

## CHAPTER 2

# THE PREMATURE INFANT IN THE NEONATAL UNIT

Aim: To provide an overview of the characteristics of premature infants and to develop an understanding of how risk factors and medical complications can affect their developmental outcome in general and more specifically their feeding abilities.

### 2.1 INTRODUCTION

A growing number of infants survive premature birth due to advanced technology and medical care. This phenomenon created a need for specialised and appropriate intervention to avoid the developmental delays that these infants are at risk of. According to Widerstrom et al. (1997), the increasing number of premature infants is causing a major public health problem, contributing to a rise in infant mortality and children with disabilities.

Premature infants experience unique problems, which are very different from the problems that full-term infants may display. Feeding difficulties are a major part of these problems. The service provided by the speech-language therapist / feeding specialist for the feeding problems that premature infants experience is part of the specialised care needed by these infants. However, the speech-language therapist is at present not necessarily equipped or adequately trained to render this service (Rossetti, 1986).

According to Hunter et al. (1994), in order to provide safe and effective evaluation and intervention in the NICU, four essential components of knowledge and skill

need to be acquired, namely: *firstly*, a strong medical foundation, *secondly*, a familiarity with the relevant technology and terminology in the NICU, *thirdly*, an awareness of the pathophysiology of individual diseases in the premature infant and *finally*, an understanding of the basic medical management of the infants in the NICU. The information presented in this chapter is an attempt to familiarise the reader with the premature infant and the aspects surrounding these infants, and to provide an overview of the requirements to work with this population, as formulated by Hunter et al. (1994).

## **2.2 PREMATURE INFANTS**

A discussion of premature infants in terms of who they are, what their appearance is like and how they behave, follows. This will provide a knowledge framework for the discussion of the feeding skills and feeding problems of the premature infant.

### **2.2.1 DESCRIPTION OF POPULATION**

An infant born before 37 weeks gestational age, or one month before the estimated date of delivery, is considered to be a premature or preterm infant (Rossetti, 1996). A further distinction is made by dividing these infants into groups according to the degree of prematurity: *mild prematurity* refers to infants born between 35 and 36 weeks gestational age; *moderate prematurity* refers to infants born between 31 and 34 weeks gestational age and *extreme prematurity* refers to infants born between 24 and 30 weeks gestational age (Rossetti, 1996). Prematurity and low birth weight (LBW) are still considered to be the greatest birth complications and accounted for 11% (preterm) and 7% (LBW) of live births in the USA in 1992 (McCarton et al., 1995). Due to improved and more sophisticated technology and medical procedures, more and more moderate and extreme premature infants with birth weights of below 2 500 g (LBW) and 1 500 g (VLBW),

survive the neonatal intensive care unit (NICU) (Bazyk, 1990; Oehler et al., 1996; Rossetti, 1986; Sweeney, 1985).

In South Africa the same phenomenon is experienced, maybe even more so, because an increased number of infants are being born in hospitals as a result of the implementation of the new primary health care plan which provides free medical care for all infants and young children (De Witt, 1999). At the Pretoria Academic Hospital's Maternity section only approximately 2% of all infants born previously, were LBW infants, compared to 16,8% in 1997, 18,8% in 1998 and 15,8% in 1999. The neonatal mortality decreased from 26,1/1000 in 1997 to 13/1000 in 1998 to 11/1000 in 1999 (De Witt et al., 2000). These numbers reflect the vast growth of the NICU population and by implication the growing need for appropriate early intervention strategies.

The preterm and/or small for gestational age (SGA) infant is at risk for a number of medical and developmental problems, including feeding disorders (Widerstrom et al., 1997). As a general statement of risk, the following parameter can be used: The younger the preterm infant is and the lower his/her weight, the higher the risk of long-term sequelae. The fact that the frequency of premature infants is increasing necessitates knowledge of the characteristics of this population.

### **2.2.2 CHARACTERISTICS OF THE PREMATURE INFANT**

The premature infant displays a unique set of characteristics, which differ from those of the full-term infant and therefore need special consideration. The preterm infant is not yet adapted to extra-uterine life. He or she cannot regulate their body temperature or manage sensory input. The motor system is not ready for gravitation and living in an unrestricted environment (Sheahan & Brockway, 1994).

Clinical experience has proven that women are not always certain about the expected time of delivery and the degree of prematurity may therefore be unknown. If the feeding specialist knows what a premature infant looks like and how they behave and respond, it should be possible to clinically identify such an infant and know what behaviour and abilities to expect from such an infant. That should enable him/her to plan appropriate and effective evaluation and intervention strategies. The ways in which premature infants differ in appearance and behaviour from full-term infants, are set out below:

### **2.2.2.1 General Appearance**

The premature infant is different in appearance from the normal full-term infant. The premature infant is very small and has very little body fat. Their skin therefore appears to be wrinkled, making them look like the elderly (Vergara, 1993). Their heads are characteristically flat bilaterally, but the shape usually changes to the normal shape by 2-3 years of age. Their ears are soft and pliable. In the extremely premature infant the pinnae are flat and shapeless (Vergara, 1993). The tongue seems to be small in the oral cavity and tends to be in an elevated position to stabilise itself (Morris & Klein, 1987). The skin appears transparent and thin at first and becomes cracked and peeled later (Sweeney, 1985). Extremely premature infants have no lanugo (the fine hair that covers the body of a newborn) (Sweeney, 1985). They exhibit a very weak cry or may be unable to cry at all (Witt & Rusk, 1993). The feeding specialist should be able to recognise these features, set appropriate expectations and take appropriate precautions in managing such infants.

### **2.2.2.2 Physiological / Autonomic State**

This state involves the respiratory status, heart rate, thermo-regulation and digestive system of the premature infant (Sheahan & Brockway, 1994). They often experience apnea; as Mandich & Ritchie (1996) put it, they forget to

breathe. Of importance to the feeding specialist is the fact that feeding apnea occurs even more often than sleep apnea (Dreier, Casaer, Devlieger & Eggermont, 1979; Garg, Kurzner, Bautista & Keens, 1989; Rosen et al., 1984). This is an important fact that should be included in a comprehensive evaluation of the feeding skills of the premature infant, as the feeding specialist should closely monitor the infant's breathing during feeding intervention.

Hunger cycles and regular feeding rhythms only develop after 35 weeks gestational age (Morris & Klein, 1987; Vergara, 1993). This has implications regarding the realistic stage at which the feeding specialist should introduce oral feeding intervention, because satiety inhibits sucking (Morris & Klein, 1987). In other words, if the infant is not hungry, he or she is not going to suck well.

When the infant is able to achieve physiologic homeostasis, he or she is healthy enough to organise his/her other states and is by implication ready to feed orally. The physiologic homeostasis can easily be disturbed because the brain of the premature infant is still immature and sensitive to stimulation. The neurological system is therefore more vulnerable to excessive, inappropriate stimulation. The feeding specialist should endeavour to avoid this scenario. This can be done by knowing and recognising the stress signals which involve the physiologic state. As soon as a stress signal is identified, intervention should be terminated.

Some of the stress signals involving the physiological system are: apnea (during sleep and feeding); tachypnea; irregular or shallow breathing; change of heart rate: brady- or tachycardia; fall in saturation levels; colour change (especially around the mouth); visceral responses, e.g. vomiting; sneezing, yawning, hiccups, grunting and mottling of the skin (Billeaud, 1993; Creger, 1995; Hussey, 1988; Morris & Klein, 1987; Sheahan & Brockway, 1994; Witt & Rusk, 1993). A comprehensive assessment of feeding skill and feeding associated behaviour should include the observation of these stress signals.

### 2.2.2.3 Neurological Status

Neurodevelopment follows a predictable sequence. A detailed discussion of the overall neurodevelopment of the premature infant is not within the scope of this study and only a brief discussion, concentrating on the development of oral-motor, sucking and swallowing skills will be presented. Knowledge of infant neurodevelopment should enable the feeding specialist to estimate the developmental age of an infant, which skills to expect and to efficiently and realistically apply feeding therapy. Sucking, even non-nutritional sucking (NNS), is disorganised at 27-28 weeks, but becomes more organised by 30 weeks. The co-ordination between sucking, swallowing and breathing will, however, only improve from 34-35 weeks (Glass & Wolf, 1994). Sucking is also weak and insufficient before 35 weeks gestational age (Vergara, 1993). Introduction of safe and efficient oral feeding can therefore not occur before at least 34 weeks.

