ABSTRACT:-

Aims and objectives: This study will describe the oral health status of critically ill children over time spent in the paediatric intensive care unit, examine influences on the development of poor oral health and explore the relationship between dysfunctional oral health and healthcare-associated infections.

Background: The treatment modalities used to support children experiencing critical illness and the progression of critical illness may result in dysfunction within the oral cavity. In adults, oral health has been shown to worsen during critical illness as well as influence systemic health.

Design: A prospective observational cohort design was used.

Method: The study was undertaken at a single tertiary-referral Paediatric Intensive Care Unit. Oral health status was measured using the Oral Assessment Scale and culturing oropharyngeal flora. Information was also collected surrounding the use of supportive therapies, clinical characteristics of the children and the occurrence of healthcare-associated infections.

Results: Of the 46 participants, 63% (n=32) had oral dysfunction and 41% (n=19) demonstrated pathogenic oropharyngeal colonisation during their critical illness. The potential systemic pathogens isolated from the oropharynx and included *Candida sp*., *Staphylococcus aureus*, *Haemophilus influenzae*, *Enterococcus sp*. and *Pseudomonas aeruginosa*. The severity of critical illness had a significant positive relationship (p<0.05) with pathogenic and absent colonisation of the oropharynx. Sixty-three

percent of healthcare-associated infections involved the preceding or simultaneous colonisation of the oropharynx by the causative pathogen.

Conclusions: This study suggests paediatric oral health to be frequently dysfunctional and the oropharynx to repeatedly harbour potential systemic pathogens during childhood critical illness.

Relevance to clinical practice: Given the frequency of poor oral health during childhood critical illness within this study and the subsequent potential systemic consequences, evidence based oral hygiene practices should be developed and validated to guide clinicians when nursing critically ill children.

KEYWORDS:-

Oral health, oropharyngeal colonisation, paediatric intensive care, healthcare-associated infection.

INTRODUCTION AND BACKGROUND:-

Critically ill children experience physiological changes which result in instability and acute crises, requiring intensive nursing care to support single organ or systemic dysfunction. Treatment modalities used to support children experiencing critical illness and the progression of critical illness may result in dysfunction within the oral cavity (Johnstone, Spence et al. 2010). In a healthy child, the oral cavity harbours over 250 strains of commensal bacteria which change in response to numerous factors including the child's general health and well-being (O'Reilly 2003; Johnstone, Spence et al. 2010). The changes generated by critical illness may produce an imbalance of commensal oral bacteria, which allows the oral cavity to become a haven for potential systemic pathogens (Fourrier, Duvivier et al. 1998). The anatomical connection between the oral cavity and the respiratory and circulatory systems allows, in some instances, pathogens colonising the oropharynx to cause systemic infections (Munro, Grap et al. 2006).

In the paediatric intensive care environment, respiratory and blood-stream infections caused by fungal and bacterial pathogens are associated with substantial financial costs, as well as increased morbidity and mortality (Safdar, Dezfulian et al. 2005; Suljagic, Cobeljic et al. 2005; Turton 2008; Inwald, Tasker et al. 2009; Thorburn, Jardine et al. 2009; Venkatachalam, Hendley et al 2010). Despite this, research describing the prevalence of fungal and bacterial pathogens in the paediatric oropharynx and their systemic effects during critical is scarce. In comparison, adult critical care research has recognised that poor oral health may have an impact on the morbidity and mortality outcomes of patients (Scannapieco, Stewart et al. 1992;

Abele-Horn, Dauber et al. 1997; Garrouste-Orgeas, Chevret et al. 1997; Fourrier, Duvivier et al. 1998; Brennan, Bahrani-Mougeot et al. 2004; Grap, Munro et al. 2004; Berry and Davidson 2006; Munro, Grap et al. 2006; Chan, Ruest et al. 2007; Jones and Munro 2008; Stonecypher 2010). Adult critical care research is now focussing on treatment strategies which can be used to improve oral health during critical illness.

Interest in the systemic effects of oral health during paediatric critical illness is growing. A recent publication by Thorburn et al. (2009) examined the carriage of abnormal bacterial flora and antibiotic resistant flora in the pharynx and gut of children with cerebral palsy requiring mechanical ventilation, and their associated infection rates. Thorburn found that in 65% of children with cerebral palsy who developed an infection while mechanically ventilated, the infecting pathogen was carried in the patients' pharynx on admission or in their gut flora. They concluded that early targeted antibiotic therapy may be beneficial.

Healthcare Associated Infections (HAI) (previously known as nosocomial infections) are a significant cause of mortality and morbidity for critically ill children (Safdar, Dezfulian et al. 2005; Suljagic, Cobeljic et al. 2005; Turton 2008; Inwald, Tasker et al. 2009; Thorburn, Jardine et al. 2009; Venkatachalam, Hendley et al. 2010). The most common and clinically significant HAI within the PICU population are pneumonia (Schleder 2003; Turton 2008) and blood-stream infection (Jones and Munro 2008).

