3. THE NON-INVASIVE ASSESSMENT OF UTERINE ACTIVITY

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1.0 INTRODUCTION

The monitoring of uterine activity is of importance in prenatal medicine for the surveillance of the course of the pregnancy, and for assessing the condition of the fetus. The relevant information about uterine activity can be derived from mechanical effects, such as deformation, tension in the abdominal wall, changes in intrauterine pressure, and from electrical phenomena produced by the uterus, i.e. the muscle potentials which trigger the mechanical activity.

Since the uterus is not readily accessible for the direct recording of the mechanical aspects of its activity, the results of such measurements, as performed clinically, provide only little information about the state of the uterus. Normally, the activity is evaluated in terms of the intrauterine pressure, usually in the form of the so-called tocogram. Two methods, one invasive and the other non-invasive, may be employed. For internal (invasive) tocography, the pressure is determined by introducing a catheter-tip pressure-transducer, or a catheter connected to an external transducer, into the uterine cavity. With this method, it is possible to determine the absolute intrauterine pressure. Often, however, internal tocography is not employed because of the practical difficulties involved. Moreover, the insertion of a catheter may stimulate parturition, so that this method may be contra-indicated for possible premature deliveries,

NON-INVASIVE MEASUREMENTS: 2
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where monitoring of the uterine contraction might be of particular clinical value. External (non-invasive) tocography, in which intrauterine pressure changes are inferred from measurements on the abdominal wall, avoids these disadvantages of the invasive measuring techniques. However, it has the drawback of being only a relative measurement, and is also subject to numerous interfering influences which may severely affect the results it provides.

Although invasive and non-invasive tocography have, until fairly recently, been the only methods employed for the assessment of uterine activity, it has been shown that they do not allow detailed information about the mechanical activity of the myometrium to be determined. Simultaneous recordings of intrauterine and intramural pressure reveal local increases in wall tension that are not always mirrored by the intrauterine pressure.

Since the fundamental problems of tocography cannot be eliminated by technical improvements to the measuring equipment, it would seem justifiable to consider assessing uterine activity by processing the uterine electrical action potentials, which are directly related to the activity of the myometrium. As clinical trials have shown, this technique yields much more information, in particular with respect to the excitation and propagation of the contractions.

2.0 TOCOGRAPHY

The procedure of recording uterine activity can be traced back to the period around 1870. At that time, Kehrer (1867) and Schatz (1872), published the first ever intrauterine pressure curves which, in their precision and quality, remain exemplary even today. They measured the intrauterine pressure by means of a liquid-filled balloon catheter, which they had introduced between the uterine wall and the membranes. The disadvantages of invasive pressure recording, in particular the associated danger of infection, precluded its use in the clinical setting for a long time. Since internal (invasive) tocography is still relatively rarely employed, and since this article is limited to non-invasive techniques, this method will not be dealt with further here.

External (non-invasive) tocography, which still remains the most commonly employed method for the monitoring of uterine activity, can also be traced back to the last century. In the obstetrical literature, reference can be found to an initial attempt to carry out external tocography using a large, air-filled metal box made by Schäffer in the year 1896. Subsequently described external devices for the measurement of uterine contractions,

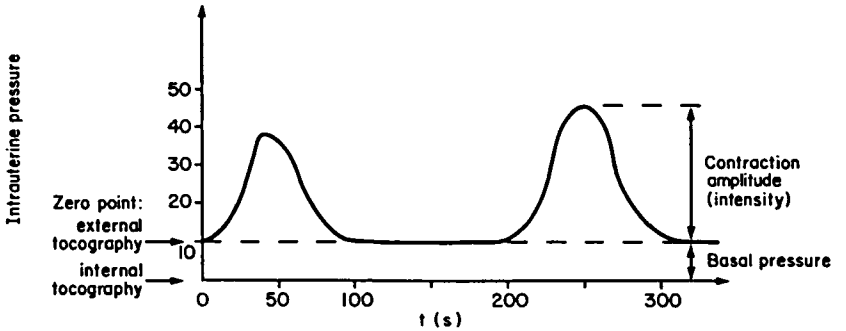

FIG. 1 Schematic representation of the course of uterine contraction. With internal tocography, pressure and intensity are recorded in absolute values, external tocography providing only relative values, without the possibility of determining the basal pressure. In the monitoring of uterine activity, apart from pressure, the form of the contractions also plays an important part.

such as the tocodynamometer (Crodel, 1927) and the external hysterograph (Rübsamen, 1920) had considerable shortcomings, but showed that there was very early interest in obtaining a continuous, external measurement of uterine contractions. Further progress was made by the development of a hysterotonograph (Frey, 1933) and a tocograph (Löwi, 1933), but they required that the entire recording unit be affixed to the abdomen of the

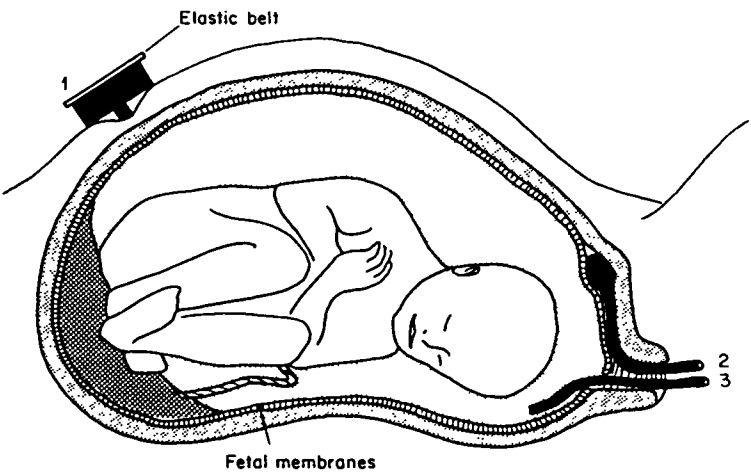

FIG. 2 Common methods of tocometry. (1) External tocometer with "tocometer pin", which is affixed to the abdomen by means of a strap. (2) Intrauterine, extra-amniotic method using a fluid-filled balloon catheter. (3) Trans-cervical intra-amniotic pressure measurement using an open-end catheter.

mother. An electromechanical uterine contraction recording device that is relatively insensitive to interference has been known since the work of Rech (1934). The uterine contraction transducer is attached to the abdomen by means of a rubber strap, and the recording device is set up at a distance from the patient.

The transducers in common use today still employ the principle described by Rech. Contractions of the uterus cause a sensing pin contained within the transducer to be deflected mechanically from its resting position, and the deflection is detected by a strain gauge. A number of strain gauges are connected together to form a measuring bridge (full or half-bridge). The change in resistance of the strain gauge accompanying a mechanical deformation is converted into an electrical signal, which represents a measure of the deformation and thus of the strength of the uterine contraction.

Despite very thorough investigations, agreement has still not been reached as to what, in fact, this transducer is measuring. The "hardness" of the uterine wall is a complex parameter which depends upon the wall tension, radius, wall thickness, internal pressure, transverse elasticity and the hardness of the uterine musculature, and is just as involved in the measurement, as is the deformation of the uterus and its "erection" during contractions. A further component of the measured signal results from the elastic nature of the transducer attachment. It can thus readily be appreciated that external tocography does not permit any statement about the absolute contraction amplitude nor the absolute level of the basal pressure or tone. All that we can obtain is an impression of the relative intensity of uterine contractions and changes in the basal pressure. Even this restricted performance may only be obtained when it is ensured that the transducer remains at its original site throughout the recording period and that the measuring conditions are not changed by any alteration of the patient's position. In very many cases, these requirements cannot be met; indeed, the maintenance of a given position by the patient might even be dangerous for her and the fetus.