Further neurological behaviour that will give an indication of gestational age is the following: A reflexive smile appears from 30 weeks (Hunter, 1993), while other primary reflexes may still be absent, reduced or inconsistent (Creger, 1995) until approximately 36 weeks gestational age when all primary reflexes can be elicited (Glass & Wolf, 1994). The development of the primary reflexes progresses in a cephalocaudal direction (from head to feet) (Creger, 1995). In other words, the lower down the reflexes are located that can be elicited, the older the infant is and the more ready to feed orally. Premature infants can easily become neurologically overloaded, because they have a low sensory threshold (Witt & Rusk, 1993). Sensory overload can have serious consequences, e.g. intra-cranial haemorrhage (Semmler, 1989). They react to sensory overload with stress signals, which can manifest themselves in any system of the infant, e.g. motor, state, physiological or neurological (Creger, 1995). The feeding specialist should be mindful of these facts, be skilled in reading these signs and should in no way contribute to an infant's stress (Rossetti, 1996). Further neurological risk signs that the feeding

specialist should be aware of are: that the infant may be jittery, or have hyperactive reflexes (Creger, 1995; Hussey, 1988; Sheahan & Brockway, 1994).

Should the premature infant experience any stress during feeding intervention, that session should be terminated so as not to contribute to stress in the premature infant.

#### **2.2.2.4 Motor System**

Knowledge of the motor system and its development also enables the feeding specialist to estimate a premature infant's gestational age (if it is unknown) and to make the necessary changes in the handling and expectations of the infant.

The motor system of the premature infant is generally not ready for gravitation or living in an unrestricted environment (Semmler, 1989). These infants are usually very hypotonic. The severity of the hypotonia is related to the degree of prematurity – the younger the infant the more hypotonic he/she will be (Hunter, 1993; Sheahan & Brockway, 1994; Witt & Rusk, 1994). After 30 weeks gestational age, the tonus will improve in a caudocephalic direction (from legs to head) – the opposite as that of reflex development (Creger, 1995; Hunter, 1993; Sheahan & Brockway, 1994). The level of hypotonia can give the feeding specialist an indication of the age and readiness for oral feeding.

The extremities of premature infants are typically postured in extension and abduction (Sheahan & Brockway, 1994), as the preterm infant lacks flexor tone and the strength to counterbalance the extension which is favoured by gravity (Morris & Klein, 1987; Vergara, 1993). Gravity also limits arm and leg movement (Morris & Klein, 1987). The premature infant displays extreme headlag when pulled into a sitting position and is unable to maintain head alignment with the trunk (Creger, 1995; Witt & Rusk, 1993). The implication for the feeding specialist is that the positioning of premature infants when giving feeding therapy should be

carefully considered. They should be swaddled with their arms forward and head control should be applied to keep the head and trunk in alignment, thereby avoiding headlag, as this is the most favourable position for deglutition (Morris, 1989).

The infants who have to be maintained on a ventilator for a prolonged period, display hyperextension of the neck, scapular elevation, retraction of the shoulders and the upper extremities, and arching of the trunk with immobility of the pelvis (Sheahan & Brockway, 1994). This body posture is unfavourable for efficient feeding (Lau & Hurst, 1999; Morris & Klein, 1987). The tubes and wires of the monitors complicate the environment in which the premature infant moves (Morris & Klein, 1987). Opportunities for motor movement and its development are therefore limited. When they do begin to move, the movements are spasmodic and jerky, but become more purposeful and smooth by 36 weeks gestational age. Their movements will, however, never be as good by 40 weeks gestational age as those of the full-term infant (Creger, 1995; Sheahan & Brockway, 1994). Special consideration should be given to the positioning of the infant who has been on a ventilator, during feeding.

Active movements can also contribute to instability of the autonomic (physiologic) system (Sheahan & Brockway, 1994). Such movements should be avoided during feeding as part of the attempt to reduce any stress. Stress signals involving the motor system that the feeding specialist must recognise, are: Frantic movements, finger splaying, straightening of the arms, arching, stiffening, hyperextension of the body, facial grimacing, covering the face or eyes with the hand (saluting), squirming or pushing away (Billeaud, 1993; Creger, 1995; Hussey, 1988; Sheahan & Brockway, 1994; Witt & Rusk, 1993).



### 2.2.2.5 State / Attention

State is defined as the level of consciousness or alertness and the environmental interaction of the premature infant. Attention is the ability to assume and maintain an alert state (Creger, 1995). According to Rossetti (1996), an alert state is a prerequisite for communication interaction as well as oral feeding.

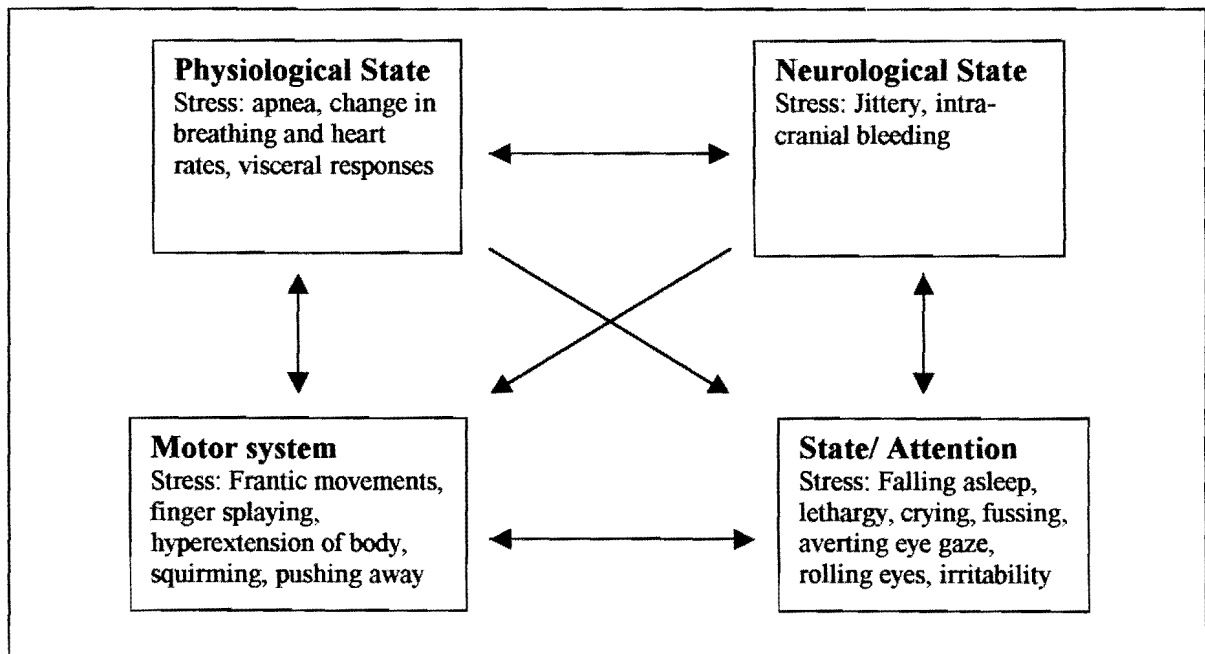
The sleep state predominates before 30 weeks gestational age. Regular sleep-awake cycles only begin around 34 weeks, but even when the premature infant reaches 40 weeks gestational age, he/she exhibits less predictable sleep-wake cycles than the full-term infant (Creger, 1995; Hunter, 1993; Witt & Rusk, 1993). The premature infant finds it difficult to maintain an alert state (Als, Duffy & McAnulty, 1988; Creger, 1995; Witt & Rusk, 1993). Sucking behaviour is known to be better when the infant is awake (Sheahan & Brockway, 1994; Geertsma, Hyan, Jeffrey, Pelletier & Reiter, 1985; Vergara, 1993). Mandich & Ritchie (1996) state that a quiet and alert state is a prerequisite for efficient sucking.