Within paediatric and adult critical care practice, pneumonia is acknowledged as being a major threat to mechanically ventilated patients. The relationship between

poor oral health, in the form of pathogenic oropharyngeal colonisation, and hospital-acquired pneumonia, has been well documented in robust adult critical care clinical research (Scannapieco, Stewart et al. 1992; Fourrier, Duvivier et al. 1998; Grap, Munro et al. 2004; Berry and Davidson 2006; Jones and Munro 2008). This association is especially prevalent in the setting of ventilator-associated pneumonia (VAP). (Abele-Horn, Dauber et al. 1997; Garrouste-Orgeas, Chevret et al. 1997; Brennan, Bahrani-Mougeot et al. 2004; Munro, Grap et al. 2006; Chan, Ruest et al. 2007; Garcia, Jendresky et al. 2009; Munro, Grap et al. 2009; Stonecypher 2010). Pathogenic microflora that have been isolated in the dental and oropharyngeal flora of critically ill adults, that are also potential microbial causative agents of pneumonia include - *Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae, Pseudomonas aeruginosa* and *Acinetobacter baumannii* (Scannapieco, Stewart et al. 1992; Fourrier, Duvivier et al. 1998; Halm and Armola 2009; Perkins, Woeltje et al. 2010; Venkatachalam, Hedley et al. 2010). Aspiration of pathogenic microorganisms from the oropharynx whilst intubated is a substantial risk factor and contributes to the development of VAP (Garrouste-Orgeas, Chevret et al. 1997; Brennan, Bahrani-Mougeot et al. 2004; Chan, Ruest et al. 2007; Garcia, Jendresky et al. 2009). Previous research fails to adequately address either the risk factors of the progression and resultant consequences of pathogenic colonisation of the oropharynx during childhood critical illness.

Traditionally, oral health and oral hygiene have been given low priority in the nursing care of a critically ill child. Oral hygiene is often neglected or performed inadequately by swabbing the children's mouths for comfort. Currently, within the Paediatric Intensive Care Unit (PICU) at the Royal Children's Hospital (RCH) no oral hygiene

protocol exists to guide nursing practice. Previous researchers in critical care have suggested that nursing practice surrounding oral hygiene is often based on tradition, individual preferences, availability of products, anecdotal or subjective evaluation rather than evidence-based protocols (McNeill 2000; Berry and Davidson 2006; Feider, Mitchell et al. 2010; Johnstone, Spence et al. 2010). No evidence-based oral hygiene protocol has been researched, validated and made available to guide clinicians when nursing critically ill children.

The aim of the study was to describe the status of oral health in critically ill children. To meet this aim, four research questions were developed:

1) What is the status of oral health in critically ill children during admission to a PICU?

2) How does the oral health of critically ill children change during their admission to PICU?

3) How is the oral health of critically ill children affected by patient characteristics or PICU therapies?

4) What is the relationship between dysfunctional oral health in critically ill children and PICU-related HAI?

METHODS:-

Design and participants

The study used a prospective observational cohort design and was conducted at the PICU at the RCH, Brisbane in Australia. This unit provides an eight bed tertiary level PICU for children (aged 0- 14 years) with a catchment which covers Queensland and Northern New South Wales. All patients admitted to the PICU at the RCH within a

seven month period were consecutively screened and recruited for participation after satisfying the inclusion/exclusion criteria:

Inclusion Criteria:

- Clinical condition suggesting a PICU stay greater than or equal to 48 hours;
- Recruitment within 12 hours of admission to PICU;
- All ages, that is both dentate and non-dentate;
- Informed parental/guardian consent and youth assent where required.

Exclusion Criteria:

- Patients who had undergone oral surgery or had an oral condition that required specialised oral care eg. cleft palate repair or oropharyngeal abscess;
- Already participated in the study on a prior admission to PICU;
- Parents or guardian unavailable or unable to give consent due to legal concerns that were under the care of Child and Family Services, or lack of English.

Data collection

The oral health of the study participants was described using the Oral Assessment Scale (OAS) (see Table 1) and second daily culturing of oropharyngeal flora. The OAS is primarily an objective tool and multiple authors (Eilers, Berger et al. 1988; Holmes and Mountain 1993; Barnason, Graham et al. 1998; Andersson, Persson et al. 1999; Jiggins and Talbot 1999; Ferozali, Johnson et al. 2007) have previously undertaken inter-rater reliability testing (r=0.75-0.92) within adult oncology, critical care and paediatrics. The OAS involves assessment over five categories: lips, tongue, saliva, mucous membranes/gingival and teeth. Three levels of descriptors are identified for each category and the overall oral assessment score is the sum of the

subscale scores. The scores from the five categories are calculated with a normal mouth given a score of five, and the highest possible score being 15. Additionally, it can be categorically analysed using a score of five as a no oral dysfunction, six to ten as moderate dysfunction and greater than ten as severe dysfunction. The OAS was carried out by the bed-side nurse every twelve hours, within their initial patient assessment at the commencement of their shift.