In general, external tocography can reliably reproduce contraction rate and approximate form. Movements of the fetus can be recognized as small arrhythmic peaks, while respiratory movements appear as superimposed rhythmic waves. Care must be exercised, however, in the identification of the movements of the fetus since similar peaks in the contraction curve can also be produced by brief contractions of the abdominal wall musculature.

Although tocography has become a routine method for the monitoring of uterine contractions, and, today, almost no pregnancy remains without tocographic monitoring, it is not capable of providing information on

the detailed mechanical activity of the myometrium. Cibils and Hendricks (1969) made simultaneous recordings of intrauterine and intramural pressures by means of open-tip catheters inserted into the uterine cavity and the myometrium, respectively. They observed local increases in wall tension, which were not always accompanied by measurable increases in intrauterine pressure. These measurements were made in the post-partum uterus, but this does not invalidate the conclusion that a recording of intrauterine pressure is not a faithful reproduction of detailed activity of the uterine musculature.

3.0 THE ASSESSMENT OF UTERINE ACTIVITY ON THE BASIS OF ITS MYO-ELECTRIC SIGNALS

The myo-electric signal is the electrical manifestation of any contracting muscle. It seems reasonable to assess the possibility of obtaining accurate information about the mechanical activity of a muscle by analysing its myo-electric signals (electromyography, EMG). Many attempts have been made to record and analyse the electrical activity of the pregnant uterus. The lack of suitable measuring and signal-processing methods, and the resulting poor, or even false, results, meant that the possibilities of electromyographic monitoring of uterine activity long remained unrecognized. Only recently has a procedure been described (Nagel and Schaldach, 1980a) which resolves the earlier problems and permits the reliable determination of uterine activity on the basis of its myogram.

3.1 The Electrical Activity of the Uterus

Numerous attempts have been made to record the electrical activity of the uterus. Larks (1960) and Wolfs and van Leeuwen (1979) published a detailed review of the historical development in this area of research. In the majority of cases, the myo-electrical signals were picked up via skin electrodes affixed to the abdomen; in a number of cases, the EMG was recorded invasively using needle or micro-electrodes. A wide variety of different measuring methods and equipment were employed. Thus, it is not surprising that the numerous investigations produced widely varying results. The signals measured were simply attributed to the uterine activity without any attempt to check their actual origin. There are, of course, numerous possible sources of artefacts, such as movement of the patient, including respiratory movements, the electrical activity of the muscles of the abdominal wall, the smooth muscles of the intestine and the bladder, the electrocardiogram of the mother, skin potentials and movement

artefacts caused by the contracting uterus, all of which tend to degrade the signal-to-noise ratio.

Bode (1931), Clason (1934) and Mestwerdt (1944) recorded slow, biphasic waves, with faster fluctuations superimposed upon them. They suggested that part of the activity they had recorded was due to the heart, or the mechanical or electrical activity of the respiratory and abdominal wall muscles. Many other investigators, such as Dill and Maiden (1946), Steer and Hertsch (1950) and Halliday and Heins (1950) also recorded very low-frequency electrical signals (0·1–2 Hz), their measurements varying greatly with respect to the shape and occurrence of the signals. Müller and Liechty (1954) discovered more electrical activity between contractions than during contractions. Steer (1954) described two different types of electrical activity during uterine contraction: slow waves having a periodicity of several seconds, upon which faster waves (0·3–2 Hz) were superimposed. Sureau is one of the leading investigators in this field (Sureau, 1955, 1956, 1964; Sureau *et al.*, 1965). During contraction, he recorded sinusoidal waves having a frequency of 0·3–1 Hz. Larks (1956) obtained results similar to those of Steer. The amplitude of the electrical signals measured by different investigators varies considerably, covering a range of between 50 μV and 150 mV. Very extensive investigations were carried out by Wolfs and Leeuwen (1979), and although they did not succeed in obtaining all of the information contained within the uterine EMG, their measurements did reveal a marked correlation between the EMG and intrauterine pressure.

A very relevant question is why so many working groups recording the uterine myopotentials have obtained such a wide variety of different results. The reason for this would appear to be that the investigations have been based on incorrect or incomplete theoretical models of the origin, propagation and measurement of the electrical signals, and on the mode of electromechanical coupling. The result of this was the use of unsuitable measuring equipment for the recording of the signals. Thus, for example, in many cases, DC-coupled or very low-frequency measuring amplifiers were employed because adequate attention had not been paid to the problem of temporarily changing electrode potentials. The frequencies of these changes are in the same frequency range as the signals under investigation and can almost completely mask the useful signal. Thus, the question as to the frequency spectrum of the signals was completely ignored, and as a result, most measurements recorded only the fluctuating resting potentials, but not the action potentials typical for the activity of the musculature. Furthermore the selection of the most suitable combination of electrodes and recording positions and of the origin and composition of the signals, was not subjected to a systematic examination. The resolution of these

problems is, however, an essential pre-condition for the careful analysis of the electromyographic signals picked up from the uterus. An understanding of the physiology of labour presupposes a knowledge of a number of basic biological principles. Here, therefore, the fundamental processes in the formation of bio-electrical potentials, and electromechanical coupling, are briefly described.

Every animal cell is bounded by a highly differentiated membrane (cell membrane) which regulates the exchange of substances between the intracellular and extracellular spaces. This membrane has the capability of being selectively permeable and effecting active transport. The intracellular and extracellular spaces differ in their ionic concentrations. Within the cell, the concentration of potassium ions is 40 to 50 times as high as that on the outside. Sodium ions, on the other hand, have an extracellular concentration 3 to 10 times that of the intracellular concentration. Owing to this difference in ion concentrations, an electrical potential difference develops between the inside and outside of the cell. The resting potential of the cell membrane is between −60 and −90 mV. In the resting state of the cell (polarized), the intracellular potential is negative with respect to the extracellular space.

Nerve and muscle cells are characterized by the fact that stimulated or autonomous activation can have an effect on the cell membrane. As a

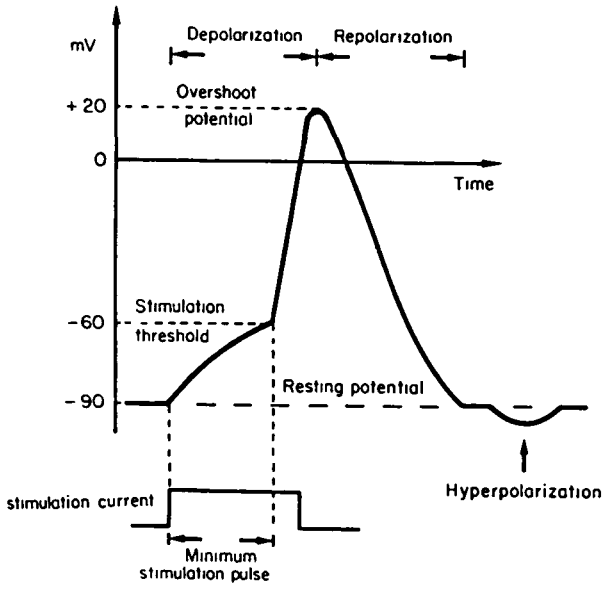

FIG. 3 The action potential of a cell membrane.

result of considerable changes in membrane permeability during activation, the ion concentration gradients undergo a shift, which leads to a change in the trans-membrane potential. The time course of membrane potential change from the start of excitation to the return to the resting state is defined as the action potential of the cell (Fig. 3).

