REVIEW OF LITERATURE

Pulse oximetry represents the most important advance in monitoring during anaesthesia since the introduction of sphygmomanometer.

Pulse oximetry has its origin in the work of Nicolai who in 1931 applied Beer's law to the transmission of light through the hand to study the dynamics of tissue oxygenation. He demonstrated that occlusion of the circulation produced an exponential fall in oxyhaemoglobin and rise in deoxyhaemoglobin.

Kramer in 1935 reported the continuous recording of oxygen saturation of blood flowing through unopened vessels using a spectrometric method. At the same time Matthes constructed the first device to measure oxygen saturation in vivo by transilluminating the ear.

Interest in aviators' oxygenation during World War II stimulated further development. In 1942 Squire, and later Goldie developed oximeters that set their zero value on tissue that had been compressed to squeeze out the blood, marking the beginning of modern pulse oximeters.
Milliken, in 1942, developed a light weight, practical aviation ear oxygen meter which he called an oximeter. Based on this work, Wood and Gracie, at the Mayo Clinic in 1949, built an oximeter with improved optics and an inflatable balloon that could make the ear bloodless for a reference setting. This device was manufactured by the Waters Company, and was extensively used in research, including evaluation of hypoxia during anaesthesia. More recently, Shaw, reported by Merrick and Hayes, developed a self-calibrating ear oximeter using eight wavelengths of light that was commercially produced in 1976 and has become the standard against which other oximeters are judged.

In 1974, Ioyagi, as reported by Nakajima et al developed an oximeter that used the variations in volume which occurs with pulsatile arterial flow to obtain a signal representing oxygen saturation. Because of sensitivity to motion and because of its large size, the device was not widely used. Yoshia et al, in 1980, incorporated plethysmography and oximetry in an instrument that needed no calibration or heat to determine oxygen saturation. Using plethysmography to identify a volume change resulting from the arterial impulse, the device compared the light absorbance in the absence of a pulse - its zero - and in the presence of the arterial pulsation to determine arterial oxygen saturation. With this
integration of plethysmography and oxymetry and subsequent application of solid-state electronics, the pulse oximeter determines values within 2% of those measured in vitro, bringing us close to accurate, practical non-invasive assessment of arterial oxygen saturation. Motion artifact and difficulties with low perfusion states continue to limit performance of currently available devices.

Regarding the utilization of pulse oximeter in operation theatre, it is only since 1985 that it has found widespread use. The clinical utility of the non-invasive oximeter in operating theatre was rediscovered by William New and Yelderman (Evaluation of pulse oximetry. Anaesthesiology, 59 : 349-352, 1982).

Various workers have demonstrated the usefulness of pulse oximetry in detecting preclinical hypoxaemia and hence avoiding the hypoxic fatality to the patient.

Cote and his workers described the incidence, duration, and severity of arterial oxygen desaturation as detected by pulse oximetry in infants and children. Additionally, in a cleverly designed single-blinded prospective manner they determined the impact of pulse oximetry information on the anaesthesia care team function.

A recent study of adult patients by Cooper et al. focusses upon an analysis of unanticipated,
undesirable events that were possibly related to anaesthetic management and required intervention in the recovery. In this study, the random use of pulse oximetry did not significantly increase the number of reports in the recovery room of hypoxic or hypoxaemic events, although the authors note that pulse oximetry may have provided information significantly earlier to clinician, thus preventing on hypoxaemic event (Cooper, J.B., Cullen, D.J., Neneskel, R., Hoagelin, D.C., Gevirtz, C.C., Csete, M., Venable, C.).

Another recent study by Severinghans and Naifeh describes in detail the performance of six commonly available pulse oximeter under conditions of sudden and profound transient arterial desaturation in adult volunteers (Anaesthesiology, 67: 551-558, 1987).

In another recent study carried out by J.T.Moller, P.F. Jensen, N.W. Johannessen and K. Eskersen from Denmark it has been again proved that pulse oximetry reduces hypoxaemia in O.T. and recovery room (B.J.A., 1992; 68: 146-150).

Regarding the use of electrocardiography in anaesthesia, J.B. Heard, A.E.Strauss and E.B.Krumbhaar, are considered pioneers who were the first to use electrocardiography in patients under anaesthesia in 1918.
Michael Johnson of University of Belfast had presented a paper on the usefulness of electrocardiography in 1948.