Participants had samples of oropharyngeal saliva collected for bacterial and fungal culture completed within 12 hours of admission to PICU and then every second day for the course of their admission to PICU. Nurses were trained individually by the principal investigator and information was placed at the bedside regarding the saliva sampling protocol. The TRANSWAB® was placed in the patient's mouth for a period of at least 30 seconds to allow for saliva absorption, as per the manufacturer's instructions. Swabs were collected at approximately the same time (1000hrs), at least two hours after oral hygiene care or oral intake (feeds, medications, diet) (Sixou, Medeiros-Batista et al. 1998). Samples were then transported to the Queensland Health Pathology Services Laboratory within one hour of collection, for semi-quantitative analysis. In line with previous studies (Rubenstein, Kabat et al. 1992; Scannapieco, Stewart et al. 1992; Fourrier, Duvivier et al. 1998; Thorburn, Jardine et al. 2009; Perkins, Woeltje et al. 2010), microorganisms that commonly cause infection or disease in critically ill children were categorised as 'pathogenic' flora, excluding bacteria which are considered commensal in a paediatric mouth. The diagnosis of colonisation by pathogenic organisms was based on the positive culture of the oropharyngeal saliva swab when greater than or equal to 10^6 colony forming units per litre without signs of clinical infection (Fourrier, Duvivier et al. 1998).

Clinical characteristics such as presence of an endotracheal tube, antibiotic usage, oral hygiene received and length of PICU admission, were collected. Critical illness severity was described using the Pediatric Logistic Organ Dysfunction score (PELOD) score (Leteurtre, Martinot et al. 1999; Lacroix and Cotting 2005; Leteurtre, Duhamel et al. 2006; Thukral, Kohli et al. 2007; Yung, Wilkins et al. 2008; Santanae, Leite et al. 2009), and the Paediatric Indicator of Mortality 2 (PIM2) (Shann, Pearson et al. 1997; Slater, Shann et al. 2002; Slater and Shann 2004; Thukral, Lodha et al. 2006; Eulmesekian, Perez et al. 2007; Wolfer, Silvani et al. 2007; Baghurst, Norton et al. 2008; Inwald, Tasker et al. 2009). The PELOD score is a measure of the severity of multiple organ dysfunction syndrome in the PICU, which is calculated for each patient by adding the scores for individual organ systems based on recorded levels of variables included in the systems. Six organ systems (neurological, cardiovascular, renal, respiratory, haematological and hepatic), each containing multiple variables are stratified into age groups. The PIM2 is a regression model that uses admission data to predict intensive care outcomes for children. The score is designed to be generated using physiological and patient data available within the first hour of the patients' admission, to generate an overall risk-score. The diagnosis and incidence of PICU-related HAI was defined using the Centres for Disease Control and Prevention criteria (Horan, Andrus et al. 2008).

Ethics approval to conduct the study was obtained from the RCH & University Human Research Ethics Committee.

Data analysis

Descriptive statistics were used to describe: oral health; the frequency and type of oral hygiene provided; demographic information; the main clinical characteristics of the participants; and incidence of PICU-related HAI. A time series analyses provided categorical and continuous values to examine oral health over the PICU length of stay. An Analysis of Variance (ANOVA) was used to examine the relationship between dysfunctional oral health and multiple clinical characteristics on day two of admission to PICU. A variety of parametric and nonparametric statistical tests of variance were used depending on normality of distribution and the presence of categorical or continuous variables (Spearman's rho, Kruskal Wallis, Mann-Whitney and Fishers exact test). Statistical analysis was performed using SPSS version 15.0 and statistical significance was set at $p \leq 0.05$.

RESULTS:-

A total of 46 participants were recruited to the study. The demographic characteristics of the study participants are presented in Table 2. As would be expected from a heterogeneous study population, the participants had a wide variety of age, length of PICU stay, dentate status, primary diagnosis, severity of critical illness and admission sources.

1) The status of oral health in critically ill children during admission to a PICU

Within the study, no (0%) participants had severe oral dysfunction during their critical illness, 32 (62.6%) had moderate oral dysfunction and 14 (37.4%) had no oral dysfunction during the course of their critical illness (see Table 3). Nineteen

participants (41.3%) had pathogenic oropharyngeal colonisation during their critical illness.

One hundred and fifty-two oropharyngeal swabs were taken during the course of the study. *Candida sp.* were the most common pathogenic organisms to colonise the oropharynx (46.1%), with *Staphylococcus aureus* frequently present (16.9%) and a range of gram positive and gram negative bacteria less frequently. Eighty percent of participants were colonised with several pathogenic and/or commensal bacteria at the one time. The types and frequency of pathogenic organisms colonising the oropharynx are summarised in Table 4.

The oral hygiene care that the participants received during the course of their critical illness varied widely, as summarised in Table 5. The table shows the most frequent oral cleansing solution used on day two of the participants admission to PICU was water (77.5%), the most frequent oral cleansing implement used was a foam swab (62.5%), and the most common frequency of the provision of oral hygiene was every six hours (40.0%).