The excitation takes place when the membrane potential is raised beyond a critical value (≈80% of the resting potential) by means of an impressed ion current. When this threshold has been crossed, the permeability of the membrane to sodium ions is markedly increased. As a result, driven by their concentration gradient, sodium ions pass into the cell where, on account of their positive charges, they give rise to a further depolarization which, in turn, leads to a further increase in the permeability of the membrane to sodium ions. This mechanism leads to an extremely rapid, avalanche-like, inflow of sodium ions. Before the membrane potential reaches a sodium-equilibrium potential, however, the permeability of the membrane to sodium ions drops again. The permeability of the membrane for potassium ions also increases during the depolarization procedure, leading to an increased outflow of potassium. When the permeability of the membrane to sodium ions decreases again, the outflow of potassium ions predominates, so that the cell again returns to its resting potential. At this point, the specific permeabilities of the membrane have returned to their original levels.

The depolarization that occurs when the critical threshold is exceeded represents an automatic process, invariable with respect to the course of potential changes, and independent of any further increase in the intensity of the stimulation. This behaviour is described as the "all or nothing" law of excitation. Since the energy required to activate the cell has to be provided by the cell membrane itself, a certain period of time, the refractory period, has to elapse before the cell has returned to its original state, and depolarization can occur again.

Locally occurring currents (flows of ions) accompanying the depolarization of the cell result in the stimulation of neighbouring areas within the same cell, thus propagating the stimulation throughout the cell body. If the coupling impedance to the neighbouring cells is small enough, the excitation spreads beyond the cell boundary.

The physiology of the uterus musculature has been studied in particular by Bozler (1942), Jung (1972) and Wolfs and Leeuwen (1979). The resting potential of the uterine musculature is dependent upon hormonal influences; the action potentials occur in salvos and in series. The musculature of the uterus produces its own excitation, the conduction of the excitation also occurring within the muscle fibres. No nervous pulses are needed to trigger uterine contraction. In principle, autonomic excitation

appears possible in all parts of the muscle although various investigations seem to indicate the presence of certain local "excitation centres" or pacemakers, where excitation preferentially originates. The rate of conduction of the excitation is also dependent upon hormonal influences.

In any contracting cell, mechanical shortening is triggered by depolarization. This electromechanical coupling represents a fundamental biochemical process, in which calcium ions have a particular role to play. At excitation, Ca^{2+} ions flow into the intracellular space where they activate the myofibril ATPase; ATP is split and energy liberated for the contraction process. The contraction itself involves an interaction of myosin, actin and ATP. For the interaction of the contractile apparatus with ATP, Ca^{2+} ions are required. By employing calcium inhibitors, an electromechanical decoupling can be brought about, so that, although the bio-electric excitation processes may persist, no further contraction of the muscle fibre occurs.

3.2 Measurement of the Uterine Myo-electric Potential

The action potentials of individual cells can be measured either directly inside or at the cell surface with the aid of micro-electrodes. Here, however, we are concerned with measuring the state of excitation of the entire uterus. A further point is that invasive procedures may not be used for such measurement. This means that the recording of the action potentials cannot be achieved at the cellular level using micro-electrodes, but must be made at a distance, on the surface of the body. The distribution of the flow of ions over the cell membrane gives rise to a characteristic electromagnetic field, which spreads throughout the neighbouring space. By measuring this field, conclusions can be drawn as to the time-space behaviour of the field-producing cell community, here the uterus. The total field resulting from the summation of the action potentials of the individual cells depends upon the form of the action potentials, their time sequence and also on the distribution of the cells in space, and the material properties and form of the medium surrounding them. It can be shown that, instead of measuring the overall electromagnetic field, the measurement of the scalar electric potentials produced at the surface of the body suffices to provide the information needed about the source functions, i.e. the condition of the field-producing musculature (Faust, 1965; Nagel, 1979). However, to permit conclusions to be drawn as to the state of excitation of the muscle, or rather the uterus, from the electrical potentials measured, the *a priori* knowledge of the physiology, geometry and the possible states of the uterus, is a prerequisite. The electrical potential at any given point of the body is a function of the space- and time-dependent ion currents

$\vec{G}(\vec{r}, t)$ and the conductivity of the transmission medium for the electromagnetic field. Assuming, for the sake of simplicity, a homogeneous medium (uniform conductivity), the potential $U(\vec{r}, t)$ at the measuring point $P(\vec{r})$ is given by

$$U(\vec{r}, t) = \frac{1}{4\pi\sigma} \int_{V'} \frac{-\operatorname{div} \vec{G}(\vec{r}', t)}{|\vec{r} - \vec{r}'|} \, dV', \tag{1}$$

where $\vec{r}'$ is the distance vector from the origin of the co-ordinate system to the current source, $\vec{r}$ is the distance vector from the origin to the measuring point, V' is the source volume, and σ is the conductivity.

Equation (1) permits the evaluation of the potential field provided that the source function is known and the conductivity is uniform. When a region contains inhomogeneities, it is usually convenient to take them into account by subdividing the region into a finite number of uniformly conducting subregions. In such a case one can account for the inhomogeneities by determining the secondary sources that necessarily arise at the interface between regions of different conductivity. In order to simplify the understanding of the following explanations, such effects are not included here; the results remain essentially unaffected.

In accordance with Eqn (1) the electrical potential of the uterus musculature as measured at any given point, is given by the volume integral of the ion currents that flow on contraction of the musculature. Conversely, this means that when adequate information is available about the possible physiological states and the resulting potentials, the measurement of the potential distribution at the surface of the body permits us to draw conclusions about the activity of the uterus. In this connection, our major interest is the question as to whether or not it is possible to derive from the electrical signal a measure for the strength of contraction of the uterus. Theoretically, on account of the strict electromechanical coupling, such a possibility should exist, provided this coupling has not been interfered with by the use of calcium inhibitors. In order to represent the relationship between the externally measurable EMG and the strength of uterine contractions in a more readily appreciable manner, Eqn (1) must be put into a somewhat different form.

In order to represent the sequence of excitations of the individual muscle fibres of the uterine musculature in time, the term spike train has been introduced. The spike train $a_n(t)$ of the nth muscle fibre is represented with the aid of a delta function $\delta(t)$, and can be expressed as follows:

$$a_n(t) = \sum_k \delta(t - t_{nk}). \tag{2}$$

In this equation, t_{nk} is the time at which the fibre is stimulated for the kth time. Although the electromagnetic fields produced by the action potentials spread throughout the body at the speed of light, the conduction velocity for the propagation of the excitation in the biological tissue is very small (approximately 0·1–100 ms^{-1}). As a result, the excitation of the individual, spatially distributed muscle fibres is associated with a time delay, which, *vis-à-vis* the duration of the action potentials, is not negligible and which is of considerable importance for the form of the EMG curve.