Lunn & Mushin in 1982 and Lykes in 1987 recommended for E.C.G. as essential part of monitoring of the patient under anaesthesia. Later on, E.C.G. recording was considered a minimum standard (in addition to another monitor of the circulation) by Harvard group (Eichhorn et al, 1986).

The pulse oximeter is an excellent monitor for transport from the operating room to recovery because of its portability and ease of use. In a study of American Society of Anaesthesiologists, Class I and II patients being transported while breathing room air, Tyler and associates (1985) found that 35% of their patients exhibited SpO₂ values below 90% during transport. This hypoxemia correlated with obesity and a pre-operative history of asthma. In a related study, Graham and colleagues (1986) found that 18 patients transported while breathing room air desaturated to an average SpO₂ of 89%, whereas 19 similar patients transported with supplemental oxygen experienced no major desaturations. In these two studies, the pulse oximeter firmly established the value of supplemental oxygen during transport to the recovery room. Hensley and co-workers (1986) also documented the necessity of administering supplemental
oxygen while inserting pulmonary artery catheters in pre-medicating cardiac surgery patients. In 12 of 20 patients, the $SpO_2$ values fell below 90% during catheter insertion, and $SpO_2$ fell to a low of 74% in 1 patient. Oxygen administered by nasal cannula at a rate of 4 L/min increased all of these patients' saturation to 95% or greater. Although these patients had various degrees of pulmonary disease in addition to their cardiac disease, all had adequate saturation values on arrival in the operating room before catheter placement. These data imply that nasal cannula oxygen should be administered to all patients in this situation.

The pulse oximeter is also a useful monitor of respiration in patients who are not in the operating room or intensive care unit setting. Choi and associates (1986) used $SpO_2$ to monitor post-cesarean section patients who were being treated with either epidural or parenteral narcotics. Each patient was monitored for approximately 1,000 minutes. Both groups exhibited an average of 3 to 4 minutes of desaturations below 90%, with no significant difference between the groups. The pulse oximeter was found to be a valuable monitor for these patients.

The study of patients with sleep apnea is another application of non-invasive oximetry. Strohl and Atene
(1984) used the Hewlett-Packard ear oximeter to monitor patients with sleep apnea during awake breath-holding maneuvers and during sleep. They found that the rate of fall of saturation was a function of the initial saturation at the onset of apnea. It was independent of whether the patient was awake or asleep and whether the apnea was obstructive or non-obstructive.

New applications for the pulse oximeter are being discovered on a regular basis. A pulse oximeter placed on the great toe has been used as an aid in cannulating the femoral artery in obese patients (Introna, R.P.S. & Silverstein, P.I., 1986). The pulse oximeter is now accepted as the primary indicator for the monitor of home oxygen therapy in patients with severe obstructive lung disease (Fulmer, J.D., and Snider, G.L., 1984).

If abnormal hemoglobin species can affect the accuracy of the pulse oximeter, one must also be concerned about dyes that are often injected intravenously during operation. These include methylene blue, indigo carmine, indocyanine green, and fluorescein. Scheller and Unger (1986) conducted a volunteer study in humans similar to the animal study by Sidi and co-workers (1986) and found that intravenously administered methylene blue caused very large decreases in SpO₂. In 1 volunteer the pulse oximeter reading fell to 1% after injection. Consistent
with the animal study, indo-cyanine green caused similar but less pronounced decreases in SpO₂ while fluorescein and indigo carmine had little effect on SpO₂.

Other technical problems in estimating \( \text{SaO}_2 \) by pulse oximetry have been reported. Motion artifact and external light interference have been reported by Brooks, T.D. et al, 1984. Kim and colleagues (1986) have reported that the pulsations in light absorbance may be primarily venous rather than arterial in origin. This implies that SpO₂ may read falsely low in circumstances leading to venous congestion, such as a dependent extremity.

Yelderman and New (1983) suggested that circumstances that reduce finger pulsation amplitude, such as hypothermia, hypotension, or administration of vasoconstrictor drugs, will adversely affect pulse oximeter accuracy. In the intensive care unit studies referred to earlier, several limitations were noted in this respect. In the study by Mihm and Halperin (1985), no pulse oximeter signal was obtained in 4 of 18 patients. Curiously, none of these patients was hypotensive or hypothermic. One of the patients had a history of vascular disease, and 2 patients were receiving dopamine, 1 with the addition of epinephrine. At the same time in this study the pulse oximeter was able to present a value on all patients who were hypotensive, most of whom were also receiving
vasopressors. Although these patients would be assumed to have high systemic vascular resistance, this was not established since cardiac output was not measured.