It should also be noted that premature infants are less responsive and vocalise less than full-term infants do. They tend to be irritable and have poor impulse and motor control. A limited ability to organise their behaviour and adapt to the environment, is also exhibited. Thus, difficulty of self-regulation is displayed by premature infants (Als et al., 1988; Witt & Rusk, 1993). Light, sound, temperature and general environmental conditions may also influence the infant's alertness (Becker, Grunwald, Moorman & Stuhr, 1991; Rossetti, 1998). Environmental control becomes even more important in feeding intervention, as part of an attempt to avoid an overload of the premature infant's systems. It is well known that their systems overload quickly and they may then respond with stress signals. Some of the stress signals that involve state and attention are: falling asleep, diffuse sleep state, lethargy, averting of eye gaze, rolling of eyes, irritability, crying and fuzziness (Billeaud, 1993; Creger, 1995; Hussey, 1988; Sheahan & Brockway, 1994; Witt & Rusk, 1993).

An infant struggling to maintain cardiorespiratory homeostasis finds it difficult, if not impossible, to assume an alert state and to interact with the environment. They may rather react with bradycardia, tachycardia, apnea or a decrease in saturation levels (Als, Lawhon, Brown, Gibes, Duffy, McAnulty & Blickman, 1986; Billeaud, 1993; Sheahan & Brockway, 1994). Feeding intervention in such circumstances will be out of the question, - medical conditions are always the first priority and therapeutic intervention secondary (Hunter et al., 1994).

The physiologic system, motor state control and attention do not develop simultaneously as in the full-term infant. The systems therefore do not support or enhance one another, but rather disorganise the behaviour of the premature infant (Creger, 1995). They have to learn self-regulatory skills like postural change, hand-to-mouth action, grasping, sucking, visual locking or hand-clasping to maintain or return to a balanced equilibrium. This in part explains why oral feeding does not come easily and effortlessly to the premature infant.

#### Homeostasis



(Source: Creger, 1995; Hussey, 1988; Sheahan & Brockway, 1994)

**Figure 2.1 Disorganizational potential of systems on each other**

All of the states and systems discussed above, influence one another and have the potential to disorganise each other (Figure 2.1). For example, the irritable infant may cry with accompanying frantic movements that causes change in breathing and heart rate, which results in the disturbance of the homeostasis.

### **2.2.3 CONCLUSION**

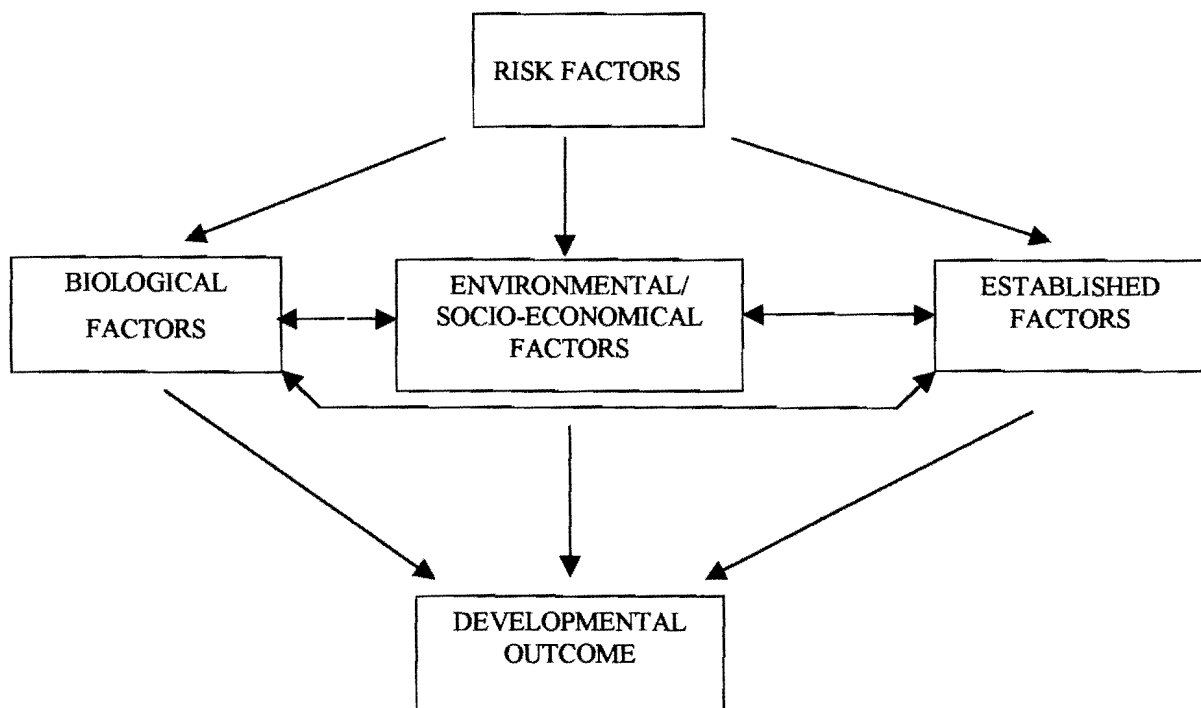
Oral feeding skills in premature infants are influenced by a number of factors. The feeding specialist should have a sound knowledge of the characteristics of the premature infant and the influence that the different states and systems may have on each other, to plan safe and efficient feeding intervention.

## **2.3 RISK FACTORS FOR DEVELOPMENTAL DISORDERS IN PREMATURE INFANTS**

When a premature infant is born, the first question raised is whether he will live or die. As his condition improves, questions about his future and developmental outcome are raised. It is not possible to say with certainty what his/her outcome will be, but the likelihood can be determined. That can be done by establishing which factors have an effect on the premature infant's development (Allen, 1984; Allen & Capute, 1989).

Risk factors can be simply defined as "a prediction of the manifestation of later developmental disability" (Thurman & Widerstrom, 1990:23). The reason for establishing risk factors is to prepare parents for their child's future. The infant's development should be monitored closely and family support be given where necessary, to minimise secondary complications (Allen, 1984; Allen & Capute, 1989).

The risk factors fall into three main groups based on the aetiology, i.e. biological, environmental and established (Aylward, 1990; Kirby, Swanson, Kelleher, Bradley and Casey, 1993; Widerstrom et al., 1997). Rossetti (1996), on the other hand, uses only two main groups in a symptomatological classification system, i.e. *firstly*, Established-Risk and *secondly*, At-Risk (which includes biological and environmental factors). Rossetti (1996) considers a child with known patterns of developmental delay, e.g. Down syndrome, not at risk for these delays, because it is known that certain delays will be present as part of the syndrome. Although Allen (1984) states that it is important to remember that risk does not imply causation, she also mentions that there are conditions that inevitably lead to developmental delays, such as those conditions mentioned as established-risks by Rossetti (1996). Rossetti (1996) also mentions that variations can stem from whether and at which age, these children receive early intervention services. So, in effect, these infants can then also be considered as being at risk of further and/or more serious developmental delays unless they receive early intervention.



(Source: Aylward, 1990; Thurman & Widerstrom, 1990; Vohr, 1991)

**Figure 2.2 The relationship between risk factors and developmental outcome**

Allen (1984) and Thurman & Widerstrom (1990) also group the factors according to the period of time in which they occur, pre-, peri- and/or postnatally. The discussion of risk factors that follows will be based on the three main categories of involvement or causation of developmental delays: biological, environmental and established. Those groups will then further be discussed according to the period of time in which they occurred. These main factors can also influence each other and further influence the developmental outcome (Figure 2.2). Reference to the relationship between these factors forms part of the discussion that follows.

### **2.3.1 BIOLOGICAL FACTORS**

Biological factors are called medical risk factors by Kirby et al. (1993). A variety of medical conditions can put a premature infant at developmental risk (McCarton et al., 1995). The greater the number of predictor variables, the more accurately a risk status can be assigned to a specific infant (Thurman & Widerstrom, 1990). The Kauai Longitudinal Project did not support this idea and found environmental influences to play a greater role (Aylward, 1990). Traditionally, the developmental outcome of infants with biological risk factors (particularly the LBW infants) has been emphasised and the environmental influences ignored (Aylward, 1990). Regardless of how big the influence on developmental outcome may be, or which influence may have the greatest effect, medical complications/biological factors will influence development and medical follow-up will be necessary in most cases.