If the potential of the electrical field produced at the site of a measuring electrode by a single stimulation of a single muscle fibre n, is given as $h_n(\vec{r}, t)$, then the time course of the potential of this muscle fibre at the measuring point is given by the convolution (*) of h_n and the associated spike train a_n:

$$U_n(\vec{r}, t) = h_n(\vec{r}, t) * a_n(t). \tag{3}$$

In the representation of U_n, use is made of the fact that the course of the action potential is a function which is characteristic for the individual cell involved, and this does not change in time, i.e. it is independent of the number of times the cell has been stimulated. The potential of the muscle as a whole is given from the summation of the individual potentials of all N muscle fibres in the muscle:

$$U(\vec{r}, t) = \sum_{n=1}^{N} h_n(\vec{r}, t) * a_n(t). \tag{4}$$

According to Eqn (1), the potential of the individual excitation of a muscle fibre $h_n(r, t)$ can be expressed, as a function of the ion current, as:

$$h_n(\vec{r}, t) = \frac{1}{4\pi\sigma} \int_{V'} \frac{-\operatorname{div} \vec{G}_n(\vec{r}', t)}{|\vec{r} - \vec{r}'|}. \tag{5}$$

Integration of the volume of the muscle fibre n must be carried out. Equation (4), then becomes:

$$U(\vec{r}, t) = \sum_{n=1}^{N} \left\{ \frac{1}{4\pi\delta} \int_{V'} \frac{-\operatorname{div} \vec{G}_n(\vec{r}', t)}{|\vec{r} - \vec{r}'|} \, dV' * a_n(t) \right\}. \tag{6}$$

If the biphasic impulse responses $h(\vec{r}, t)$ are summated at the surface electrode in accordance with Eqn (4), or (6), a complicated interference pattern occurs, which is dependent upon the firing rate and firing pattern of the individual muscle fibres. For the interspike intervals, greatly varying

distribution functions are observed. In the literature, they are characterized as having a Poisson or a gamma distribution (Sanderson *et al.*, 1973), a Gaussian distribution (Clamann, 1969) or a Weibull distribution (De Luca and Forrest, 1973). In practice, it is impossible to characterize precisely the EMG, since the probability distributions for the patterns of excitation must be known, and so it is impossible to obtain more than rough estimates. A more suitable measure for the evaluation of the EMG is its modulation envelope $I(\vec{r}, t)$. This is determined by rectification and subsequent low-pass filtering of the EMG signal.

If the impulse response of the filter is designated $m(t)$, then the intensity of the EMG is given by:

$$I(\vec{r}, t) = |U(t)| * m(t) \tag{7}$$

The waveform of the envelope is strongly dependent upon the time constant of the low-pass filter. If small time constants ($\tau < 1$ s) are employed, $I(\vec{r}, t)$ reveals a marked structuring. Greater values of τ result in smoother waveforms which, however, are associated with a loss of information on short-term changes in intensity. To keep these losses to a minimum, great care must be exercised when choosing the cut-off frequency of the low-pass filter to be employed. Below, the question is examined as to whether or not, and under what conditions, $I(\vec{r},t)$ represents a measure of the force of contraction of the muscle.

3.3 Contraction Intensity and Intrauterine Pressure

If a muscle fibre is stimulated with a single pulse, it responds with a twitch, i.e. a brief contraction that exerts a force of $\vec{g}(t)$. Since the cells can only be either in an active or a passive state, and the active state of a cell is an unchangeable state ("all-or-nothing" law), the force produced by a single twitch is invariable. The gradation of the force of contraction of the muscle as a whole involves two regulating mechanisms. On the one hand, the number of activated muscle fibres can be tailored to the force requirement, on the other, with an increasing requirement of force, the individual fibres depolarize at ever-shortening time intervals. In the presence of a persisting spike train, the sequential contractions sum to form a maximum, resulting in complete tetanic contraction. Here, the stimulation interval must be larger than the refractory period of the muscle fibre membranes and smaller than the decay time of the individual twitch.

The pulse response of the contraction mechanism to stimulation manifests a considerably larger time constant than the action potential of the stimulated cell. On account of the resulting low-pass filtering of the spike train, and the fact that the contraction of the fibres cannot be negative, the

force produced by the muscle is continuous, despite the quasi-stochastic distribution of the stimulation pulses, in contrast to the superimposition of the biphasic action potentials.

The time course of the force produced by a muscle fibre is obtained from the convolution of the associated spike train $a_n(t)$ with the force $\vec{g}_n(t)$ produced by an individual twitch:

$$\vec{f}_n(t) = \vec{g}_n(t) * a_n(t). \tag{8}$$

The force developed by the muscle as a whole is obtained from the summation of all N fibres:

$$\vec{f}(t) = \sum_{n=1}^{N} \vec{g}_n(t) * a_n(t). \tag{9}$$

The computation is restricted to a linear superimposition of the individual forces. Non-linearities can, as experimental studies confirm, be neglected. For the further derivation, it is assumed that, to a first approximation, the forces of the individual muscle fibres are directed tangentially to the surface of the uterus.

The variable, which is of diagnostic importance and which may be measured directly, is not the force developed by the uterine musculature, but the increase in intrauterine pressure during the contractions. The relationship between muscle action and pressure increase can be approximated from a simple theoretical model. With the fetal membranes intact, the uterine musculature encloses a fluid-filled space, in which the pressure may be considered to be uniformly effective in all directions. The force acts tangentially to the surface. It is well known in mechanics that with such an arrangement the external force and the internal increase in pressure are proportional, i.e.:

$$\Delta p(t) = k_1 \cdot f(t), \tag{10}$$

Thus, for intrauterine pressure as a function of the force exerted by the uterine musculature, we have the equation

$$p(t) = p_0 + k_1 \cdot f(t), \tag{11}$$

where p_0 represents the basal pressure which is present in the absence of uterine musculature contraction. It is dependent upon a number of physiological factors, such as, for example, the elasticity of the uterus. It is not intended to discuss such details here, since they are of no importance for the measurement of the uterine activity.

3.4 Relationship between Mechanical Activity and Myo-electric Potential

As a result of the electromechanical coupling, there is a strong correlation between the strength of the uterine contraction, or the intrauterine pressure, and the myo-electrical potential. An essential difference is the fact that, in contrast to the intrauterine pressure, the EMG is dependent upon the site of the measuring electrode. With the aid of a number of simplifications the relationship can be made clear and a basis for the measurement of the uterine activity or the pressure changes from the EMG, found. Because of the low-pass filtering of the spike train through the mechanical contractile mechanism, as mentioned above, and the assumed uniform tangential direction of the individual forces contributed by the muscle fibres, Eqn. (9) can be simplified. This is achieved by the approximation of the temporarily accurately defined spike train by a medium stimulation frequency $\omega_n(t)$, which, of course, is dependent upon time. The force $f(t)$ can then be expressed thus

$$f(t) = C_1 \cdot \sum_{n=1}^{N} g_n(t) \cdot \omega_n(t). \tag{12}$$

If it be assumed that the individual muscle fibres are excited synchronously, and, further, that they each develop an identical contraction force, then we obtain, for the force $f(t)$

$$f(t) = C_2 \cdot k(t) \cdot \omega(t), \tag{13}$$

in which $k(t)$ is the number of activated muscle fibres. The question must now be examined as to whether the intensity of the EMG can be expressed in a similar manner, with the aid of the stimulation frequency. For this purpose, Eqn (6) is put into a more easily interpretable form. We shall first consider the case in which the measuring point and the co-ordinate origin coincide—in the centre of the spherical theoretical uterus. Here, $r = 0$. Equation (6) reduces to:

$$U(t) = \sum_{n=1}^{N} \left\{ \frac{1}{4\pi\sigma} \int_{V'} \frac{-\operatorname{div} G_n(\vec{r}', t)}{r'} \, dV' * a_n(t) \right\}. \tag{14}$$