In other study, cardiac output measurements were determined along with systemic vascular resistance, body temperature, and hemoglobin (Tremper, K.K. et al, 1985). In 57 of 383 data sets, low signal values were obtained. In these data sets, 42% had a cardiac index less than 2.5 L/min/m², 16% had body temperatures less than 35°C, 9% had hemoglobin values less than 8 gm/dl, and 35% had a systemic vascular resistance greater than 2600 dyn/sec/m²/cm². Although all these physiological values were out of the normal range, good pulse oximeter readings were obtained in other patients with more abnormal values. This study also evaluated "clinically significant false negative information". Clinically significant false-negatives were defined as data points in which $\text{SpO}_2$ values were 2% greater than $\text{SaO}_2$ values while the $\text{SaO}_2$ values were 95% or less. This occurred approximately 12% of the time and was associated with either abnormally high or abnormally low systemic vascular resistance. With regard to hypothermia, of the 15 data sets collected with a patient temperature less than 35°C, the pulse oximeter read a low signal nine times and read an average of 7% less than the arterial value the remaining six times (false-positive information). Results from these two
intensive care unit studies indicate that situations that cause low signal are apt to be associated with high or low systemic vascular resistance or hypothermia. With regard to the data from the study of Tremper et al (1985), Barker and Tremper (1987) used an older model pulse oximeter (Biox III, Ohmeda, Boulder, CO), and newer pulse oximeters with newer software may not yield the same results. It can also be seen that the pulse oximeter did obtain values over wide-ranges of cardiac output, systemic resistance, blood pressure, and temperature. These results indicate that there is some degree of patient-to-patient variability in the pulse oximeter's ability to analyse the pulse signal and produce an accurate saturation value.

Several authors have estimated anesthetic and surgical risk in the elderly patients. The multifactorial cardiac risk index of Goldman and colleagues identified increased risk in patients greater than age 70 years. Djokovic and Hedley-Whyte reported that the mortality in American Society of Anesthesiologists physical status 2 (based on age) octogenarians was less than 1.0%, though this is still perhaps 100-fold greater than that of younger patients. The age-related surgical risk in apparently healthy, elderly persons is due in part to chronic physiological abnormalities.
Physiological changes secondary to aging:

Aging inevitably results in a decline in physiological reserve.

With regard to pulmonary function, the progressive loss of alveolar surface area, termed ductectasia decreases the contact area between air and blood. This process, which resembles emphysema, leads to increased mismatching of perfusion and ventilation leading to increases in both shunting and dead space.

As surface area declines, the alveolar lining exerts less surface tension, thereby decreasing pulmonary elastic recoil. As a person ages, closing capacity (the lung capacity below which small airways begin to close) progressively increases, exceeding functional residual capacity by age 45 years in the supine and by age 65 years in the upright position. The resulting tendency toward airway closure is a major cause of the decline in arterial oxygenation that occurs with age. Other pulmonary changes that occur as a result of aging include decreased ventilatory response to hypercapnia and hypoxemia, decreased chest wall compliance, decreased muscle strength, and reduced maximal expiratory flow rates.

The age-related decline in pulmonary reserve generally limits the elderly less than does the deterioration of maximal cardiovascular performance,
whether due to age, chronic disease, or physical deconditioning (Spurgeon et al, 1983). Although resting cardiac output is well maintained, the cardiac response to stress diminishes with aging. When stressed, elderly patients increase cardiac output by increasing left ventricular end-diastolic volume and stroke volume rather than by increasing heart rate (Rodeheffer, et al, 1984). Myocardial compliance also declines with age, thereby decreasing the margin of error in intravascular volume management. Aging also prolongs the time required for myocardial contraction and relaxation, resulting in several adverse effects, including higher heart chamber filling pressures, increased risk of subendocardial ischemia, and poor tolerance of tachycardia. Myocardial performance also appears to deteriorate because of abnormalities in calcium homeostasis and because of myocardial hypertrophy secondary to age-related increases in myocardial work (Geer, R.T., 1986).