The feeding specialist should take note of these factors as part of the medical history in the evaluation of the feeding skills of premature infants, to anticipate possible feeding problems and plan intervention. The influence of biological factors on developmental outcome and feeding skills will be explained in more detail in the following pages, which will be organised from the prenatal period until after birth (postnatally).

### 2.3.1.1 Prenatal Period

A number of influences can put an infant at risk of developmental delay, namely:

- *Intra-uterine growth retardation (IUGR)*

IUGR refers to the fact that the infant has not grown to his full potential in utero. The infant's weight is inappropriate for its gestational age and falls below the 10<sup>th</sup> percentile for gestational age on the standard IUG curve (Lubchenco, 1976; Mullen, Coll, Vohr, Muriel & William, 1988). The terms *dismature* or *small for gestational age (SGA)* are also used to refer to this group of infants. If the growth retardation started before 26 weeks gestational age, both the head circumference and weight will fall below the tenth percentile. Restricted head growth is associated with less than optimal neurodevelopmental outcome. It is therefore an important prognostic factor for developmental outcome. If the growth retardation started after 26 weeks, the intelligence of the *dismature* infant may be unaffected. In general, the SGA infants are associated with poorer developmental outcome (Rossetti, 1986; Sweeney, 1985; Thurman & Widerstrom, 1990; Vohr, 1991). They are also poor oral feeders because of their overall weak condition.

Traditionally these infants are introduced to oral feeding later than their appropriate weight for gestational age (AGA) counterparts, because it is general practice in the NICU's to introduce oral feeding based on the weight of the infant ( $\pm 1.8$  kg), rather than gestational age (Mullen et al., 1988). The literature does not support this viewpoint and uses gestational age as marker to introduce oral feeding (Bazyk, 1990; Merenstein & Gardener, 1989; Sheahan & Brockway, 1994; Vergara, 1993). The reason is that the ability to co-ordinate sucking, swallowing and breathing successfully is seen as a neurobehavioural maturation phenomenon (Semmler, 1989). Adequate neuromuscular co-ordination is a function of gestational age (Bu'Lock et al., 1990). Thus, it can be assumed that gestational age rather than weight effects maturation. Maturation, rather than weight, should therefore be considered when a decision on when to introduce oral

feeding is made. The feeding specialist should know whether an infant of low weight is SGA, but old enough to start sucking safely, or whether he is too young to suck. If the clinical gestational age according to a Ballard or Dubowitz count can not be found in the medical file, knowledge of the appearance and other characteristics of premature infants will give the feeding specialist an indication of the gestational age of the infant. This information should also be included in a comprehensive evaluation of the feeding skills of the premature infant to enable the feeding specialist to plan safe and effective feeding therapy for these vulnerable infants.

If the introduction to oral feeding is delayed in the dismature infant, he becomes prone to some of the complications of prolonged nasogastric feeding. The feeding specialist should endeavour to avoid these consequences. Infants who have received long-term nasogastric feeding experience more problems to transfer to oral feeding; they may become orally hypersensitive, develop aversive behaviour towards oral feeding and eventually become failure-to-thrive (FTT) infants (Bazyk, 1990; Geertsma et al., 1985; Morris, 1989; Merenstein & Gardener, 1989; Palmer et al., 1993; Vergara, 1993).

Another aspect of importance to the feeding specialist is that long-term nasogastric feeding negatively influences the mother-infant relationship, since the nasogastric tube is a constant reminder to the mother that a feeding problem exists and it is unnatural (Morris, 1989). If the mother wishes to breast feed, she might become despondent due to the vast amount of time she has to spend expressing milk and the difficulty of eventually getting the infant "on the breast". The delay in transfer to oral feeding means that the infant has an extended hospital stay which impedes bonding with the mother. It can place a financial burden on the parents as well (Mandich & Ritchie, 1996; Meier & Pugh, 1985).

One of the criteria for transfer to oral feeding is sustained weight gain (Creger, 1993; Mandich & Ritchie, 1996). If the dismature infant still has a very low weight,

he might still be too weak to feed orally and maintain a good weight gain, although he is of an appropriate gestational age to feed orally. The feeding specialist will have to make a decision in consultation with the medical staff as to how soon and often the infant can start with oral feeding in a safe and efficient way.

- *Placental abnormalities*

This may affect the oxygen and/ or nutritional supply to the infant in turn affecting the growth and the neurological development of the infant. Prenatal anoxia can cause anencephaly (Rossetti, 1986; Sweeney, 1985). Neurological problems are one of the main causes of feeding problems (dysphagia) (Arvedson & Brodsky, 1993; McBride & Danner, 1987; Miller, 1986; Rosenthal et al., 1995).

- *Viral infections: **TORCH** group:*

Toxoplasmosis, Others e.g. (a) Syphilis (the mothers should be treated before 18 weeks of pregnancy (Thurman & Widerstrom, 1990), (b) HIV - 80% of the infants who have the HIV virus, contracted the virus via transplacental transfer. These infants may have recurrent bacterial infections, respiratory problems, microcephaly and neurologic abnormalities, 90% experience static or progressive encephalopathy and painful swallowing (odynophagia).

**Rubella, Cytomegalovirus, Herpes simplex:** All of these viruses can cause neurologic sequelae, psychomotor retardation, microcephaly, learning disabilities, seizures, blindness, a sensory neural hearing loss and hydrocephalus (Creger, 1989; Rossetti, 1986; Sweeney, 1985). Any of these viruses which affect the neurological system, do not only place the infant at risk of developmental delays, but of feeding problems as well.

- *Repeated miscarriage, blood group incompatibilities and unexplained previous foetal or neonatal deaths*

These are other prenatal risk factors for poor developmental outcome (Rossetti, 1986).



It is clear that the health of infant and mother during the prenatal period is already important for the later development of the infant and mother-infant bonding and attachment.

### **2.3.1.2 Perinatal Period**

A number of factors during the birth process and/or in the first week after birth can also jeopardise developmental outcome, e.g.

- *Gestational age*

Prematurity is the number one risk factor for cerebral palsy, with the infants being born before 32 weeks being at the highest risk. This population is also at risk of developmental delays and feeding problems (Bennett, 1995; McBride & Danner, 1987; Rossetti, 1986; Sweeny, 1985). The premature infant is also at risk for medical complications, which will be discussed later. The infant younger than 34 weeks gestational age cannot co-ordinate sucking, swallowing or breathing successfully and is at risk of aspirating the fluid he is consuming, with dangerous consequences. (See par. 2.4 for more detail of the effect on feeding skills.)

- *Hyperbilirubinemia*

Bilirubin is a red bile pigment circulating in the blood. Excessive amounts of bilirubin can lead to central nervous damage (Witt & Rusk, 1993). Low levels of bilirubin in the very premature infant may have subtle effects on learning development. Physiologic hyperbilirubinemia is common in premature infants due to their limited ability to excrete bilirubin from their systems. In maternal foetal blood incompatibility, excessive haemolysis of red blood cells can lead to excessive amounts of bilirubin, which is a leading contributor to neurologic sequelae (Sehna & Palmeri, 1989). Recent prevention techniques have almost eliminated kernicterus, the disease resulting from hyperbilirubinemia. Extreme kernicterus can cause mental retardation, deafness, hypotonia and rigidity choreosis. Psychomotor delays can exist in the absence of overt signs of

kernicterus. If neurological impairment is a result of the hyperbilirubinemia, feeding problems can be expected. Infants with hyperbilirubinemia who receive phototherapy, also tend to be lethargic, which may influence feeding negatively, but it does not necessarily imply long-term feeding problems (Rossetti, 1986; Sheahan & Brockway, 1994; Witt & Rusk, 1993).

- *Metabolic disturbances*, e.g. **hypocalcemia**, **hypoglycaemia**, **metabolic acidosis** (Oehler et al., 1996; Sehnaal & Palmeri, 1989).

