those cases where induction or immediate caesarean section is indicated from those in which pregnancy may be allowed to proceed with safety.

Summary

The oxygen content of venous and arterial blood from the umbilical vessels was determined after 100 first deliveries. Pregnancy in all the primigravidae had been clinically normal. The average oxygen saturation became less as maternal age and the length of gestation increased, and was sometimes dangerously low, especially in primiparae aged 30 or more delivered after the 41st week of pregnancy.

It is suggested that the relatively high rate of perinatal mortality in elderly primiparae—often clinically unexplained and accompanied by post-mortem evidence of foetal asphyxia—is due in part to inadequate foetal oxygenation after term.

Since 1953, in Aberdeen, labour has been induced routinely by rupture of the membranes in primigravidae aged 25 or more who are undelivered before the end of the 41st week, and caesarean section has been used more frequently if there are signs of foetal distress, especially meconium in the liquor amnii. Apparently as a result, the excess perinatal mortality in elderly primigravidae has been eliminated. Possible limitations and hazards of the procedure are discussed.

We wish to thank Mr. W. Z. Billewicz for undertaking the statistical analysis.

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RESPIRATORY AND CARDIAC ARREST DURING ANAESTHESIA IN CHILDREN

BY

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Outside the realms of cardiac surgery, cardiac arrest often occurs unexpectedly, sometimes during quite minor operations. During recent years an ever-increasing number of publications have appeared in this country and in America suggesting both increased awareness and increased incidence of the condition. In the United Kingdom nearly three million operations are performed annually with an average anaesthetic death rate of 600 (Milstein, 1956).

Stephenson *et al.* (1953) pointed out that cardiac arrest is by no means rare in children. Out of 212 fatalities, 20% were found to have occurred in children under 10, the highest incidence of any age group reviewed.

Following the occurrence of two deaths on the table in 1951 and 1952, a method of resuscitation was developed by one of us (E.H.R.) which has been employed successfully on eight occasions without a

single failure. We believe it saved the lives of at least five of these children. In the other three instances there is some doubt whether the heart had definitely stopped beating after cessation of respiration, but, as is stressed later, the method can be applied so easily and quickly that we would not wait for confirmatory evidence before resorting to it.

The literature on cardiac arrest has been amply reviewed in recent years (Palomera, 1952; Stephenson *et al.*, 1953; Johnson and Kirby, 1954; Lewis and Stanley-Brown, 1955; Milstein, 1956). Survival rates generally were low (from 10 to 28%). A notable exception was the 66% survival rate reported by Lahey (1953). Prompt action, a prearranged plan, and readily available equipment are considered to have been the reasons for this good survival rate.

Diagnosis and Treatment

Cardiac arrest is presumed to have occurred if it is impossible to feel the peripheral pulses (carotid, radial) or the apex beat. In addition there will be no respiratory movements, no operative bleeding, ashen-grey pallor, dilating pupils. (Looking for capillary refill and recording blood pressure are probably a waste of time.) We agree that a diagnosis can be made with certainty only by electrocardiogram or by opening the chest and inspecting the heart directly.

Resuscitation in cardiac arrest requires restoration of heart action and pulmonary ventilation within a very short time. After three to four minutes permanent damage will occur to the vital brain centres. Thus an effective cerebral circulation must be instituted in this time by artificial means, and maintained until a spontaneous heart-beat returns.

We claim that in young children it is possible in most—if not all—cases to restore heart and lung action by a single, simple, and effective manœuvre which can, if necessary, be carried out single-handed. Of our eight cases only two can be described as "poor risk" cases. The patients' ages varied from 8 weeks to 13 years. In all cases respiratory failure preceded cardiac arrest.

One of us (J.B.) had personal experience of only one of these cases, which occurred after this paper had been prepared. The other seven cases occurred at four different hospitals, each with a different anaesthetist.

Five cases are described in some detail. Of the other three cases, unfortunately, no adequate data were kept, though in none of them could a pulse be felt before resuscitation began. All our cases were preceded by respiratory arrest due to one or more of the following factors: prolonged hypoxia or anoxia; hypercapnoea; overdosage of anaesthetic agents—for example, thiopentone, cyclopropane; and possibly vagal stimulation.