2) Changes in the oral health of critically ill children during their admission to a PICU

In order to examine the change of oral health of the participant's critical illness and admission to PICU, two analyses were undertaken. Firstly, the participants were divided into groups using PICU length of stay (group A <48 hours; group B 48-96 hours; group C 97-144 hours; group D 145-192 hours; group E >192 hours). The second analysis was undertaken using the sample as a whole, rather than subgroups.

Using either techniques, there was neither upward nor downward trend in the incidence of pathogenic colonisation or oral dysfunction, nor a change in median OAS over increasing length of stay in PICU.

3) Patient characteristics and PICU therapies affecting the oral health of critically ill children

Data collected on day two of the participants' admission to PICU was used to examine the effect of patient characteristics and PICU therapies on the participants' oropharyngeal colonisation and OAS. The patient characteristics examined were critical illness scores (PIM2 and PELOD), age, dentate status, primary diagnosis, oncological condition and neutropenia. The PICU therapies examined were presence of an oral or nasal endotracheal tube (ETT), antibiotic therapy and oral anti-fungal therapy. A secondary analysis was undertaken to describe the effect admission source to PICU had on oropharyngeal colonisation on day zero of PICU stay. Only one independent variable had a statistically significant effect on the OAS. The critical illness measurement PELOD was significantly associated with oropharyngeal colonisation (x^2=6.166, *df* =2, *p*-value = 0.046).

4) The relationship between dysfunctional oral health in critically ill children and PICU-related HAI

Eight (17.4%) participants developed a PICU-related HAI during their critical illness. Compared with the 38 participants free of HAI, these eight participants had an increased median length of stay in the PICU (*p*=0.002), a higher median OAS on day two of admission to PICU (*p*=ns) indicating moderate dysfunction, and a higher critical illness score (PELOD: *p*=0.072).

Table 6 outlines the types of infection, isolated strains and results of oropharyngeal flora sampling. During their PICU stay, two participants acquired a blood-stream infection or bacteraemia (*Enterococcus faecal is* (*n*=1) and *Escherichia coli* (*n*=1)) without concurrent oropharyngeal colonisation. One participant developed pneumonia (*Pseudomonas aeruginosa*) on day two of admission to PICU, and the same organism was isolated in their oropharyngeal flora four days later.

In the five remaining participants (four with pneumonia, one with bacteraemia), the HAI causative pathogens isolated in their blood or endo-tracheal tube (ETT) aspirates were also isolated from oropharyngeal sampling. In one participant oropharyngeal colonisation occurred simultaneously, while for four participants colonisation with the causative pathogens occurred prior to the development of the HAI. Additionally, one participant developed two PICU-related HAIs during their critical illness and one of their HAI was associated with oropharyngeal colonisation prior to its development.

DISCUSSION:-

The study aimed to describe the status of oral health in critically ill children through use of the OAS and microbial colonisation in the oropharynx. In this study the OAS indicated greater than half of critically ill children had oral dysfunction during their critical illness (n=32; 62.6%). Oral dysfunction manifested in various ways - development of ulcers, dental plaque, cavities, cracked lips, decreased salivary flow, or generalised inflammation and infection. Oral dysfunctions represent a breakdown in the local and systemic health of critically ill children.

Decreased salivary flow, or xerostomia, causes a change in the immunological defences within the oral cavity facilitating adhesion of pathogenic organisms. Cracked lips, generalised infection and ulcers, display a breakdown in the primary defence mechanism of the mouth and this allows infiltration of these pathogenic organisms into the wider circulatory system. In adult populations, a build-up of dental plaque has been shown to be a reservoir for respiratory pathogens, such as *Staphylococcus aureus* and *Pseudomonas aeruginosa* (Scannapieco, Stewart et al. 1992; Fourrier, Duvivier et al. 1998). Considering this, it is possible that oral dysfunction via each of these mechanisms potentially contributes to the systemic health of children during critical illness. Oral dysfunction, as an indicator of poor oral health, has been shown to be equally prevalent in adult critical care studies (Scannapieco, Stewart et al. 1992; Munro, Grap et al. 2006).

Oral dysfunction (as quantified by the OAS) may be symptomatic of underlying microflora changes. Forty-one percent of critically ill children in this study had pathogenic oropharyngeal colonisation during their critical illness which reinforces previous work by Thorburn (2009). The most common pathogenic oropharyngeal colonisation was with *Candida sp*., with similar prevalence seen in Singhi et al's (2008) descriptive study examining generalised Candidaemia in the PICU. *Candida sp.* are frequently present in the oropharynx of healthy children (Hannula, Jousimies-Somer et al. 1999) as part of commensal flora. However within this study the *Candida sp.* were considered pathogenic when prevalent in high numbers ($\geq 10^6$ colony forming units (cfu) per litre) indicating opportunistic colonisation. *Candida* sp. frequently are opportunistic pathogens of blood-stream and respiratory infections in critically ill patients and those with compromised immune status (Singhi, Raman Rao et al. 2008).