**Hypocalcemia** and **hypoglycaemia** can cause jitteriness and even convulsions. **Hypoglycaemia** can also cause hyperirritability, apnea, cyanosis and irregular respiration. **Acidosis** means the pH in the body is lower than normal (Sheahan & Brockway, 1994). In severe cases it can result in neurologic deficits with associated feeding problems (dysphagia). Although some metabolic disorders are genetic and occur prenatally, others damage the central nervous system after birth (phenylketonuria). If the metabolic disturbances can be detected early enough, developmental sequelae can be prevented or minimised (Allen & Capute, 1989). If the feeding specialist should detect any signs of jitteriness or convulsions, she/he should report it to the medical staff to investigate. If the metabolic condition is known, the feeding specialist should plan evaluation and management accordingly.

- *Respiratory problems*

One of the respiratory problems the premature infant can have, is **respiratory distress syndrome (RDS)** or **Hyaline membrane disease (HMD)**. This disease most commonly affects the premature infant - 70% of infants born between 28-30 weeks, 60% born before 32 weeks and only 10% of those born between 34-36 weeks develop HMD. Respiratory problems constitute the single largest cause of death in the neonatal period (Rossetti, 1996). The lungs have not developed enough surfactant, a protein that moisturises the lungs to reduce the surface tension. Due to the low surface tension the alveoli collapse during expiration. The infant has to work much harder to reinflate the lungs and needs much higher

energy expenditure. These infants therefore fatigue very quickly, which inhibits their neuromotor and feeding development. HMS can lead to low oxygenation, asphyxia, metabolic acidosis and acute respiratory failure, which can be fatal, unless the infant is mechanically ventilated. The ventilator will provide the lungs with sufficient oxygen at the right pressure to keep the lungs open. Being on a ventilator will delay the premature infant's transfer to oral feeding further and may expose him/her to the complications of long-term nasogastric feeding as discussed earlier (Garg et al., 1988; Vergara, 1993). Feeding problems of infants suffering from cardio-respiratory problems will be discussed in more detail later.

**Asphyxia, Anoxia and Hypoxemia** are other respiratory complications the premature infant may experience. **Asphyxia** means that the infant experienced a shortage of oxygen as a result of placenta abruptio, prolonged separation of the placenta, or substance abuse. Asphyxia can cause metabolic disturbances or result in an inadequate exchange of oxygen and carbodioxide, which is called **anoxia**. The results of **anoxia** can be non-progressive neurologic deficits, mental retardation and cerebral palsy (spasticity, choreoathetosis and ataxia), deafness and seizures. The impact the anoxia has on the brain is called **hypoxemia**, which leads to Hypoxic Ischemic Encephalopathy (HIE). In severe cases the infant will lapse into a coma. The survivors suffer significant neurologic impairments and associated dysphagia. In moderate cases, they may be lethargic and hypotonic and need short-term ventilation, which may again influence their feeding abilities negatively. They may also develop necrotizing enterocolitis (NEC) or acute renal problems. In mild cases the asphyxia could have occurred immediately before birth. The infant is then born lethargic and later on becomes jittery, hyperalert, irritable and exhibits hyperreflexes (Sweeney, 1985; Sheahan & Brockway, 1994). Feeding problems can also be expected in the mild cases, but more in terms of the influences of state and behaviour on feeding skills. They usually have a more optimistic developmental outcome than severe cases of asphyxia (Sheahan & Brockway, 1994).

- *Cardiovascular problems*

**Patent Ductus Arteriosus (PDA).** The infants with PDA have less endurance, as they are unable to increase the heart rate to respond to the increased energy demands needed for the extra efforts whilst feeding or moving. The development of their motor and feeding skills are influenced negatively (Sheahan & Brockway, 1994; Sehnal & Palmeri, 1989).

The efficacy of oral feeding is thus also dependent on the condition of the (premature) infant during and shortly after birth.

### **2.3.1.3 Postnatal Period / Medical Complications**

Although the premature and low birth weight (LBW) infants' survival rates have improved due to improved technology, they are still vulnerable to medical complications as a result of the very treatment that lead to their survival. A variety of medical complications are associated with prematurity (Rossetti, 1996).

- *Bronchopulmonary dysplasia (BPD)*

This is a chronic lung disease related to prematurity, low birth weight and prolonged ventilation. Vohr (1991) states that BPD occurs in 5 - 45% of the premature infants. It can begin as early as 25-26 weeks gestational age. The respiratory tract cilia are destructed by hyperinflation of the lungs during ventilation. Necrosis of the cells of the respiratory epithelium and the cell lining of the alveoli can occur. The oxygen requirements to sustain the lives of these infants may also have deleterious effects on their hearing and vision (as described below Retinopathy of Prematurity), placing them at risk for difficulties in their communication development (Billeaud, 1993). The infants with BPD are very vulnerable to infection during their first year. Recovery is slow and the infants experience a chronic lack of oxygenation, which leads to constant fatigue that will influence their endurance with oral feeding. Rossetti (1996) mentions that it may also cause growth retardation and impair their neuromotor development.

Prolonged hospitalisation negatively effects development of parent-infant relationships, language, feeding and motor skills (Als et al., 1986; Glass & Wolf, 1994; Sheahan & Brockway, 1994; Vohr, 1991).

- *Intraventricular haemorrhage (IVH) and Intracranial haemorrhage (ICH)*

Both conditions are common in infants born under 32 weeks gestational age. Vohr (1991) states that 40% of all low birth weight (LBW) (under 2 500 g) premature infants and Sweeney (1985) says that 50% of the very low birth weight (VLBW) (under 1 500 g) infants develop IVH. The haemorrhage occurs because of the fragile vascular state of the germinal matrix and because of the poorly developed cerebral auto-regulation. Semmler (1989) states that the fluctuation in intra-cranial pressure can be a contributing factor in ICH. The infants with an unstable respiratory status, pneumothorax, swinging blood pressure, or hypoxemia, are at a greater risk of developing IVH. Cellular damage from IVH may occur in areas of the brain causing attention deficits, low or high activity, diminished affect and less skilled motor activity, which may interfere with social interaction (Oehler et al., 1996). The severity of the IVH is related to the incidence of major neurologic abnormalities. Grade I (less than 10% of the ventricle area is bleeding) and grade II (between 10 and 50% of the ventricle area is bleeding) have low long-term risks for neurologic deficits. Grades III and IV (more than 50% of the ventricle area is bleeding) are at high risk for neurologic deficits, hydrocephalus, mental retardation, seizures, deafness or microcephaly (Sheahan & Brockway, 1994; Sweeney, 1985). The neurological impairment suffered by the infant with Intraventricular or Intracranial haemorrhage can cause different degrees of feeding difficulties, which should be evaluated comprehensively to plan effective feeding intervention. It is also believed that excessive handling, movement and stress can trigger ICH (Hunter et al., 1994). It is of the utmost importance that the feeding specialist knows this to avoid being the cause of such serious complications.

- *Periventricular leukomalacia (PVL) follows IVH*

The lesions caused by the bleeding are called PVL and are reduced to cystic cavities. PVL is associated with cerebral palsy, mental retardation, hypertonicity and seizures (Oehler et al., 1996; Sheahan & Brockway, 1994). This means that PVL is also a high risk factor for feeding difficulties.

- *Necrotizing enterocolitis (NEC)*

This is the term used when some of the infant's intestines die because of infection (Thurman & Widerstrom, 1990). NEC is a major complication in the under 2 000 g infant (Sheahan & Brockway, 1994). NEC is a setback in the overall medical progress of an infant and puts him/her under additional stress, which interferes with the neurodevelopment as well as his/her behavioural progress. During the illness the infant with NEC is only on parenteral feeding – no oral feeding is permitted. Depending on the severity of NEC, experience in oral feeding will be delayed. The feeding specialist should therefore be aware that long-term feeding problems can develop unless appropriate intervention is planned.