There is no doubt that some degree of anoxia was present in every case, either as part of a general oxygen lack or owing to an inadequate airway. In one case inhalation of a small quantity of blood appears to have been a factor. It was not always possible to be quite certain of the exact stimulus or agent responsible. Most accidents occurred during induction of anaesthesia or at the end of operation.

Description of Method

As soon as it appears reasonably certain that cardiac arrest has occurred (the signs of which have already been described), no time-wasting manœuvres for confirmation of diagnosis are employed. It is then possible to start effective resuscitation in a matter of seconds, as follows:

1. Any blankets, towels, instruments, or other impediments close to the patient having been quickly discarded, the table is lowered to approximately 10 degrees Trendelenburg by the anaesthetist, while the surgeon positions himself at the patient's right side.

2. A nurse is instructed to check the time and call out each minute (as suggested by Bailey, 1941, 1947).

3. Having made sure that the airway is clear, the anaesthetist rhythmically inflates the lungs with 100% oxygen. Unless an endotracheal tube is already in position or can be inserted immediately, no time is wasted over intubation at this stage. The lungs can be adequately ventilated through a face-piece and reservoir bag connected to a Boyle's machine.

4. Meanwhile the surgeon places his right forearm under the patient's knees and his left forearm behind his neck.

5. The patient's bent knees are raised and the hips fully flexed, forcing the thighs against the upper abdomen and chest, the spine being flexed at the same time (Fig. 1, A).

6. When full flexion is reached, the surgeon compresses the patient's chest with the bent knees, using some of his own weight judiciously to add to the pressure (Fig. 1, B).

7. He then lowers the patient's legs, fully extending them on the operating table (Fig. 1, C). At this point the anaesthetist inflates the lungs.

An effective method of resuscitation should fulfil the following criteria; it must be simple to apply; it must not be too fatiguing for the operator; it must be effective in providing adequate ventilation, and at the same time have an effect on the circulation sufficient to maintain a cerebral blood flow so as to prevent cell damage. To this we might add that it must be successful every time. In our experience to date it has been so.

(N.B.—One of our patients—Case 5—twice became apnoeic in the ward in spite of a good airway being present. Artificial respiration by the above method was carried out by the nurse in charge and was rapidly successful both times.)

The following five cases out of a total of eight illustrate the successful use of the method.

FIG. 1.—A, Patient's bent knees are raised and hips flexed so as to bring thighs in contact with upper abdomen and chest. B, Surgeon uses some of his own weight to press patient's thighs and knees against his chest and upper abdomen. C, Patient's legs are lowered and fully extended on the operating table. (At this stage the lungs should, if possible, be inflated with oxygen.) If an anaesthetic machine is not at hand, an assistant standing at the head of the table should here extend the patient's arms above his head as in Silvester's manœuvre.

FIG. 2.—Above: Spirometric tracing of patient aged 10 years and weighing 5 st. 1 lb. (32.2 kg.). Spontaneous breathing at A. Arrow=given injection of suxamethonium. Spontaneous respirations ceased at B. Our resuscitative manœuvre applied at C (N.B., omitting lung inflation). Arm-raising added at D. Vertical divisions represent minutes, horizontal divisions 100 ml. Spontaneous respirations=average 300 ml. Our manœuvre=average 200 ml. With added arm-raising=average 350-400 ml. Below: Spirometric tracing of patient aged 11 years and weighing 5 st. 11 lb. (36.7 kg.). Spontaneous respirations at A. Given 75 mg. of suxamethonium (arrow). Respiratory paralysis at B. Flexion-extension manœuvre applied at C. Added arm-raising started at D. Spontaneous respirations=average 250-300 ml. Our manœuvre=average 250 ml. With added arm-raising=350-400 ml.

The manœuvre is repeated 12 to 15 times a minute, which appears to be the optimum rate. If there is no response after one to one and a half minutes, the chest must be opened and the heart massaged (always provided the airway is satisfactory).