In addition to *Candida* sp., the other potential respiratory and systemic pathogens colonising the oropharynx of critically ill children during this study included *Staphylococcus aureus*, *Haemophilus influenzae*, *Enterococcus* sp., and *Pseudomonas aeruginosa.* These findings are in accordance with previous adult and paediatric studies (Rubenstein, Kabat et al. 1992; Scannapieco, Stewart et al. 1992; Garrouste-Orgeas, Chevret et al. 1997; Fourrier, Duvivier et al. 1998; Thorburn, Jardine et al. 2009; Perkins, Woeltje et al. 2010). Respiratory and systemic pathogens are not usually prominent members of the oral commensal flora of healthy adults (Scannapieco, Stewart et al. 1992) or children (Kononen 2005). This suggests the oropharynx of critically ill child may act as a reservoir for pathogens which potentially cause systemic infections (eg. Pneumonia, blood-stream infection) and consequently increase morbidity and mortality (Safdar, Dezfulian et al. 2005; Chan, Ruest et al. 2007; Jones and Munro 2008).

Within this study, information surrounding oral care practices was collected by audit. While this may not be completely reflective of actual practice, the nurses caring for these children demonstrated wide variation in oral hygiene practices. A large proportion of the critically ill children described in the study had oral dysfunction and the nurses' practice surrounding choice of instruments, solutions and frequency of oral care was not informed by patient characteristics or current research. Like all patient management practices, oral health can be best managed through the institution of evidence-based oral hygiene protocols specific to critically ill children.

Our findings which suggest there is no change (either positively or negatively) surrounding oral health over PICU admission were in contrast to other critical care studies. The previous descriptive study completed by Franklin et al (2000) found a statistically significant increase in mean plaque scores during PICU stay and gingival inflammation. Fourrier et al (1998), in their descriptive study on adult critical care, also found a statistically significant increase in dental plaque on patients remaining ICU for five days or greater. Their study also found the frequency of colonisation by aerobic pathogens increased over length of ICU stay but the number of participants in each group was too low to reach statistical significance.

Literature suggests that PICU therapies including intubation (Jiggins and Talbot 1999), antibiotics (Sixou, Medeiros-Batista et al. 1996; Jiggins and Talbot 1999), and patient characteristics including age (Kononen 2000), severity of critical illness (Rubenstein, Kabat et al. 1992; Thorburn, Jardine et al. 2009), admission source (Toltzis, Hoyen et al. 1999), neutropaenia (Sixou, Medeiros-Batista et al. 1996) and admission diagnosis (Sixou, Medeiros-Batista et al. 1996; Thorburn, Jardine et al. 2009) would have a relationship with the oral health of critically ill children. Unexpectedly, the majority of patient characteristics and PICU therapies examined in this study had little or no relationship with the status of oral health of participants. An increase in the severity of critical illness, as measured by the PELOD, demonstrated a statistically significant positive association (p=0.046) with pathogenic or absent oropharyngeal flora, in comparison to commensal flora. It has been established in adult studies that severe illness alters the oropharyngeal flora (Scannapieco, Stewart et al. 1992; Fourrier, Duvivier et al. 1998; Thorburn, Jardine et al. 2009). The findings of this study correlate with the study by Rubenstein et al. (1992), which found that

PICU patients who were colonised orally with pathogenic microflora such as *Candida* sp. had higher levels of critical illness.

Of the population described by this study, 17.4% (eight) developed a PICU-related HAI during their critical illness. HAIs are a common, serious problem in critically ill children and are associated with substantial morbidity and mortality along with increased attributable costs. Within this study, in comparison to the participants who did not develop a PICU-related HAI, this group had higher critical illness scores (PELOD: p=0.072) and longer length of admission (p=0.002). Notably within the scope of this study, the participants who developed a PICU-related HAI had moderate dysfunction on day two of admission to PICU in comparison to no dysfunction in the remaining PICU population. However this relationship did not reach statistical significance and sample size does not allow further statistical analysis. Despite this, the trend towards poor oral health in critically ill children who developed a PICU-related HAI may indicate a potential relationship between the development of HAI and poor oral health. Adult critical care studies have confirmed that poor oral health (Munro, Grap et al. 2006), and pathogenic oropharyngeal colonisation (Pugin, Auckenthaler et al. 1991; Abele-Horn, Dauber et al. 1997; Garrouste-Orgeas, Chevret et al. 1997), increases the risk of HAI, such as pneumonia. Further research regarding this area in paediatrics is required before any causal relationships can be suggested.