As cardiovascular performance declines, so does the response to endogenous or exogenous B-adrenergic simulation (Lakatta, E.G., 1980). In contrast, the response to a-adrenergic drugs may be enhanced.

Concurrent disease processes compound age-related cardiovascular deterioration. Atherosclerosis generates obstructive lesions in both coronary arteries and
peripheral vessels. Myocardial infarction, hypertension, and cardiac valvular disease may precipitate congestive heart failure. An increased incidence of arrhythmias, especially heart block, atrial fibrillation, and premature ventricular contractions, may be due to the deposition of amyloid or calcium in the conduction system or to the atherosclerotic loss of elements of the conduction system with age (Ceer, R.T., 1986).

Because the decline in cardiac and pulmonary reserve may be difficult to assess, quantitation of physiological abnormalities may require invasive monitoring. Del Guercio and Cohn (1980) prospectively placed pulmonary artery catheters in 148 apparently fit patients greater than age 65 years. Of the patients studied, only 13.5% were physiologically normal; 63.5% demonstrated mild to moderate deficits, while 23% showed severe deficits. Of 34 patients with severe physiological compromise, operation was cancelled in 19, changed to palliative surgery under local anaesthesia in 7 others, and continued as planned in the remaining 8 patients, all 8 of whom died. This study suggests that many elderly patients have unrecognized diminished physiological reserve that contributes to peri-operative morbidity.
The autonomic nervous system and geriatric anesthesia:

With advancing age come declining physiological capabilities. The elderly patient's ability to preserve hemodynamic, respiratory, metabolic, and thermal homeostasis against perioperative assault is diminished. The mechanisms that maintain tissue perfusion and cellular function within acceptable limits depend, in a variety of ways, on the autonomic nervous system (ANS) and its effectors. Impairment of autonomic responses with age can result from deterioration of the sensor organs, afferent or efferent innervation, neurotransmitter production, autonomic receptors, cellular events distal to the receptor, or mechanical factors such as the vascular tree. Much research activity has been aimed at determining which functional changes predictably occur with age, independently of concurrent disease, and elucidating the causes of these changes at subcellular levels. Although the clinical implications of some research results remain speculative, the growing body of information on age-related changes in the ANS should help the anesthesiologist to better understand how the management of elderly patients and their responses to per-operative events will differ from that of young adults.

The most basic monitored parameters in anaesthesia, heart rate and blood pressure, are the foci of most
clinical studies of ANS changes with aging. Among healthy adults, resting heart rate and average heart rate do not seem to change with age. Resting heart rate is not identical to the intrinsic discharge rate of the sinoatrial node; it is modulated predominantly by tonic parasympathetic input. When parasympathetic and sympathetic blockade are imposed pharmacologically with atropine and propranolol, the intrinsic heart rate does decline with age (Kostis et al, 1982). Clinically important deterioration of cardiac pacing and conducting tissues occurs in some of the elderly, independently of ANS changes.

An age-related alteration in non-stressed heart rate that could involve the ANS is the loss of sinus arrhythmia. In young adults (without diabetes or other causes of autonomic neuropathy) the heart rate changes rhythmically with the phases of the respiratory cycle. Afferent inputs for sinus arrhythmia originate from pulmonary and intrathoracic vascular stretch receptors and the arterial baro-reflex receptors. As atropine has been found to ablate sinus arrhythmia, the efferent mechanism is considered to be the vagal slowing of the heart rate during exhalation. The variation of heart rate with respiration is consistently found to decline with age. In young adult diabetics with autonomic neuropathy (in which parasympathetic dysfunction seems to precede sympathetic), sinus arrhythmia is also lost.
In addition, the elderly manifest smaller heart rate increases after treatment with atropine than do young adults. For all of these reasons, some interpret the age-related loss of sinus arrhythmia to indicate a loss of parasympathetic influence on heart rate. If so, one also might expect the elderly to display diminished vagolytic tachycardia in response to pancuronium. Alternative explanations for the progressive loss of beat-to-beat heart rate variation could involve the aforementioned stretch receptors of baroreceptor reflex mechanisms (Vargas, E. & Lye, M., 1980; Davies, H.E.F., 1975; O'Brien, I. et al, 1986).