Even if the manœuvre is not successful in restoring the heart-beat within that time, both surgeon and anaesthetist have been doing something active to the child (and, as we believe, maintained a cerebral circulation) while the theatre nurse prepares for thoracotomy.

In one of our cases the above manœuvre was continued for one and a half minutes before a heart-beat could once more be felt.

Case 1

A healthy boy aged 5½ was admitted for adenoidectomy. Premedication: "Omnopon" 1/9 gr. (7 mg.), and scopolamine, 1/450 gr. (0.14 mg.). Anaesthesia was induced with nitrous oxide, oxygen, and cyclopropane followed by ether. During the course of induction laryngospasm developed. The anaesthetic was stopped and the lungs were inflated with 100% oxygen without effect. After about 30 seconds no pulse could be felt, the patient was white, and the pupils were dilated. The surgeon was called to the anaesthetic room and agreed that cardiac arrest had occurred. Immediate resuscitation by our method was begun—that is, about one minute after cardiac arrest had been diagnosed by the anaesthetist. After one minute's resuscitation the patient's pulse could once more be felt. It was weak and rapid. Soon after, feeble spontaneous respiration began and the pulse progressively improved. The patient recovered consciousness about five minutes later. The operation was abandoned and the patient was returned to his ward, where he fell asleep. When seen about five hours later he was fully conscious and there was no sign of cerebral irritation. Three days later he developed chicken-pox and was discharged home. Adenoidectomy was performed without mishap a year later.

Comment.—In this case cardiac arrest was probably due to a combination of anoxia and cyclopropane sensitization.

Case 2

A healthy girl aged 7 was admitted for tonsillo-adenoidectomy. Premedication: pentobarbitone sodium, 2 gr. (0.13 g.), and atropine, 1/100 gr. (0.65 mg.). Induction: nitrous oxide, oxygen, ether. Patient struggled during induction. She was taken into the theatre and a Boyle-Davis gag was inserted. While adenoidectomy was being performed the patient started to "gag." The anaesthetist proceeded to give an intravenous injection of thiopentone (about 0.25 g.): apnoea followed. Meanwhile the adenoid bed was packed with gauze and resumption of respirations awaited, before proceeding with tonsillectomy. No respirations had begun after half a minute, when the anaesthetist

reported that the pulse was impalpable. The surgeon quickly inserted a Magill endotracheal tube and removed the mouth gag. Cardiac arrest was suspected and the patient's head was lowered and towels and sandbags were discarded. Within one and a half minutes following respiratory arrest, resuscitation by our method was under way (between 30 and 40 seconds after cardiac arrest was thought to have occurred). After one to one and a half minutes of resuscitation a feeble pulse could be felt and spontaneous respiration quickly returned while the pulse increased in volume. The operation was completed and there were no sequelae.

Comment.—In this case cardiac arrest was probably due to a combination of anoxia and thiopentone.

Case 3

A healthy girl aged 4 was taken to the theatre because of bleeding following tonsillectomy. She was anaesthetized by nitrous oxide and oxygen followed by ether about one hour after the original operation. Although she was placed in the head-down position, it appears that some blood reached the larynx and produced laryngeal spasm. The blood was removed by suction, and oxygen was inflated from the anaesthetic machine by face-mask while the head was lowered still further. Quick release pressure on the chest was also tried but proved to be ineffective. After about one minute the pulse became impalpable. Immediate application of our method resulted in speedy return of both pulse and respiration after about nine or ten manœuvres (40–50 seconds). Subsequently the bleeding-point in the tonsil bed was secured, no further anaesthetic being required.

Apart from some rise in temperature for three days after operation, convalescence was uneventful.

Comment.—It appears that a combination of hypoxia and laryngeal spasm due to blood on the cords was responsible.