The point of departure for further simplification of (14) is the vector identity:

$$\nabla \cdot (\vec{G}/r) = (\nabla \cdot \vec{G})/r + \vec{G} \cdot \nabla (1/r). \tag{15}$$

The integration of all three terms in volume V, which contains all sources, provides us, on applying the divergence theorem to the first term, with the

equation

$$\int_S \left(\frac{\vec{G}}{r}\right)\cdot \mathrm{d}S = \int_V \frac{(\nabla\cdot\vec{G})}{r}\mathrm{d}V + \int_V \vec{G}\cdot\nabla\left(\frac{1}{r}\right)\mathrm{d}V. \tag{16}$$

Since $G = 0$ on the entire limiting surface area S, it follows from (16) that

$$\int_V \frac{(\nabla\cdot\vec{G})}{r}\,\mathrm{d}V = -\int_V \vec{G}\cdot\nabla\left(\frac{1}{r}\right)\mathrm{d}V, \tag{17}$$

So that (14) can be re-formulated as follows:

$$\mathrm{U}(t) = \sum_{n=1}^{N}\left\{\frac{1}{4\pi\sigma}\int_{V'}\vec{G}_n(\vec{r'}, t)\cdot\nabla\left(\frac{1}{r'}\right)\mathrm{d}V' * a_n(t)\right\} \tag{18}$$

or

$$U(t) = \sum_{n=1}^{N}\left\{\frac{1}{4\pi\sigma}\int_{V} -\vec{G}_n(\vec{r'}, t)\cdot\frac{\vec{r'}}{r'^3}\,\mathrm{d}V' * a_n(t)\right\}. \tag{19}$$

The flow of ions, G, is a source function, which is interpreted as a dipole moment per unit of volume. A contribution to the potential at the measuring point is made only by the radial (G_{nr}), but not by the tangential, components of the dipole vectors $\vec{G}_n$. Accordingly, the following equation

$$U(t) = \sum_{n=1}^{N}\left\{\frac{1}{4\pi\sigma}\int_{V'}\frac{-G_{nr}(\vec{r'}, t)}{r'^2}\,\mathrm{d}V' * a_n(t)\right\} \tag{20}$$

applies.

With the assumed spherical symmetry and the relatively small thickness of the myometrium, for an estimation of the potential the contribution of the radius vector can be considered constant for all fibres, so that the factor $1/r'^2$ can be removed from the integral and the sum. Under these preconditions, the integral can be expressed as a function $y_n(t)$, now depending only on time. Equation (20) becomes:

$$U(t) = \frac{1}{4\pi\sigma r'^2}\sum_{n=1}^{N} y_n(t) * a_n(t). \tag{21}$$

With synchronous stimulation of all muscle fibres, and the same shape of the curve of all $y_i(t)$, because of the refractory period of the cells, no

interference phenomena can occur. In this case, the potential can be expressed by

$$U(t) = \frac{1}{4\pi\sigma r'^2} k \cdot (t) \cdot (y(t) * a_n(t)). \tag{22}$$

where $k(t)$ is the number of muscle fibres stimulated. If the potential function described in (7) is rectified and filtered, applying the same arguments as for the strength of the mechanical contraction, the spike train $a(t)$ can be replaced by the stimulation frequency $\omega(t)$ and we obtain, with constant c_3 for the intensity of the EMG, an expression having the form:

$$I(t) = \frac{c_3}{4\pi\sigma r'^2} \cdot k(t) \cdot \omega(t). \tag{23}$$

A comparison of (23) and (13) shows that for constant r', the force of contraction and the intensity of the EMG (IEMG) are proportional to each other:

$$f(t) = c_4 \cdot I(t). \tag{24}$$

In addition, using (11)

$$p(t) = p_0 + c_5 \cdot I(t). \tag{25}$$

applies. Accordingly, in the special case under consideration, both the intensity of contraction and the relative intrauterine pressure can be determined from a measurement of the myo-electric potential.

Of course, the question may now be asked as to what practical significance this result has. Although, in the derivation of (24) and (25), so many approximations were made that one might not expect the result to be quantitatively correct, experimental investigations show that at least qualitatively it does in fact conform to the physiological situation. The reason for including this derivation here, however, is to make it clear that, at least in principle, it *is* possible to find a fixed relationship between the EMG, and the intensity of contraction of the uterus. This would seem all the more important since, to date, reports in the literature have all denied this possibility. This is possibly the result of the fact that, formerly, it has always been the EMG itself, but not its intensity, that has been evaluated.

We must now investigate the nature of the relationship between $f(t)$ and $I(t)$ without the restricting conditions assumed in the derivation of (24) and (25), in particular in the case of an external recording of the myopotentials. In this connection, we must first examine the question as to whether the dependence of the intensity of the EMG on the force of

contraction of the muscle changes when the assumption of synchronous stimulation of the muscle fibres—which is certainly not really the case—is dropped. For the mathematical determination of the relationship then applicable, the statistics of muscle excitation, and the geometry of the individual muscle fibres, must be known. On account of the complexity, perhaps even the impossibility, of this computation, no attempt was made to adopt this approach. Instead, experiments were performed to discover whether the linear relationship between $f(t)$ and $I(t)$ is preserved.

According to the literature (Person and Libkind, 1967; De Luca and Forrest, 1973), this is not the case for all muscles; sometimes, there is a square law relationship ($f(t) \propto I^2(t)$). With respect to the myometrium, however, we have been able to confirm the linear relationship already found by Milner-Brown and Stein (1975) for a number of other muscles. Accordingly, therefore, the assumption of synchronicity of fibre stimulation made in the derivation of the relations (24) and (25), does not result in a qualitative falsification of the result.

A further simplification, whose influence on the results has to be investigated, is the assumption of a spherical uterus and potential measurement in the centre of the sphere. Only under the above-mentioned conditions do the contributions of the individual dipole vectors in the overall potential, have identical weight. Both in the case of potential measurement outside of the centre of the sphere, and also in a change in the geometry of the uterus, a non-uniform weighting of the individual sources results. In accordance with Eqn (6), the influence of such sources that are closer to the measuring point becomes more marked, while those potentials originating in more distant muscle fibres become more attenuated. The result of this is that on moving the measuring point to a given part of the myometrium, mainly the activity of the muscle fibres in the immediate neighbourhood is recorded. Thus, by appropriately siting the electrodes, the local activity of individual regions of the uterus, or the spread of the contractions can be picked up. In this manner, motility disturbances, such as incoordination for example, can also be recognized.

For the global, non-invasive determination of the uterine activity, the measurement of the myopotentials at a single point is not adequate. For there is no point outside of the uterus that is equidistant from all the muscle fibres. Owing to the large spatial extension of the uterus, this condition is not even approximately fulfilled. Nevertheless, the uterine activity *can* be determined globally, if the potential is picked up simultaneously at a number of points. By appropriately siting the measuring electrodes and summating the individual potentials a measuring signal is obtained which can be used in Eqns (24) and (25) to give adequately accurate results. The degree of this approximation depends upon the

number of measuring points and on their sites. Clinical investigations have shown that, in the majority of cases, the measurement of potentials at two points on the maternal abdomen is sufficient to ensure a result that is representative for the whole uterus.