Not all participants in this study who had pathogenic oropharyngeal colonisation during their admission to PICU developed a PICU-related HAI. However, of the eight participants who did develop a PICU-related HAI, six participants (75%; five pneumonias, one blood-stream infection) had the causative pathogens isolated from

oropharyngeal sampling previously or simultaneously. While these results are limited by small sample size, they reflect previous findings in adult critical care by Fourrier et al (1998) and Munro et al. (2006). Also, the study results suggest that the oropharynx of critically ill children could be a reservoir of potential systemic bacterial and fungal pathogens. PICU-related HAI as a result of translocation of pathogens from the oropharynx to the respiratory and cardiovascular systems are physiologically plausible. Oral hygiene treatment strategies should be directed towards reducing the prevalence of pathogenic oropharyngeal colonisation and improving oral health.

Limitations of this study include its observational design and low sample size (n=46). Consequently the study lacks the ability to generate powered correlations (Bhopal 2008; Friis and Sellers 2009) and is limited in its generalisability. However, the study was intended to be exploratory. Considering the paucity of current literature surrounding the oral health of critically ill children, this study has broadened the body of knowledge available on the subject.

While limitations are present in the study, it is the first of its kind to fully describe the oral health of critically ill children. In comparison to previous studies, oral health was systematically described using a validated assessment scale in combination with microbiological analysis.

CONCLUSION:-

Oral health has the potential to influence systemic health during critical illness in childhood. This study has indicated that oral health is frequently dysfunctional and the oropharynx frequently harbours potential systemic pathogens during childhood

critical illness. It is worrying then, that PICU nurses within this study had variable oral hygiene practices, some of which were not supported by current research. The only clinical characteristic which had a relationship with dysfunctional oral health described in this study, was severity of critical illness. This had a significant positive relationship with pathogenic or absent colonisation of the oropharynx. In contrast to previous research (Fourrier, Duvivier et al. 1998; Franklin, Senior et al. 2000) the oral health of critically ill children admitted to the RCH PICU did not worsen over length of PICU stay. In addition to the physiological plausibility of translocation of oropharyngeal flora to the blood stream and respiratory tract, this study found a large percentage of PICU-related HAI involved preceding or simultaneous colonisation of the oropharynx by the causative pathogen. While further study is required to determine the full merit of the conclusions generated in this study, given their potential impact upon clinical practice, further investigation appears warranted.

RELEVANCE TO CLINICAL PRACTICE:-

It is inevitable that some children in the general population will become critically unwell and require intensive treatment in a PICU. Whilst critically ill, some children may have characteristics and receive PICU therapies which put them at an increased risk for poor oral health and pathogenic oropharyngeal colonisation. The oral cavity is fragile, and because of this, may easily become dysfunctional and harbour pathogenic microorganisms. Pathogenic microorganisms, such as *Staphylococcus aureus*, can cause severe systemic illnesses including pneumonia and blood-stream infection.

The prevalence of poor oral health during childhood critical illness in the RCH PICU population, combined with potential systemic consequences supports the development

of evidence-based paediatric oral hygiene practices. The development of this evidence-based practice should include well-controlled clinical trials incorporating all aspects of oral care interventions including solution, instruments and frequency. A summary of these interventions could then be used to develop a protocol for appropriate clinical practices. Preferably, the protocol should involve regular oral assessment utilising a validated oral assessment scale, be tiered by severity of critical illness and be governed by the practical elements of dentate status, conscious level, intubation status and developmental age.

CONTRIBUTIONS:-

Study Design: AU, DL & PL

Data Collection and Analysis: AU

Manuscript Preparation: AU, DL & PL

CONFLICT OF INTERESTS:-

None to declare

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REFERENCES:-

Abele-Horn, M., A. Dauber, et al. (1997). "Decrease in nosocomial pneumonia in ventilated patients by selective oropharyngeal decontamination (SOD)." Intensive Care Medicine **23**: 187-195.

Andersson, P., L. Persson, et al. (1999). "Testing an oral assessment guide during chemotherapy treatment in a Swedish care setting: a pilot study." Journal of Clinical Nursing **8**: 150-158.

Baghurst, P., L. Norton, et al. (2008). "The application of the risk-adjusted control charts using the Paediatric Index of Mortality 2 for monitoring paediatric intensive care performance in Australia and New Zealand." Intensive Care Medicine **34**(7): 1282-8.

Barnason, S., J. Graham, et al. (1998). "Comparison of two endotracheal tube securement techniques on unplanned extubation, oral mucosa, and facial skin integrity." Heart and Lung **27**(6): 409-417.

Berry, A. and P. Davidson (2006). "Beyond comfort: Oral hygiene as a critical nursing activity in the intensive care unit." Intensive and Critical Care Nursing **22**: 318-328.

Bhopal, R. (2008). Concepts of epidemiology: integrating the ideas, theories, principles and methods of epidemiology. Oxford, New York, Oxford University Press.

Brennan, M., F. Bahrani-Mougeot, et al. (2004). "The role of oral microbial colonization in ventilator-associated pneumonia." Oral Surgery Oral Medicine Oral Pathology Oral Radiology And Endodontics **98**(6): 665-671.