Case 4

A boy aged 8 weeks with polycystic disease of the lungs had been in respiratory difficulties since birth. He had areas of collapse and consolidation and was too weak to cough up his secretions. Respiration was "grunting" in character. He had received various antibiotics. He had been in an oxygen tent for two weeks, which was keeping him alive, as when he was taken out he became grey and limp in a few minutes. Bronchoscopy and removal of secretions by suction was proposed. Atropine, 1/150 gr. (0.43 mg.), was given, and a "polymask" with a high flow of oxygen was attached for transportation to the theatre; this actually improved his colour.

Induction was attempted with cyclopropane and oxygen, but this resulted in almost immediate apnoea several times, so "open ether" with added oxygen was given for 15 minutes, when breathing was regular and rhythmical. The larynx was sprayed once with 4% lignocaine. A suckling bronchoscope was passed without difficulty; oxygen was insufflated down the bronchoscope. Some secretions were removed from the bronchial tree by suction. After three minutes the baby started to strain weakly on the bronchoscope and cyanosis began to appear, the colour previously having been pink. The surgeon was informed; a finger was kept on the pulse. A minute later the pulse disappeared and the colour became grey. It was not possible to inflate the lungs satisfactorily via the bronchoscope, which was removed, a face-mask was applied, and the lungs were rhythmically inflated by bag-pressure. There was no immediate response to this, so the legs were intermittently flexed on to the abdomen and chest in time with the lung inflation. Half a minute later the pulse and colour returned; thus preparations for cardiac massage were left. Spontaneous breathing soon returned, and the child's general condition over the next few days was actually improved. There were no signs of cerebral irritation in spite of perhaps two minutes' circulatory arrest. (Cardiac massage in this case would have been technically difficult.)

Comment.—Cardiac arrest in this case was obviously due to several factors: prolonged hypoxia caused by pulmonary disease, further restriction of available lung surface during bronchoscopy, presence of secretions in bronchi, and possibly sensitization of myocardium following earlier use of cyclopropane.

Case 5

A previously healthy 12-months-old boy had been admitted three weeks earlier suffering from laryngo-tracheo-bronchitis. An emergency tracheotomy was performed the same night and the child seemed to make a good recovery on antibiotic treatment and steam inhalations. However, blocking of the tracheotomy tube by sticky secretions once or twice gave trouble on the first day and subsequently.

During the third week further respiratory difficulties seemed to indicate the presence of sticky bronchial secretions, which could not be removed by suction through the tracheostoma. It was therefore decided to perform a bronchoscopy.

Premedication was by atropine, 1/100 gr. (0.65 mg.). Before induction the child was noted to have marked dyspnoea, a poor colour, chest retraction, and a tracheal tug.

Induction of anaesthesia was by nitrous oxide and oxygen (50%) via a tracheotomy tube, with a small quantity of trichlorethylene added. As this proved to be insufficient for anaesthesia, the same mixture was given from another machine simultaneously by face-mask. The child lost consciousness gradually, and colour and respiration seemed to improve. He was then given 0.05 g. of thiopentone intravenously in order to produce sufficient relaxation; 100% oxygen was then given for 60 seconds. Apnoea lasted for 45–60 seconds, and was then replaced by even shallow respiration. A bronchoscope was introduced into the larynx, while the tracheotomy tube was removed. It was impossible to pass the instrument through the cords without using unjustifiable force. A further 0.025 g. of thiopentone was given with no effect on the cords, which remained closed. At that point the child seemed to "collapse," lost colour, and stopped breathing altogether. 100% oxygen was rapidly and forcefully given via a face-mask, while the surgeon attempted to reintroduce the tracheotomy tube (which proved to be very difficult). Colour seemed to return, but was rapidly lost again and the child completely failed to respond to oxygen (very little entered the chest, as there was scarcely any airway present).

Up to that point a strong apex beat could be felt. The colour now became ashen grey, the pupils were diverging and dilating, and a pulse could no longer be felt. Immediate resuscitation by our method was begun, while a knife for thoracotomy was brought. After 30–40 seconds the patient's colour changed from that of pre-mortem cyanosis to light cyanosis and the pulse could again be felt. After a further two minutes the anaesthetist managed to introduce a gum-elastic catheter via the cords, so that effective synchronous inflation with 100% oxygen could be given. The combined manœuvre was kept up for a total of 10 minutes before the child made any attempt to breathe. Spontaneous respirations, however, were intermittent (Cheyne-Stokes) for the next 45 minutes.