- *Retinopathy of Prematurity (ROP)*

ROP affects 4% of infants weighing less than 1 000 g (Glass & Wolf, 1993). ROP is the main cause of blindness in premature infants. Oxygen therapy can cause abnormal growth of blood vessels in the immature retina. Most of the abnormal vessels will heal themselves, but in severe cases permanent scar tissue can develop, causing permanent damage. Partial healing can result in night blindness and strabismus (Sheahan & Brockway, 1994; Thurman & Widerstrom, 1990). Glass & Wolf (1993) name blood transfusions, hypo- or hypercarbia, sepsis and light as further possible causes of ROP. The visually impaired infant does not reach out to objects, or manually explore objects, until a much later age than infants with normal vision. Adults tend to talk less to the visually impaired infant, because of a lack of behavioural clues used by the visually impaired infant. The onset of words occur at a later age for the visually impaired than for the normal infant. Bonding and attachment between the infant and the parents are affected

too (Glass & Wolf, 1993). The speech–language therapist should be aware of the impact of Retinopathy of Prematurity on the infant, as it appears that it is a high risk factor for delays in communication development and should therefore be followed up regularly.

In conclusion, it is clear that medical complications play a significant role in the oral feeding skills and developmental outcome of premature infants (Duffy, Als & McAnulty, 1990).

### **2.3.2 ENVIRONMENTAL / SOCIO-ECONOMIC FACTORS**

Numerous factors interfere with normal environmental interaction that can increase the risk of developmental delay (Rossetti, 1996). It has been found that a mother with a low socio-economic status (SES) is more likely to give birth to a premature infant than other mothers (Thurman & Widerstrom, 1990). Aylward (1990) states that cognitive function is the area most influenced by the environment and Vohr (1991) supports this idea by stating that there is a relationship between family socio-economic status and mental development of children. It was found that development differed significantly between the appropriate for gestational age (AGA) infant and the small for gestational age (SGA) infant. SGA refers to infants whose weights fall below the 10<sup>th</sup> percentile on the growth chart of Lubchenco (1976) (Mullen et al., 1988). The small for gestational age low SES premature infants do not catch up with their development as well as the appropriate for gestational age low SES premature infants do (Vohr, 1991). Traditionally, environmental influences were not monitored well during follow-up sessions, but it has become clear that they are of equal importance as the biological influences (Aylward, 1990). Environmental factors are discussed in the following section in terms of the period in which it may have occurred.

### 2.3.2.1 Prenatal Period

Environmental factors which can influence the infant in utero are:

- *Maternal nutritional status and health*

Maternal viral infections (e.g. TORCH viruses) can have devastating effects on the infant's nervous system, which may cause feeding problems and developmental delays. Maternal illnesses like hypertension, diabetes and lung disease can compromise nutrient and oxygen supply to the infant and thereby increase his risk of Intra-uterine growth retardation (IUGR), premature delivery and/or poor developmental outcome (Allen & Capute, 1989). Pre-eclampsia is a major contributor to premature births and exposure to X-rays can cause microcephaly. The feeding specialist should take a comprehensive history to fully understand and anticipate problems of the premature infant and to be able to plan effective intervention.

- *Maternal age*

Adolescent mothers, especially under 16 years, are at risk of giving birth to an infant with mental retardation (Rossetti, 1986; Sweeney, 1985; Thurman & Widerstrom, 1990; Wilcox, et al., 1989). Mothers older than 35 years are at greater risk of having an infant with Down's syndrome (Thurman & Widerstrom, 1990).

- *Maternal substance abuse*

If the mother consumes more than 2 fluid ounces of alcohol a day during pregnancy, the infant might display poor motor control, mental retardation, facial dysmorphism, pre- and postnatal growth deficiencies, congenital hip dislocation and attention deficit disorder and be classified as Foetal Alcohol Syndrome (FAS). The risk for the above-mentioned problems can be aggravated if the mother smokes as well. Cocaine abuse may not be such a widespread problem in South Africa, but should be known for its effects, which are: Low birth weight (LBW),



Intra-uterine growth retardation (IUGR), a small head circumference, prematurity and haemorrhagic infarctions. Although Sweeney (1985) mentions that the long-term effect of cocaine is still unknown, the problems associated with LBW, IUGR, etc. are well known. Alcohol and substance abuse can cause congenital anomalies, dysmorphic features and developmental delays. Infants born from addicted mothers tend to have excessive sucking behaviour (Sheahan & Brockway, 1994).

The prenatal environment proves to be an important factor for the oral feeding skills and development of the neonate.

### **2.3.2.2 Perinatal Period**

The environment in which the infant is born may influence the developmental outcome of an infant; for example, if an infant is born before arrival at the hospital, he/she may be at risk for infections. Infections may cause serious illness, which may or may not have long-term effects on the infant. If the infant needed any kind of medical assistance, e.g. suctioning or oxygen, just after birth, the infant is at risk of asphyxia, with all its associated sequelae, as discussed earlier.

### **2.3.2.3 Postnatal Period**

After birth of an infant, several factors can influence his developmental outcome, e.g.:

- *Health care of infant*

Prolonged or chronic hospitalisation heightens the risk for developmental delays and poor parent-child relationships (Sweeney, 1985). Poor nutrition of an infant after hospital discharge can impede development. Unsolved feeding problems in the NICU can cause Failure to thrive (FTT) infants. That is why proper management of feeding problems in the hospital and collaboration with the parents from the earliest days are important.

- *Maternal educational and mental status*

Both play a roll in the level of stimulation, as well as the opportunities for stimulation, that the premature infant will receive (Aylward, 1990; Sweeney, 1985; Vohr, 1991; Wilcox et al., 1989). The quality of the *mother-infant interaction*, competency in parenting roles, poor bonding and the irritable temperament of the premature infant can have a negative influence on the ability of the premature infant to catch up growth and development (Aylward, 1990; Sweeney, 1985). According to Rossetti (1996), the mortality of infants declines with increasing maternal education. This should then also be part of the history when assessing an infant for developmental delays. It might not have a direct influence on the feeding abilities of the infant, but possibly on the nutritional management of the infant.

- *Birth order and Gender*

The infants with a later birth order and male infants are at higher risk of communication developmental delays (Ogletree & Daniels, 1993). Rossetti (1996) states that second-born infants have a lower mortality. Aylward (1990) says that there is evidence that the environmental effects on cognition are stronger in females than in males. Cognition and language are strongly related in the first two years (Rossetti, 1898). The apparent difference in opinion on gender differences may be explained by the fact that males seem to be genetically more prone to language delays, but may be affected less by an unfavourable environment than females.

Thurman & Widerstrom (1990) state that socio-economic status (SES) is the best predictor of later intelligence scores. Aylward (1990) supports this idea by stating that the environment affects cognitive functioning more strongly than motor or neurologic functioning. The relationship between environmental and biological factors is explained by using the “signal-to-noise ratio” analogy. It can be said that the “signal” is the early medical and biological influence on outcome, which is

gradually obscured by the “noise” generated by the environmental influences (Aylward, 1990). Children with both biological and environmental risk factors are considered to be at double hazard for delay (Rossetti, 1996), or sometimes referred to as double jeopardy (Aylward, 1990).

### **2.3.3 ESTABLISHED / MEDICAL FACTORS**

These factors refer to a medical disorder of known aetiology, which has a predictable pattern of developmental performance and is usually described as a syndrome, e.g.: Cornelia de Lange syndrome or Prader Willi syndrome (Allen & Capute, 1989; Aylward, 1990; Vohr, 1991). Other factors are congenital malformations, central nervous system disorders, chromosomal abnormalities, hydrocephaly, microcephaly, genetic/heredity conditions (15-25% of developmental disorders are related to genetic disorders), inborn errors of metabolism, musculoskeletal abnormalities, multiple births, and recurrent neonatal seizures (more than 3 seizures in the neonatal period) (Sweeney, 1985; Thurman & Widerstrom, 1990).

The developmental outcome of these infants is based on a continuum of events rather than on a single biological, social or environmental factor. The continuum begins at the time of conception and may be modified by a complex interaction between biological and environmental factors in the intrauterine climate, the neonatal period and early childhood years (Vohr 1991). Rossetti (1996) does not regard these infants as being at risk, because it is known that developmental delays will arise. But since early intervention still has a positive influence on these children and will better their outcome, even if it won't be normal, it can be argued that they are also at risk – at risk of a poorer outcome without intervention. Thurman & Widerstrom (1990) state that the developmental outcomes of these infants are strongly influenced by the socio-economic circumstances of the child's environment, which may be even stronger than earlier biological events.