Chan, E., A. Ruest, et al. (2007). "Oral decontamination for prevention of pneumonia in mechanically ventilated adults: systematic review and meta-analysis." British Medical Journal **334**: 889-900.

Eilers, J., A. Berger, et al. (1988). "Development, testing and application of the oral assessment guide." Oncology Nursing Forum **15**(3): 325-330.

Eulmesekian, P., A. Perez, et al. (2007). "Validation of pediatric index of mortality 2 (PIM2) in a single pediatric intensive care unit of Argentina." Pediatric Critical Care Medicine **8**(1): 54-57.

Feider, L., P. Mitchell, et al. (2010). "Oral care practices for orally intubated critically ill adults." American Journal of Critical Care **19**(2): 175-83.

Ferozali, F., G. Johnson, et al. (2007). "Health benefits and reductions in bacteria from enhanced oral care." Specialised Care for Dentistry **27**(5): 168-176.

Fourrier, F., B. Duvivier, et al. (1998). "Colonization of dental plaque: a source of nosocomial infections in intensive care unit patients." Critical Care Medicine **26**(2): 301-308.

Franklin, D., N. Senior, et al. (2000). "Oral health status of children in a Paediatric Intensive Care Unit." Intensive Care Medicine **26**: 319-324.

Friis, R. and T. Sellers (2009). Epidemiology for Public Health Practice. Sudbury, Massachusetts, Jones and Bartlett Publishers.

Garcia, R., L. Jendresky, et al. (2009). "Reducing ventilator-associated pneumonia through advanced oral-dental care: a 48-month study." American Journal of Critical Care **18**(6):523-32.

Garrouste-Orgeas, M., S. Chevret, et al. (1997). "Oropharyngeal or gastric colonisation and nosocomial pneumonia in adult Intensive Care Unit Patients." American Journal of Respiratory and Critical Care Medicine **156**: 1647-1655.

Grap, M., C. Munro, et al. (2004). "Duration of action of a single, early application of chlorhexidine on oral microbial flora in mechanically ventilated patients: a pilot study." Heart and Lung **33**(2): 83-91.
Halm, M. and R. Armola (2009). "Effect of oral care on bacterial colonization and ventilator-associated pneumonia." American Journal of Critical Care **18**(3): 275-8.
Hannula, S., H. Jousimies-Somer, et al. (1999). "Age-related acquisition of oral and nasopharyngeal yeast species and stability of colonization in young children." Oral Microbiology and Immunology **14**: 176-182.
Holmes, S. and E. Mountain (1993). "Assessment of oral status: evaluation of three oral assessment guides." Journal of Clinical Nursing **2**: 35-40.
Horan, T., M. Andrus, et al. (2008). "CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting." American Journal of Infection Control (AJIC) **36**(5): 309-328.
Inwald, D., P. Tasker, et al. (2009). "Emergency management of children with severe sepsis in the United Kingdom - the results of the Paediatric Intensive Care Society sepsis audit." Archives of the diseases of childhood **Jan 8**: publish ahead of print.
Jiggins, M. and J. Talbot (1999). "Mouth care in PICU." Paediatric Nursing **11**(10): 23-26.
Johnstone, L., D. Spence, et al. (2010). "Oral hygiene care in the pediatric intensive care unit: practice recommendations." Pediatric Nursing **36**(2): 85-96.
Jones, D. and C. Munro (2008). "Oral care and the risk of bloodstream infections in mechanically ventilated adults: a review." Intensive and Critical Care Nursing **24**: 152-161.
Kononen, E. (2000). "Development of oral bacterial flora in young children." Annals of Medicine **32**(2): 107-112.
Kononen, E. (2005). "Anaerobes in the upper respiratory tract in infancy." Anaerobe **11**: 131-136.
Lacroix, J. and J. Cotting (2005). "Severity of illness and organ dysfunction scoring in children." Pediatric Critical Care Medicine **6**(3): S126-S134.
Leteurtre, S., A. Duhamel, et al. (2006). "Paediatric logistic organ dysfunction (PELOD) score." The Lancet **367**(9514): 897.
Leteurtre, S., A. Martinot, et al. (1999). "Development of a pediatric multiple organ dysfunction score: use of two strategies." Medical Decision Making **19**(4): 399-410.
McNeill, H. (2000). "Biting back at poor oral hygiene." Intensive and Critical Care Nursing **16**: 367-372.
Munro, C., M. Grap, et al. (2006). "Oral health status and development of ventilator-associated pneumonia: a descriptive study." American Journal of Critical Care **15**(5): 453-460.
Munro, C., M. Grap, et al. (2009). "Chlorhexidine, toothbrushing, and preventing ventilator-associated pneumonia in critically ill adults." American Journal of Critical Care **18**(5): 428-37.
O'Reilly, M. (2003). "Oral care of the critically ill: a review of literature and guidelines for practice." Australian Critical Care **16**(3): 101-110.
Perkins, S., K. Woeltje, et al. (2010). "Endotracheal tube biofilm inoculation of oral flora and subsequent colonization of opportunistic pathogens." International journal of medical microbiology **300** (7): 503-11.