3.5 Interference Potentials

The external recording of the uterine EMG is made difficult by superimposed strong interference signals arising in the maternal ECG, the fetal ECG and the EMG of the abdominal wall musculature. The amplitudes of the interference signals are usually greater than those of the useful signal. Further possible interference components, such as electrode offset potentials, electromagnetic interference and noise potentials, are not considered here, since they can be avoided or suppressed by designing suitable measuring systems. In passing it might be mentioned that the bio-electrical interference signals are not included in the measurements indicated in the literature. One reason for this is, that in most measurements reported bipolar electrodes were used. With these the potential difference between two electrodes located close together is determined, so that the contributions to the signal of more distant sources, e.g. the heart, are strongly attenuated. The second reason is that the frequency response of the amplifiers employed only allowed signals below 2 Hz to be measured. In this low-frequency range, however, the interference signals mentioned have only a very small power density, so that their contribution to the measured signal is small. An analysis of the frequency spectrum of the uterine EMG, however, shows that it extends to about 250 Hz and has its greatest power density above 2 Hz. Accordingly, the recording of the EMG should be carried out in this frequency range. The low-frequency range below 2 Hz is not suitable for routine measurement since it is here that movement artefacts are strongly seen. The use of closely spaced bipolar measuring electrodes is reasonable only for the pick-up of local muscle activity.

For the analysis of the intensity of the EMG, the interfering components must be suppressed prior to rectification and low-pass filtering. The maternal ECG can be subtracted from the original signal using a procedure described by Nagel and Schaldach (1980b). The R-peaks of the maternal ECG (MECG) are easily detectable by means of threshold detectors on account of their prominence in the abdominal signal. Through the exponential averaging of succeeding segments of the signal, all containing the maternal QRS complex in the same phase position, a reference signal corresponding to one interval of the MECG is obtained. The other signal components are suppressed in the reference since they are statistically

independent of the MECG. Subtraction of the reference from the abdominal mixed signal after a special scaling operation, results in the complete elimination of the MECG. The fetal ECG can be eliminated in the same manner although, as practical experience shows, this is not necessary because of its small amplitudes; its influence on the labour (uterine activity) curve is negligible. A simplification of the procedure is achieved by limiting the potential measurement to the frequency range from *c.* 150 to 250 Hz. Since, here, the amplitude of the uterine EMG is considerably greater than that of the fetal and maternal ECG, signal separation is not necessary. When measurements were carried out in this restricted frequency range, no changes were observed in the labour curve.

The only interfering component that cannot be eliminated from the mixed signal, but can merely be reduced by appropriately positioning the electrodes, is the EMG of the abdominal wall musculature. This fact, however, is not necessarily a disadvantage. Its contribution to the labour curve is so characteristic that it can clearly be distinguished from the intensity of the uterine EMG. Furthermore, it can also show the behaviour of the mother under the stresses of labour, e.g., during expulsive contractions. Over and beyond this, it can be observed that the contractions of the abdominal wall muscles also lead to an increase in intrauterine pressure. Thus, the additional recording of the activity of the abdominal wall musculature is desirable rather than undesirable for the practical application of the measuring procedure described. In any case, the contractions of the abdominal wall muscles also strongly affect the external mechanical pressure recording.

3.6 Movement Artefacts

According to Eqn (6), the uterine myo-electric potential is dependent upon the measuring point. Thus it is to be expected that movements—either changes in the position of the patient, or changes in the geometrical state occurring during uterine contractions—will influence the potential of a measuring electrode affixed to the maternal abdomen. Depending upon the movement and the position of the electrode, or on the transmission path of the bio-electric signals, their amplitude decreases or increases, so that this effect also modulates the EMG, and can thus falsify the labour curve. The error can be eliminated by having the signal amplifier compensate for the fluctuations in amplitude. It is, of course, not desirable to adjust the amplitudes of the EMG to a constant level, since important information about its intensity would then be lost, for the amplitude changes caused by contraction would also be eliminated.

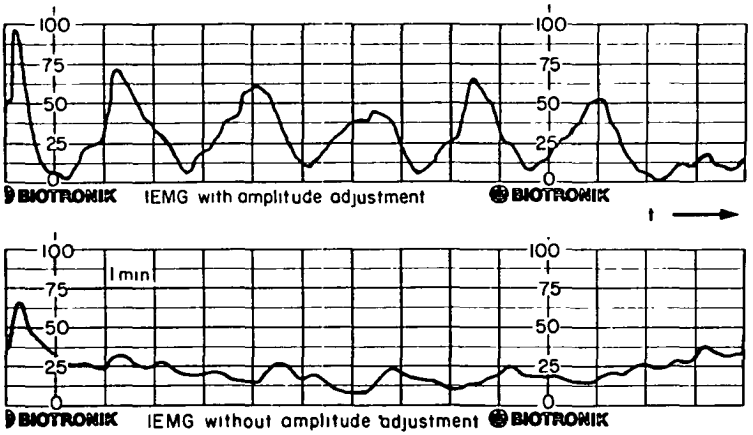

FIG. 4 Influence of amplitude adjustment of the EMG on the uterine contraction curve.

A surprising but simple solution is the use of the maternal ECG as a calibration signal for amplitude control. Experimental experience has shown that the amplitude fluctuations of the MECG correspond quite accurately to those of the EMG. From this it may be deduced that possible measuring errors can be avoided by employing automatic gain control that ensures the constant amplitude of the MECG within the original signal. In Fig. 4, the effects of adjusting the amplitude of the EMG to the contraction curve is represented by simultaneously recording the contraction curve with and without amplitude adjustment, using the same pick-up electrode.

4.0 COMPARISONS OF RECORDINGS OF THE UTERINE ELECTRICAL AND MECHANICAL ACTIVITY

Below, a number of examples of the recording of uterine activity are described, and these are intended to show the degree of conformity and also the differences between tocography and the recording of uterine activity via the EMG.

There is overall good agreement between the theory and actual measurement. Figure 6 shows a comparison of contraction curves measured mechanically and myographically. For the detection of the EMG, an electrode was placed at the isthmus and another at the fundus of the uterus. The potential reference point was obtained from a third electrode applied to the thigh. In contrast to the externally measured pressure (upper curve),