Early intervention may or may not contribute to catch up growth and development, but the most important opportunities to recover and catch up have to be provided for the premature infant. Only through a thorough understanding of the premature infant's medical conditions and the developmental risks they display, can effective accountable service be provided to them, with feeding therapy as part of the intervention.

## **2.4 RISK FACTORS FOR FEEDING PROBLEMS IN PREMATURE INFANTS**

Premature infants are not only at risk for developmental delays, but they are also at risk for feeding problems, specific to their circumstances. Premature infants have an overall weak and irritable state, which can decrease the quality of their oral-motor skills and the quantity of their oral intake (Morris & Klein, 1987). The stability of the autonomic, motor and state systems is a prerequisite for oral feeding. So is the maintenance of an awake and alert state, which is problematic for the premature infant. Feeding problems can arise from both neurobehavioural disorganisation and the actual sucking mechanism itself (Hunter, 1993). The feeding specialist must be alert to the following factors that may suggest feeding problems or may influence the oral feeding skills of premature infants.

### **2.4.1 MEDICAL HISTORY**

Aspects from the medical history which should be considered by the feeding specialist when assessing and managing the oral feeding problems of the premature infant, are:

- *Polyhydramnios*

The foetus can swallow amniotic fluid from 16 weeks. If the swallowing of the infant is impaired, the amniotic fluid seems to increase. The excessive amount of

amniotic fluid is called polyhydramnios. This information from a comprehensive history will alert the feeding specialist to the possibility of dysphagia and possible neurological problems, because one of the reasons for a swallowing problem (dysphagia) can be an insult on the CNS. It can also be indicative of a tracheoesophageal fistula (TOF) (Brodsky, 1997), which causes serious feeding problems.

- *Substance abuse*

Cocaine or heroin abuse by the mother causes excessive sucking behaviour in the infant, but the sucking is inefficient. The infants are therefore poor feeders despite their strong sucking pattern (Sheahan & Brockway, 1994).

- *Facial anomalies*

If the premature infant has anomalies like a cleft palate or Pierre Robin sequence, feeding problems normally associated with such an anomaly can be expected.

- *Low birth weight (LBW)*

LBW infants have little body and facial fat. The facial fat must provide the infant with sucking pads in the cheeks which provide stability for the sucking movements. The premature LBW infant has diminished sucking pads. Without this stability, sucking is inefficient and the infant has to spend more energy to consume his oral feeds (Morris & Klein, 1987; Glass & Wolf, 1994). The additional energy expenditure may limit appropriate weight gain. The feeding specialist should constantly, in co-ordination with the medical staff, determine the balance between access to feeding experience and sufficient weight gain. Although Bu'Lock et al. (1990) regard feeding failure to be a result of neuromuscular immaturity rather than a lack of postnatal sucking experience, but sucking experience for the infant who is mature enough to co-ordinate his sucking, swallowing and breathing is very important according to Bazyk (1990) and Vergara (1993).

- *Long hospitalisation*

The hospital routine can have a negative impact on the feeding of the infant, because the infant's biorhythms are ignored in the NICU. The hunger sensation facilitates sucking (Morris & Klein, 1987). If the infant has to be fed at a certain time according to the hospital routine, but he is not hungry yet, poor sucking will be demonstrated (Cagan, 1995). The maintenance of an awake and alert state, with which the premature infant has difficulty, is a prerequisite for oral feeding (Mandich & Ritchie, 1996; Sheahan & Brockway, 1994; Vergara, 1993). If the scheduled feeding time does not correlate with the infant's awake cycle, sucking will be weak and inefficient. In both cases the feed will be administered by tube and valuable sucking experience will be lost. Another factor that places the infant at risk for feeding problems is that hospitalisation implies multiple caregivers with inconsistent feeding techniques (Bernbaum & Hoffman-Williamson, 1991).

A feeding specialist can thus be alerted to possible oral feeding problems by studying a complete medical history of the premature infant.

## **2.4.2 NEUROLOGICAL FACTORS**

Oral feeding skills may be affected by several neurological factors, namely:

- *Immaturity*

The premature infant is not ready to adapt to the extrauterine life and is neurologically not ready to feed orally. Firstly, the co-ordination of sucking, swallowing and breathing only develops after approximately 34 weeks gestational age (Dreier et al., 1979; Bernbaum & Hoffman-Williamson, 1991; Brake, Alfasi & Fleischman, 1988). Secondly, the oral reflexes are diminished or absent (Bernbaum & Hoffman-Williamson, 1991). The gag reflex, which should protect the penetration of foreign objects into the larynx, develops at approximately 35 weeks gestational age. The young infant might not cough when he chokes, but will react with apnea (Loughlin, 1989). The risk of aspiration and apnea exists and

therefore makes oral feeding before 35 weeks unsafe. Thirdly, the premature infant cannot organise and integrate all the stimulation involved in oral feedings (Als et al., 1988). The infant becomes stressed, which disorganises the movements required for oral feeding, with the result that the infant becomes overloaded, refuses to eat, and/or falls asleep. Generally, it should be stated that oral feeding before 34 weeks gestational age will be unsafe and inappropriate for the premature infant (Morris & Klein, 1987; Glass & Wolf, 1994).

- *Insults on the Central Nervous System (CNS)*

This is associated with prematurity and with feeding problems. The infant with a neurologic insult, such as IVH or ICH, is at risk for feeding apnea and should therefore be monitored closely for any sign of feeding apnea so as to avoid hypoxemia (Rosen et al., 1984). In IVH and ICH, the sequelae from the bleeding also have an impact on the neurodevelopmental parameters, of which oral feeding is one. The impact on the feeding involves the motor aspects, oral-motor control and postural control needed for oral feeding. Cerebral palsy (CP) is a frequent outcome of ICH or IVH. Abnormal feeding patterns are often an early marker for potential CP (Hunter, 1993; Glass & Wolf, 1994). Repeated hypoxic incidents may lead to further neurologic damage and may therefore also increasingly affect the oral feeding skills of the infant.

- *Hypotonia*

Premature infants are generally hypotonic and lack postural control in the extensor cervical area. This leads to inadequate stability of the head and neck, needed for oral feeding. The low oral muscle tone causes a poor lipseal on the nipple with leakage of milk at the mouth corners (Bernbaum & Hoffman-Williamson, 1991). Sucking is a flexor skill, but the premature infant tends to be positioned in extension. Extension has a negative influence on sucking and swallowing. The strong extensor tone in the head and neck can also cause cheek retraction, which makes it difficult to maintain lip closure during sucking (Morris & Klein, 1987).

It is clear that neurological factors play a significant role in oral feeding skills. The neurological factors may determine when to introduce oral feeding, as well as the manner in which the management of the problems should be handled.

### **2.4.3 CARDIORESPIRATORY FACTORS**

The degree of respiratory illness and the subsequent medical management of the illness are critical factors in potential feeding difficulties (Glass & Wolf, 1994). Problems with respiration as experienced when an infant has congenital pneumonia, RDS or BPD, limit the co-ordination of sucking, swallowing and breathing because there is less respiratory control. As a result, abnormal sucking patterns may occur. The infant uses short sucking bursts and long pauses (Hunter, 1993; Ramsey & Gisel, 1996). The airflow to the lungs is interrupted every time the infant swallows. The time for breathing is halved during oral feeding. Higher respiratory demands upon the infant are made. In response, the infant may gasp for air, which makes the swallowing inefficient and results in the long pauses before he sucks again. In severe cases, these pauses can become an apnoeic event and even hypoxia can occur (Rosen et al., 1984; Glass & Wolf, 1994).

- *Respiratory Distress Syndrome (RDS)*

The need for extra breathing and frequent fatigue in the infant with RDS was discussed earlier on. Feeding requires even more energy and puts more demands on the respiratory system. These infants experience a low endurance for oral feeding because of the additional oxygen consumption and respiratory reserve that are needed for oral feeding (Bernbaum & Hoffman-Williamson, 1991; Glass & Wolf, 1994).