Among associated disorders, cardio-vascular diseases are particularly important; with the increased life expectancy, the number of patients suffering from cardiovascular diseases has steadily risen. According to Power's figures, the incidence of cardiovascular diseases is 6 percent in the seventh decade, and 100 percent in the eighth decade. Surgical mortality is considerably influenced by the presence of cardiovascular diseases. Nachlas et al (1961) observed a mortality of 6.6 percent in patients with cardiovascular diseases as opposed to 2.4 percent in patients without heart disease.

There is a high incidence of M.I. throughout the first post-operative week, and as most of the studies
in this field are retrospective, the exact moment of infarction often cannot be defined. This is especially true as a large number of the infarcts, varying from 21% (Tarhan et al, 1972) to more than 60% are silent. The first indication of an infarction has often been cardiovascular collapse, or hypotension or arrhythmia which have led the attending physician to investigate. It is, therefore, perhaps not surprising that PMI have been revealed throughout the peri- and post-operative periods. In 1974-75 Steen, Tinker and Tarhan reported that 25% and in 1972 Plumlee and Boettner that 41% of M.I. were discovered during the operation, while both Tarhan and colleagues and in 1975-76, 1977-82 Rao, Jacobs and El. Etr reported a peak incidence on the 3rd day after operation.

**Incidence of Peri-operative Myocardial Infarction**

Some studies do not distinguish between patients with and those without previous M.I., and report PMI rates varying from 0.08% to 24%, the highest tending to be in studies reporting mainly on patients with previous MI. In addition, the study by Baur, Nakhjavani and Kajani in 1965 reported an extreme 16% incidence in 150 patients selected at random from a surgical list. From these and other studies, it soon became apparent that whether or not the patient had had a previous MI was the factor most profoundly
affecting PMI rate. While Goldman and colleagues undertook a thorough multifactorial analysis of risk factors, including previous MI, from the outset most other authors have classified the patients into those with and those without previous MI. In this review, evaluation of other risk factors therefore refers mostly to patients with previous MI, as the infarction rate in patients without previous MI is so low that it is difficult to obtain data that are statistically evaluable.

**Congestive heart failure:**

This has also been identified as a risk factor. In addition, Pasternak and colleagues found a higher infarction rate in patients with a low pre-operative ejection fraction (less than 35%).

**Angina pectoris:**

Angina is the main symptom of coronary artery disease (CAD). Patients suffering from angina pectoris run a higher risk of cardiac complications such as MI than the general population. It is therefore, somewhat surprising that, although some authors report that many of the patients suffering a PMI had pre-operative angina most studies, including those with a more thorough multifactorial analysis, found stable angina not to be a significant independent risk factor.
Coronary artery disease shown by angiography:

In 1978 Mahar and colleagues reported that three vessel disease was a significant risk factor for developing PMI.

ECG changes before and after operation (excluding signs of previous MI):

Many patients with CAD present with a normal resting ECG before operation. In patients admitted for peripheral vascular surgery, Tomatis, Fierens and Verbrugge in 1972 found that, among those with a normal resting ECG, 30% had severe CAD, with 75-100% obstruction of a major coronary vessel, and 14% had a 50-75% obstruction. Of these patients, some show typical ST-depression as a sign of myocardial ischaemia during exercise testing. Thus Cutler and colleagues in 1979 found that 73 of 100 patients admitted for vascular surgery had no ischaemic signs on a resting ECG, but 14 (19%) of these had ischaemic signs during exercise testing. Of the 27 patients with signs of ischaemia on the resting ECG, 18 (67%) had additional ST-changes during exercise. In these patients 48 operative procedures were performed, and PMI occurred in six, all of whom exhibited ischaemic signs on the exercise ECG. Arous, Baum and Cutler in 1984 similarly found that PMI occurred in 25% of patients with positive pre-operative exercise test who underwent major peripheral vascular surgery.
Von Knorring in 1981 reported that patients with pre-operative ST-segment and T-wave changes, but no other indication of CAD, had a higher incidence of subendocardial infarction than patients with previous MI from history or ECG (12% v. 6%). Others have studied selected groups of patients with ischaemic heart disease and abnormal ECG before operation, and reported high incidence of a further deterioration in the ECG after operation, with a 4-9% infarction rate.