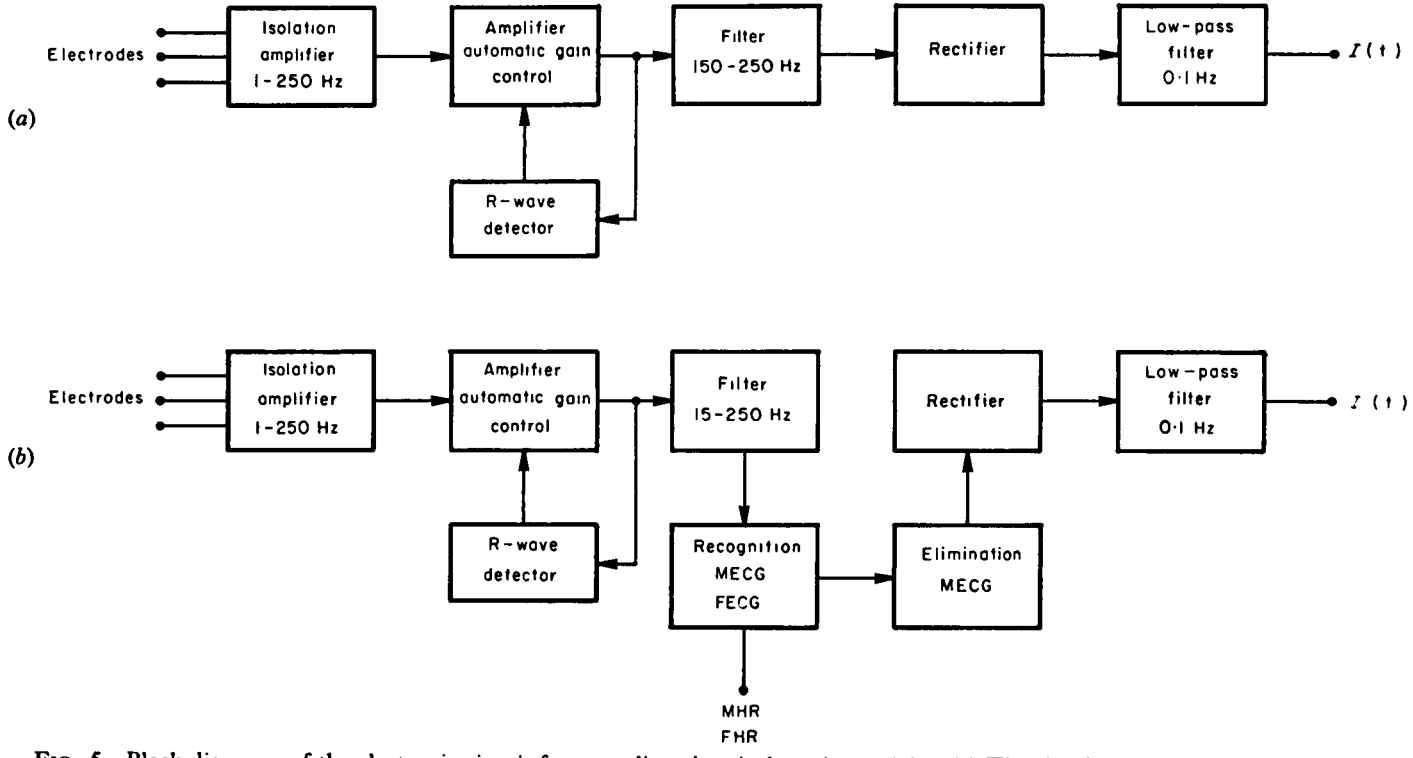

FIG. 5 Block diagrams of the electronic circuit for recording electrical uterine activity. (a) The simpler arrangement, in which only the high frequency components of the EMG are processed. (b) This circuit permits the recording of the labour curve from the complete EMG and also the maternal and fetal heart rates from the respective ECGs. Before producing the envelopes of the EMG, the maternal ECG is eliminated.

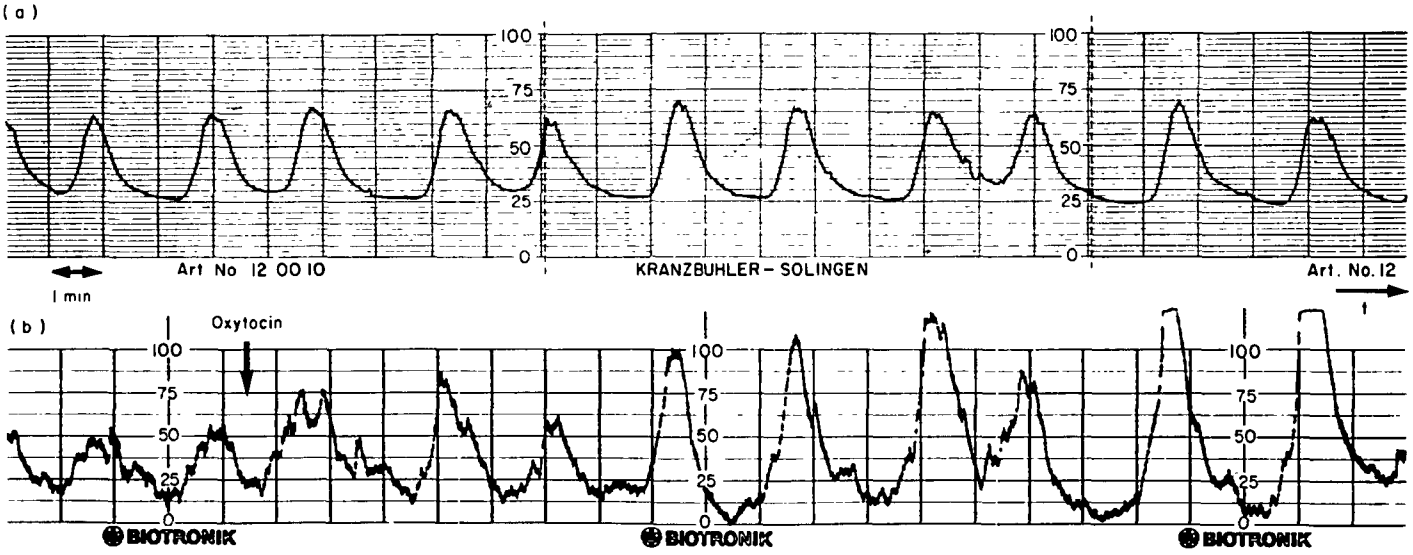

FIG. 6 Uterine contraction (labour) curve, recorded simultaneously with an external pressure pick-up (a) and from the electromyogram (b).

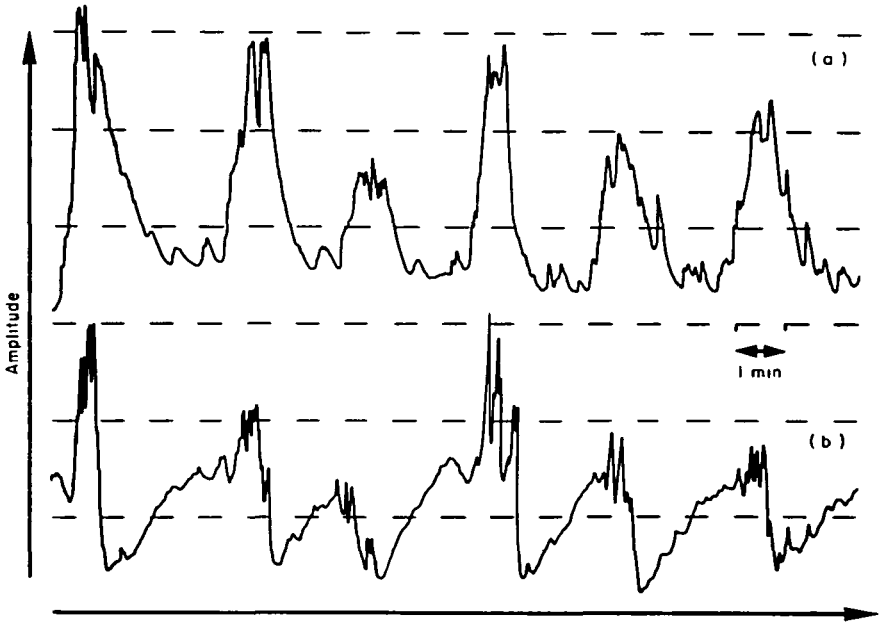

FIG. 7 Uterine contraction (labour) curve, recorded simultaneously with an external pressure pick-up (a) and from the electromyogram (b).