- *Broncho-Pulmonary Dysplasia (BPD)*

Apart from respiratory problems in the infant with BPD, feeding problems occur very often. Chronic lung disease is a risk factor for feeding problems (Bernbaum &



Hoffman-Williamson, 1991), because motor, tactile and behavioural problems can develop based on the medical interventions that have to be followed for BPD. Garg et al. (1988) found that severe desaturations occurred during feeding of the BPD infant. That could explain why these infants in general have a low endurance, but also for feeding. Infants with BPD are irritable, have poor co-ordination of sucking, swallowing and breathing and may have abnormal oral-motor patterns (Hunter, 1993; Glass & Wolf, 1994). Chronic pulmonary disease leads to poor oral intake that causes low protein energy, resulting in malnutrition. Malnutrition impairs the immunologic response to infections. This can negatively affect the growth of the young central nervous system, which is highly undesirable at this stage of rapid brain growth (Tuchman, 1989). These problems have a negative influence on oral feeding and limit optimal nutritional intake, which in turn lead to poor weight gain (Semmler, 1989; Vohr, 1991; Glass & Wolf, 1994).

- *Mechanical ventilation*

The infant receiving mechanical ventilation is unable to feed orally. Long deprivation of oral feeding limits normal sucking experiences. The sucking reflex is not facilitated and may diminish with time. Prolonged use of endotracheal tubes can cause hyperextension of the head and neck, because that position is used to ensure a more open airway, but the extension position gives little stability for sucking and causes wide jaw excursions. These infants often experience hypersensitivity of the mouth, because of the early negative experiences of intubation. The infant is deprived of the normal pleasurable oral sensations and may develop an aversion towards any oral contact, including feeding (Bernbaum & Hoffman-Williamson, 1991; Glass & Wolf, 1994). The hypersensitivity can also disorganise the early suck-swallow patterns (Morris & Klein, 1987). If sucking is present, abnormal patterns may develop to accommodate the shape and position of the endotracheal tube. The endotracheal tube may also cause damage to the structures of the larynx, causing a possible incomplete protection of the airway during swallowing, which may lead to micro-aspiration (Wolf & Glass, 1994).

- *Heart diseases: VSD, PDA and Cyanotic heart disease.*

These infants are unable to increase their heart rate required for the extra energy expenditure needed for oral feedings. They tire quickly and become tachypneic. They may not arouse to feed and do not have the strength to feed orally. They experience early satiety because of their poor appetite, slow gastric emptying and decreased gastro-intestinal motility. They show poor growth and have a poor nutritional status (Wolf & Glass, 1994).

The feeding specialist has to consider all of these factors carefully when feeding an infant with cardio-respiratory problems.

#### **2.4.4 GASTRO-INTESTINAL FACTORS**

Problems in the gastro-intestinal tract of premature infants may influence their oral feeding skills.

- *Gastro-oesophageal reflux (GER)* (Bernbaum & Hoffman-Williamson, 1991)

Infants who often experience regurgitation, associate the unpleasant feeling of GER with feedings and develop an aversive reaction towards oral feedings. In severe cases the infant can develop oesophagitis, which makes it very painful for the infant to swallow and feeds will be refused in an attempt to avoid the pain (Rosenthal et al., 1995; Glass & Wolf, 1994). Micro-aspiration can also occur with GER (Hunter, 1993). If GER is suspected, it has to be medically managed for oral feeding to be optimal.

- *Necrotizing enterocolitis (NEC)*

NEC results in a general weak state of the infant. The infants feed on hyperalimentation (Wolf & Glass, 1994). NEC can cause long-term feeding and behavioural problems (Sehna & Palmeri, 1989). Feeding problems may imply difficulties with the quantity and type of milk that can be tolerated by the intestines as well as problems with the sucking mechanism itself. The cause of the

problematic sucking patterns has to do with the deprivation of the oral sucking experience and the effects of long-term tube feedings in these infants.

- *Long-term nasogastric feedings*

This can be an early signal of a neurodevelopmental dysfunction (Glass & Wolf, 1994). Although premature infants are initially dependent on nasogastric feeds as they are unable to meet all of their nutritional needs orally, the long-term use of nasogastric feedings may create several problems. These problems are similar to the aversive reactions that may result from endotracheal intubation, as discussed earlier (Bazyk, 1990). Aversive reactions towards oral feeding can develop because of negative associations with unpleasant medical procedures like intubation, sucking procedures and naso- or orogastric tubes being inserted (Bazyk, 1990; Bernbaum & Hoffman-Williamson, 1991). The nasogastric tubes may cause nasal and pharyngeal irritation and provide a negative stimulus every time the infant swallows (Bazyk, 1990; Jaffe, 1989). The hypersensitivity in the oral area can disorganise early sucking-swallowing patterns (Averdson & Brodsky, 1993; Bazyk, 1990; Morris & Klein, 1987; Rosenthal et al., 1995). Breathing patterns may also be altered due to the partial obstruction caused by the tubes in the nasal cavity (Bazyk, 1990; Jaffe, 1989). The aversive behaviour may lead to failure to thrive in more severe cases (Bazyk, 1990; Geertsma et al., 1985; Mandich & Ritchie, 1996). Premature infants are usually fed by nasogastric tubes until they are ready to transfer to the bottle or breast. Although their medical status is good and their weight acceptable, they often do not have the oral-motor skills and co-ordination of sucking, swallowing and breathing to feed effectively. Prolonged use of the nasogastric tube can have negative influences on the development of oral feeding skills (Bazyk, 1990; Mandich & Ritchie, 1996; Morris, 1989; Palmer & Heyman, 1993; Vergara, 1993).

Clinical experience has shown that the period of time spent by many premature infants in the neonatal unit is dependent on the time they need to become successful oral feeders rather than medical reasons. That means that although they are healthy enough to be discharged, they are not able to manage all of their

feeds orally yet. 25% of the infants ( $\pm$  211 per year) in the NICU of the Pretoria Academic Maternity Hospital stayed in hospital for more than 14 days and only 3% of them for more than 60 days in 1998 (De Witt, 1999). Efficient feeding therapy in the NICU can shorten hospital stay. Prolonged hospital stay heightens the risk for developmental delays and poor parent-child relationships (Sweeney, 1985). Earlier discharge from the hospital thus has several advantages. Firstly, it is cost effective, which may be the most important factor from the hospital administrator's point of view, as medical expenses for NICU care are very high (Petryschen, Stevens, Hawkins & Stewart, 1997). Secondly, it has a positive effect on the parent-infant relationship. The mother has the opportunity to care for her infant sooner and bonding is facilitated. Family routines and stimulation of the infant are enhanced which have a positive influence on the developmental outcome of the infant (Aylward, 1990; Vohr, 1991)

#### **2.4.5 ORAL STRUCTURES**

Premature infants do not have sucking pads, so that the oral cavity appears to be bigger. The tongues of premature infants appear relatively small (because of the bigger oral cavity), have decreased motility and lack stability for sucking. The tongue is often in an elevated position in an attempt to stabilise it. The jaw exhibits exaggerated excursions, interfering with efficient sucking. The premature infant also has a decreased lipseal and depressed oral reflexes, which complicate oral feeding (Morris & Klein, 1987). The feeding specialist should consider these characteristics when planning feeding intervention.

#### **2.5 CONCLUSION**

It is clear that premature infants are different from full-term infants, not only in appearance, but also in the medical conditions and complications that they are prone to and the effects that these can have on their oral feeding abilities and

their developmental outcome. An understanding of the unique characteristics and risk factors that can influence the functioning of the premature infant is vital for the effective and efficient management of their feeding problems. A comprehensive assessment of the premature infant's feeding skills will therefore have to include all aspects discussed in this chapter, either as information collected from the medical history or from direct observation. Only when a thorough assessment of the premature infant as a whole, his medical history and current medical status, state and behaviour, physiological and neurological status and oral-motor skills, has been made, can an appropriate intervention be planned. The requirement of Bu'Lock et al. (1990) to adequately define the premature infant's feeding skills to be able to intervene effectively can be fulfilled in this way.

## **2.6 SUMMARY**

The characteristics of premature infants were described in terms of their general appearance and the different states and systems in which they function. Risk factors for the developmental outcome were examined. The risk factors were divided into three categories, namely biological, environmental and established/medical factors and were discussed according to the period (pre-, peri- or postnatal) in which the factors occurred. The risk factors for feeding problems of premature infants were also discussed. The next chapter will deal with the mechanics of the premature infant's oral feeding skills.