A No. 18 bivalve tracheotomy tube with inner tube eventually established an adequate airway, and the bronchi were sucked out through this.

The child subsequently twice stopped breathing after return to the ward without any obvious cause and was resuscitated as described.

Eventual recovery, though slow, was complete and there were no neurological sequelae.

Comment.—The use of thiopentone (even though the dosage was small), and the long-continued respiratory obstruction, producing hypoxia and hypercapnoea, possibly also reflex vagal stimulation during attempted bronchoscopy, were the factors involved in leading to cardiac arrest in this case. We believe that cardiac massage would have been of no avail, as the airway was inadequate during the critical first few minutes.

Analysis of Method

It is thought that resuscitation is effected as follows, the ventilatory and circulatory mechanisms being dealt with separately.

Pulmonary Ventilation.—(1) *First Expiratory Phase*: When the thighs are pressed against the abdomen the diaphragm is forced upwards, initiating pulmonary emptying; then as the chest is compressed this part of expiration is completed by external pressure. (2) *First Inspiratory Phase*: First, on release of pressure by the thighs, the chest re-expands by its own elastic recoil (Proctor *et al.*, 1950; Proctor, 1952). Next, on release of pressure from the abdomen the diaphragm descends, and this phase is completed as the body is fully extended. The next two phases occur if an anaesthetist has access to an anaesthetic apparatus (this is the usual intermittent positive-pressure pulmonary ventilation). (3) *Second Inspiratory Phase*: The anaesthetist inflates the lungs by positive pressure from a bag and mask, at the end of the first inspiratory phase described above (2). (4) *Second Expiratory Phase*: On release of the positive pressure in (3) the chest and the lungs recoil to the resting position, emptying the lungs. This phase then leads on to (1) above.

Circulation.—The following is an explanation of how an artificial circulation sufficient to resuscitate the patient is thought to occur. (1) Raising the legs causes venous blood to gravitate towards the abdomen. (2) Flexing the knees and hips squeezes venous blood out of the legs into the abdomen. (3) Pressure of the thighs on the abdomen forces blood from the abdomen into the chest, where it reaches the heart. (4) Pressure of the thighs on the chest squeezes blood out of the chest into the periphery, the brain receiving some blood especially, being lower than the heart. (5) When the chest re-expands on release of external pressure a negative intrathoracic pressure will initiate entry of blood into the chest, which will be continued in (1) above. This supposed artificial circulation depends, of course, on the heart valves and venous valvular system preventing blood merely moving back and forth.

As a preliminary check on its effectiveness the method was substituted for short periods in patients otherwise ventilated by intermittent bag-pressure. These patients had received an anaesthetic involving endotracheal intubation, conditions for which were supplied by the use of the muscle relaxant suxamethonium, which causes total muscular paralysis for a short time. The method was then substituted for bag-pressure, the patient being connected to a spirometer (in some cases) to obtain a tracing, instead of to an anaesthetic apparatus. (It was a proviso in these cases that no additional risk should attach to the procedure.)

It was found that the method itself—that is, without positive pressure—could maintain satisfactory pulmonary ventilation with a tidal exchange greater than by Schafer's method, there being no circulatory disturbance as judged by colour, pulse rate, and blood pressure.

It was further found that by adding arm-raising (as in Silvester's method) and thus making a two-phase into a four-phase manoeuvre, tidal exchange could be almost doubled, but bag-inflation (with oxygen from a Boyle machine) proved as effective and would normally be used in the operating theatre.

Discussion

The effects on the circulation of methods of artificial respiration do not always appear to have received the attention that they deserve. However, Pollock (1942), and Thompson *et al.* (1946) and Thompson and Rockey (1947) have shown that mechanical inflation and deflation of lungs produce "actual movement of blood column" and that "blood can be circulated over the entire body without the benefit of any heart action whatever." They also found that positive plus negative pressures (push-pull) were more efficient than only positive pressures plus release or negative plus release.