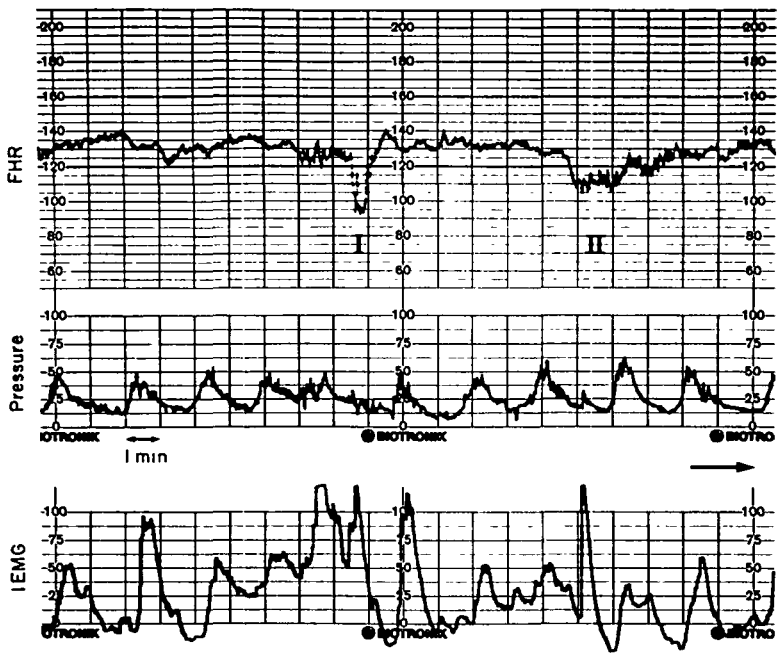

FIG. 8 Cardiotocogram with uterine contraction curve measured mechanically (external) and electromyographically.

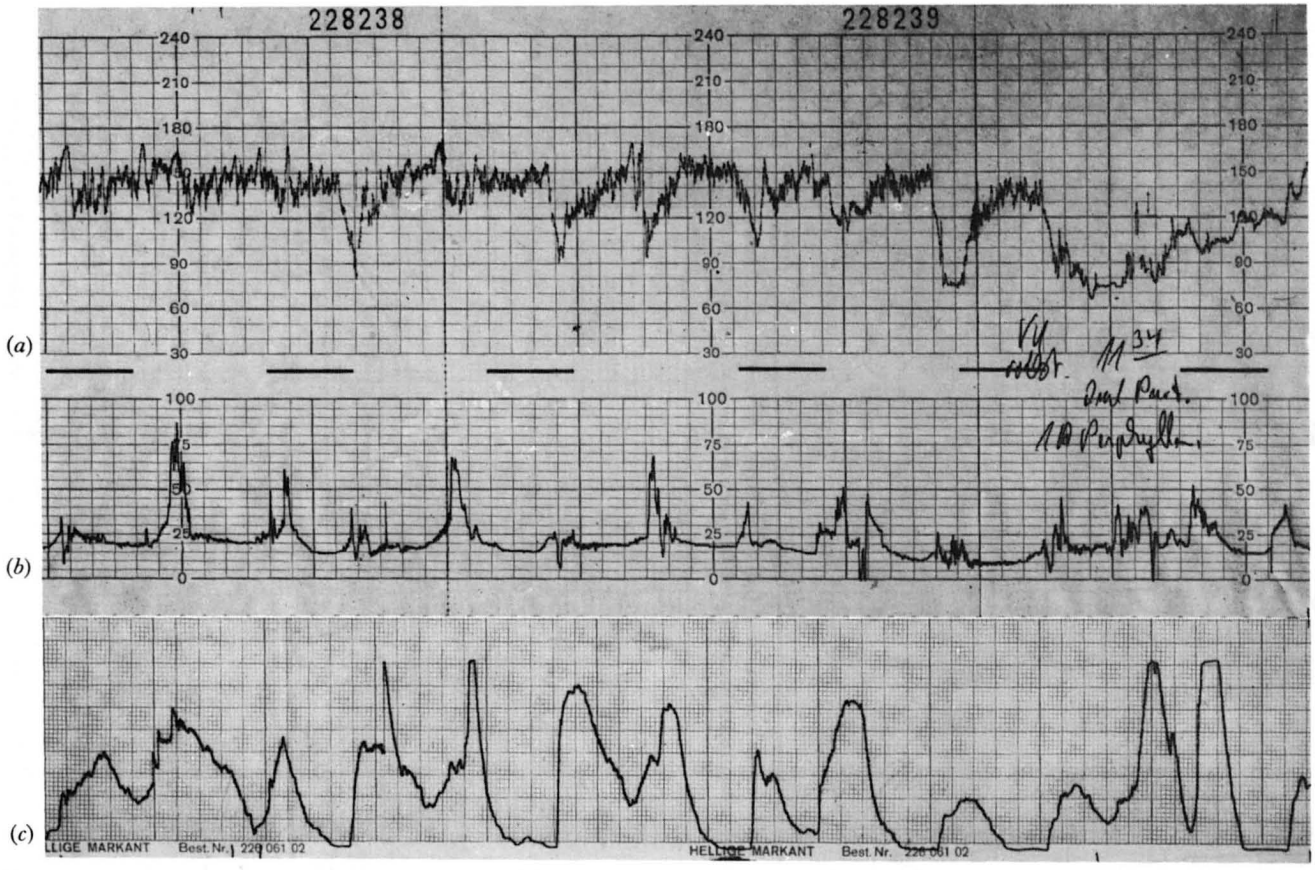

FIG. 9 Cardiotocogram with uterine contraction curve measured mechanically (internal) and electromyographically. (a) FHR; (b) intrauterine pressure; (c) IEMG.

the IEMG clearly reveals the increasing intensity of uterine contractions in response to infusion of oxytocin, the start of which is marked on the curve.

In Fig. 7, the myographic contraction curve shows that a new uterine contraction is stimulated immediately at the end of the previous contraction. The contraction is subjectively recognized only when a minimum intensity has been reached, and the activation of the abdominal musculature (expulsion contraction) is triggered. The externally recorded pressure curve does not contain this information about the course of labour.

The recordings shown in Fig. 8 reveal clear differences in the course of the uterine contraction curve. While the external pressure recording provides no explanation for the marked frequency drop at I and II of the FHR, the IEMG clearly reveals the powerful muscular contractions that may be considered as the cause.

A comparison between the intrauterine pressure and the IEMG shows that, in general, the two measuring procedures agree well. The invasive pressure recording technique, however, is less sensitive and considerably more susceptible to interference (Fig. 9).

5.0 CONCLUSIONS

All in all, uterine myographic recording has proved valuable in clinical trials. On account of the high information yield and the simplicity of handling, in particular for the simultaneous recording of fetal and maternal heart activity in addition to the uterine contraction curve, the procedure described is highly suitable for clinical routine work. The combined recording of the electrical activity of the myometrium and the intrauterine pressure will, it may be presumed, provide more information about the behaviour of the uterine musculature than either technique alone.

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