, it seems that anticholinesterase drugs markedly prolong the neuromuscular block in suxamethonium-sensitive individuals, that is those with only atypical cholinesterases, if given too early in the recovery phase. The length of time which is “too early” depends on many variables, but may be as long as 1½ hours. Contrary to expectation, the termination of this phase is not coincident with the earliest demonstration of post-tetanic facilitation of conduction, or necessarily with the onset of a primitive type of respiration. Of the two, post-tetanic facilitation is probably detectable earlier.

From the clinical standpoint it is suggested that it is time to modify the advice of Churchill-Davidson and Wise (1960) on the management of prolonged apnoea. If facilities for testing neuromuscular conduction are available the urge to administer an anticholinesterase drug must be restrained until post-tetanic facilitation is markedly present. In any case, some spontaneous, if inadequate, respiration must be present together with a near normal arterial, or mixed venous Pco₂. If edrophonium is given as a diagnostic test, the improvement must be unequivocal, producing a normal pattern of respiration, and maintained for at least 5 minutes before giving neostigmine. It remains true that adequate patience is the best and least toxic therapeutic agent, although the use of fresh plasma, or blood would be likely to terminate the apnoea, if it could be obtained in time.

From the scientific point of view one can only complete the quotation from Paton (1958) given earlier: “... it remains for the application of electrophysiological techniques to patients under anaesthesia to discover what in fact happens, and no doubt, to reveal still other influences at work”.

ADDENDUM

Since submitting this article, my attention has been drawn to a report by Ruddell (1962) of three instances of apnoea after suxamethonium occurring in three siblings. All three children received neostigmine at some stage and in two there was a satisfactory reversal of muscle weakness. These patients received neostigmine approximately 1½ hours and 2½ hours, respectively, after the suxamethonium. The third child received neostigmine whilst apnoeic 70 minutes after suxamethonium. There was only a transient improvement and the apnoea then continued for a further 4½ hours.

ACKNOWLEDGMENT

I am indebted to Professor H. Harris and Dr. M. Whittaker, of King’s College, London, for performing the biochemical investigations in this patient and his family.

REFERENCES


**SOMMAIRE**

L'auteur décrit un cas d'apnée prolongée provoqué par le suxaméthonium. Des renseignements concernant la transmission neuro-musculaire obtenus à l'aide d'un appareil stimulateur "Medilec" s'avèrent trompeurs. L'estimation de l'activité estérasique, le taux de dibucaine et celui des fluorures se tenaient dans les limites prévisibles dans le cas d'individus dont la cholinésterase est simplement atypique. L'étude électro-myographique montra une réaction normale au décaméthonium mais un blocage neuro-musculaire prolongé et grave provoqué par le suxaméthonium. Ce blocage a apparemment eu un effet dépolarisant pendant au moins 90 minutes. Après discussion d'autres cas d'apnée prolongée, l'auteur fait remarquer le rôle de la Neostigmine dans la prolongation de l'apnée. Il estime que le fait de montrer un soulagement post-tétanique n'implique pas nécessairement que des substances anti-cholinésterasiques amélioreraient l'état de blocage. L'auteur signale deux sortes de théories susceptibles d'expliquer ce type d'incidents et il indique les précautions à prendre pour éviter d'aggraver la situation par des mesures erronées.

**ZUSAMMENFASSUNG**


**HUNGARIAN SOCIETY OF ANAESTHESIOLOGY**

The Hungarian Society of Anaesthesiology has organized an International Symposium which will be held from September 25 to 28, 1963, in Budapest.

*Theme*: The selection of anaesthesia for the various types of operative surgery.

The Symposium will deal with the problems of anaesthesia for gynaecology and obstetrics, urology, ophthalmology, otorhinolaryngology, paediatric surgery, neurosurgery and neurotraumatology.

On September 28, in the morning, there will be a round table conference at which the problems involved in the organization of anaesthetic services will be discussed.

Further information can be obtained from the Secretariat.

*Postal address*: Symposion Internationale Anaesthesiologie (I. Surgical Clinic), 78 Úllói út, BUDAPEST, Hungary.