Similar conclusions were reached by Sadove *et al.* (1951). Moreover, Hill (1900) had found evidence that the brain could be kept alive by a mere trickle of blood. On the other hand, Pask (1948) considered that it was unlikely that artificial respiration could do anything worth while for the patient whose circulation had completely stopped and whose heart made no expulsive beats at all. In his opinion complete failure of the circulation for more than two or three minutes made recovery by any means impossible. Volpitto (1942) had reached similar conclusions.

Thompson's animal experiments with the use of tracer substances (radioactive sodium, oxygen, etc.) fit in with our own experience, as judged by results—namely, that prompt and efficient artificial respiration in the manner described by us can and does restore heart action (in children at any rate, whose supple bodies lend themselves more easily to such manoeuvres). We also agree with Thompson's findings that mere positive inflation of lungs plus release is not enough, and that it is essential to deflate the lungs actively as well in order to produce an effect on the circulation. The same conclusions were reached by Sadove *et al.* (1951), who investigated several methods of artificial respiration on curarized human volunteers. They found that push-pull methods were at least twice as efficient as Schafer's method.

Careful studies of ventilatory volumes and airflow patterns as well as measurements of circulatory effects were carried out by Gordon *et al.* (1951). In their opinion arterial oxygen saturation appeared to offer more convincing proof of the efficiency of resuscitative measures than mere measurements of pulmonary ventilation.

Spirometric tracings obtained during performance of our method of artificial respiration on paralysed subjects showed that slightly better tidal volumes could be obtained than by the use of Schafer's method. (Incidentally, by adding arm-raising, as in Silvester's method—thus making it a push-pull manoeuvre—tidal volumes were doubled (see Fig. 2).)

Cordier (1943) lays down the following principles underlying all methods of artificial respiration: They must (1) give sufficient pulmonary ventilation (as near as possible to normal subjects at rest); (2) stimulate heart and circulation to help respiratory exchange and transport of oxygen to the tissues; (3) be harmless in themselves and rapid in obtaining results. All these precepts are apparently fulfilled in the method described by us, and in addition it is thought that an artificial circulation sufficient to prevent cerebral damage is produced.

Until the advent of relaxants in anaesthesia, experiments on human subjects (volunteers) were difficult to perform and widely varying results were obtained (see Table taken from Cordier, 1943). Anaesthetized subjects who have been rendered apnoeic by the use of muscle relaxants correspond most closely to those in whom accidental cardiac arrest occurs (Sadove *et al.*, 1951).

Of the vast number of methods of artificial respiration advocated from time to time (Fisher, 1944, lists over 70), at most half a dozen are now being taught to first-aid teams. The one described by Byrd in about 1870 is still in use as an excellent method of resuscitation in asphyxia neonatorum. In carrying it out the obstetrician holds the supine infant in the crooks of his arms, and by compressing his whole body like a concertina and then stretching it he actively deflates the child's lungs (and incidentally at the same time stimulates his circulation) before allowing the elastic recoil of the chest wall to take in air. It will be seen that our method is very similar in its conception and thus not really new.

In conclusion we want to say that there is both theoretical and practical evidence to support our belief that artificial respiration carried out in the manner described by us is a life-saver, and that it should be tried without any delay when unexpected respiratory failure in a child is followed by cardiac arrest and carried out for 1½ minutes before proceeding to thoracotomy, as there is a very good chance that the latter may prove unnecessary. Ideally, the time to start using it is before the heart has actually stopped beating.

Widely Varying Results Achieved in Ventilation Studies Prior to the Advent of Muscle Relaxants. (After Cordier, 1943.)