Most authors have suggested that a pre-operative ECG indicating pre-existing ischaemic heart disease is linked to peri-operative cardiac complications. On the other hand, Goldman and colleagues found no ischaemic signs on the ECG, but pre-operative arrhythmias (both ventricular and supraventricular) or recent MI, to be independent predictors of post-operative cardiac complications. Breslow and colleagues (1986, Anaesthesiology, 64: 398-402) reported that, while 19% (71 of 394 consecutive patients) had ECG abnormalities 1 hour after operation, nearly all were T-wave changes (flattening or reversal), and the incidence was not greater in patients with known CAD than in those without. In 70 of these 71 patients, the authors discovered no episodes suggestive of myocardial ischaemia in the post-operative period. They suggested that T-wave abnormality in the immediate post-operative period was a frequent
event not linked to the development of cardiac complications and probably not a marker of myocardial ischaemia. Thus there is considerable difference of opinion as to the importance of T-wave abnormalities, particularly when they are transient. Driscoll and colleagues used deeply inverted T-waves as the diagnostic criterion for PMI in 75% of the cases. These changes were normalized in all patients followed-up a few months later (four of nine patients). Thus the issue remains controversial.

High arterial pressure, as a transient event is an everyday occurrence in normal healthy individuals. Sustained high arterial pressure may be a secondary effect of many disease states, or it may occur independently of any specific disease state. In the latter case, the condition has been known as essential hypertension ever since Frank (1911) coined the phrase, but current convention designates the condition as primary hypertension, to differentiate it from secondary hypertension - that is, hypertension secondary to some specific cause.

Pickering (1968) emphasized that arterial pressure has a continuous distribution in the population, and there is no bimodal distribution that delineates normotensive and hypertensive sub-groups. Thus the definition of hypertension is based on a purely arbitrary choice of a dividing line as to what is normal. There is
no agreement as to the precise limit of normality, particularly with regard to systolic arterial pressures. Most authorities would regard a sustained diastolic pressure of 120 mm. Hg as distinctly abnormal, and many would regard a diastolic pressure of 110 mm. Hg as the lower limit for effective antihypertensive therapy. For the purposes of this discussion, a diastolic arterial pressure above 110 mm. Hg will be regarded as hypertension. As a justification to this value, we may consider the evidence of Goldman and Caldera (1979) that patients with diastolic arterial pressures below 110 mm. Hg are at no increased risk during anaesthesia and surgery.

Pre-operative hypertension:

This is defined very differently; either as a given arterial pressure value on a pre-operative evaluation or as patients receiving anti-hypertensive medication. There is a positive correlation between hypertension and ischaemic heart disease, and Prys Roberts, Mollo and Foex reported that patients with un-treated hypertension had the largest peri-operative haemodynamic alterations which, again, has been reported to correlate with a high PMI rate. It would therefore not be surprising if chronic hypertension also correlated with a high PMI rate, as reported by Kerola and colleagues, von Knorring, Steen, Tinker and Tarhan and Vormittag and colleagues.
On the other hand, there was no significant correlation in the study by Rao, Jacobs and El-Etr or that of Riles, Kopelman and Imparato or in the multivariate analyses performed by Goldman and colleagues and by Cooperman and colleagues. Thus this issue also remains controversial. In the first group of studies the statistical evaluation might have been inadequate, with no sequential multivariate analysis, and the latter studies reported on smaller numbers of PMI with a greater possibility of incorporating a type-II error statistically.

Diabetes mellitus:

Patients with diabetes have a two- to three-fold increase in prevalence of atherosclerotic disease. Most authors have therefore included diabetes in their investigations of risk factors for PMI. Only Driscoll and colleagues in 1961 have suggested a possible connection (but without statistical analysis), and the statistical data seem to refute such a connection.

Age:

The patient population is growing older and, with age, there is an increasing prevalence of ischaemic heart disease and associated diseases. This makes independent evaluation of age as a PMI risk factor more difficult, although in 1977 Goldman and colleagues
reported that age exceeding 70 yr. was an independent risk factor for development of cardiac complications. The population in a great number of the studies referred to here were patients with previous MI, and a patient who has experienced MI before the age of 30 yr. must have a serious underlying disease. It is, therefore, perhaps not so surprising that approximately 50% of the authors (retrospective), reported advanced age to be a risk factor, while the other 50% have not found a significant correlation.

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