Experimental Conditions	Author	Lung Ventilation in c.c.m.	
		Schafer's Method	Silvester's Method
1. Subjects having voluntarily suspended their breathing	"Suspended Animation" Committee (1903) Schafer (1904) Burton-Opitz (1922) Ploman (1906) Hamburger (1909) Liljestrand <i>et al.</i> (1913) Eve (1932) Killick and Eve (1933)	366 520 504 (average) 500-600 616 500 1,000 350-550	458 178 — 1,200-2,000 454 540 — 200
2. Subjects in apnoea	Liljestrand <i>et al.</i> (1913) Héderer (1934)	170 100-150	190 250-280
3. Non-rigid cadaver	Héderer (1934) Bruns (1927) Bruns and Thiel (1930)	40-50 20 20	200 200 200
4. Apnoeic patients in moderately deep anaesthesia	Waters and Bennett (1936)	2 females, 180 (average) 2 males, 662 (average)	214 (average) 933 (average)

We furthermore believe that this method may have wider applications beyond those of the operating theatre—for instance, in coal-gas poisoning, drowning, electric shock, and so forth.

Summary

A new method of resuscitation after respiratory and circulatory arrest is described which has been successfully employed on eight children. There has not been one single fatality. The method consists basically in intermittently flexing and extending the whole body, to which may be added positive-pressure inflation of the lungs with oxygen.

It is believed that the method represents a sound and effective approach to dealing with cardiac arrest in children. Though it has been used by us only in the operating theatre, we think it may have wider applications.

It is more rapidly and more easily applied than other methods.

Five illustrative cases are reported.

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THE CLINICAL APPLICATIONS OF TRIIODOTHYRONINE

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Until recently thyroxine was considered to be the active thyroid hormone; however, its characterization by Harington (1926) and Harington and Barger (1927) was not followed by its extensive clinical use, desiccated thyroid being in general preferred. Gross and Pitt-Rivers (1952a) identified as triiodothyronine an unknown iodinated compound in the sera of patients treated with therapeutic doses of ^{131}I . Later this amino-acid was detected (Roche, Lissitzky and Michel, 1952a, 1952b; Gross and Pitt-Rivers, 1953a) in thyroid substance. The high biological potency of triiodothyronine, first observed by Gross and Pitt-Rivers (1952b) in rats, and by Gross, Pitt-Rivers, and Trotter (1952) in hypothyroid humans, has now been amply confirmed (MacLagan, Sprott, and Wilkinson, 1952; Colville and Bonnycastle, 1953; Deltour and Karamourtzounis, 1953; Heming and Holtkamp, 1953a, 1953b; Tomich and Woollett, 1953, 1954; Anderson, 1954). The results have shown that triiodothyronine is approximately five times as active as thyroxine on a molar basis. Some experiments, however, on intact animals have not shown any significant difference between triiodothyronine and thyroxine (Gemmell, 1953; Heming and Holtkamp, 1953a; Brown-Grant, 1955).

Various workers have compared the physiological effects of L-triiodothyronine and L-thyroxine in myxoedematous patients (Asper, Selenkow, and Plamondon, 1953; Lerman, 1953; Rawson, Rall, Pearson, Robbins, Poppell, and West, 1953; Blackburn, McConahey, Keating, and Albert, 1954; Blackburn and Keating, 1954; Starr, Snipes, and Liebhold-Schueck, 1955; Selenkow and Asper, 1955). It has been shown that L-triiodothyronine is three to five times as active as L-thyroxine on a molar basis: although there is a significant difference in the speed with which L-triiodothyronine produces metabolic changes, it appears to be qualitatively identical to L-thyroxine.

It has been suggested that triiodothyronine is formed as the result of the reductive deiodination of thyroxine (Gross and Pitt-Rivers, 1952b, 1953b) or by the coupling of one molecule of monoiodotyrosine with one molecule of diiodotyrosine (Roche, Lissitzky, and Michel, 1953). It is evident that coupling, as described above, can be expected to occur only in the thyroid gland. It has been shown by *in vivo* experiments on animals that thyroxine is converted to triiodothyronine in the peripheral tissues (Gross and Leblond, 1951; Flock and Bollman, 1955; Kalant, Lee, and Sellers, 1955; Hogness, Berg, Van Arsdell, and Williams, 1955). Pitt-Rivers, Stanbury, and Rapp (1955) demonstrated that triiodothyronine can be formed from thyroxine in human subjects in the absence of the thyroid gland. On the other hand, Roche and