Pugin, J., R. Auckenthaler, et al. (1991). "Oropharyngeal decontamination decreases incidence of ventilator-associated pneumonia." JAMA **265**(20): 2704-2710.
Rubenstein, J., K. Kabat, et al. (1992). "Bacterial and fungal colonization of endotracheal tubes in children: a prospective study." Critical Care Medicine **20**(11): 1544-1549.
Safdar, N. M. D. M. S., C. M. D. Dezfulian, et al. (2005). "Clinical and economic consequences of ventilator-associated pneumonia: A systematic review." Critical Care Medicine **33**(10): 2184-2193.
Santanae, M., H. Leite, et al. (2009). "Hypophosphatemia in critically ill children: Prevalence and associated risk factors." Pediatric Critical Care Medicine **Jan 30**: Epub ahead of print.
Scannapieco, F., E. Stewart, et al. (1992). "Colonization of dental plaque by respiratory pathogens in medical intensive care patients." Critical Care Medicine **20**(6): 740-745.
Schleder, B. (2003). "Taking charge of ventilator-associated pneumonia." Nursing Management **34**(8): 27-33.
Shann, F., G. Pearson, et al. (1997). "Paediatric index of mortality (PIM): a mortality prediction model for children in intensive care." Intensive Care Medicine **23**: 201-207.
Singhi, A., D. Raman Rao, et al. (2008). "Candida colonization and candidemia in a pediatric intensive care unit." Pediatric Critical Care Medicine **9**(1): 91-95.
Sixou, J., O. Medeiros-Batista, et al. (1996). "Modifications of the microflora of the oral cavity arising during immunosuppressive chemotherapy." Oral Oncology: European journal of cancer **32B**(5): 306-310.
Sixou, J., O. Medeiros-Batista, et al. (1998). "The effect of chemotherapy on the supragingival plaque of pediatric cancer patients." Oral Oncology **34**: 476-483.
Slater, A. and F. Shann (2004). "The suitability of the Pediatric Index of Mortality (PIM), PIM2, the Pediatric Risk of Mortality (PRISM), and PRISM III for monitoring the quality of pediatric intensive care in Australia and New Zealand." Pediatric Critical Care Medicine **5**(5): 447-454.
Slater, A., F. Shann, et al. (2002). "PIM2: a revised version of the Paediatric Index of Mortality." Neonatal and Pediatric Intensive Care **29**: 278-285.
Stonecypher, K. (2010). "Ventilator-associated pneumonia: the importance of oral care in intubated adults."Critical Care Nursing Quarterly **33**(4):339-47.
Suljagic, V., M. Cobeljic, et al. (2005). "Nosocomial bloodstream infections in ICU and non-ICU patients." American Journal of Infection Control (AJIC) **33**(6): 333-340.
Thorburn, K., M. Jardine, et al. (2009). "Antibiotic resistant bacteria and infection in children with cerebral palsy requiring mechanical ventilation." Pediatric Critical Care Medicine **10**(2): 222-226.
Thukral, A., U. Kohli, et al. (2007). "Validation of the PELOD score for multiple organ dysfunction in children." Indian Pediatrics **44**(9): 683-6.
Thukral, A., R. Lodha, et al. (2006). "Performance of Pediatric Risk of Mortality (PRISM), Pediatric Index of Mortality (PIM) and PIM2 in a pediatric intensive care unit in a developing country." Pediatric Critical Care Medicine **7**(4): 356-61.
Toltzis, P., C. Hoyen, et al. (1999). "Factors that predict preexisting colonization with antibiotic-resistant gram negative bacilli in patients admitted to a Pediatric Intensive Care Unit." Pediatrics **103**(4): 719-723.

Turton, P. (2008). "Ventilator-associated pneumonia in paediatric intensive care: a literature review." Nursing in Critical Care **13**(5): 241-248.

Wolfer, A., P. Silvani, et al. (2007). "Pediatric Index of Mortality 2 score in Italy: a multicenter, prospective, observational study." Intensive Care Medicine **33**(8): 1407-13.

Venkatachalam, V., J. Hendley, et al. (2010). "The diagnostic dilemma of ventilator-associated pneumonia in critically ill children". Pediatric Critical Care Medicine **Epub ahead of print**

Yung, M., B. Wilkins, et al. (2008). "Glucose control, organ failure and mortality in pediatric intensive care." Pediatric Critical Care Medicine **9**(2): 147-52.

TABLES AND FIGURES:-

TABLE 1
Oral Assessment Scale (OAS)

Lips – feel, observe	1 = Smooth, pink, moist 2 = Dry or cracked 3 = Ulceration or bleeding
Tongue – feel, observe	1 = Smooth, pink, moist 2 = Coated / shiny appearance, increased/decreased redness 3 = Thick and large, inflamed, blistered or ulcered
Saliva - observe	1 = Thin, watery and plentiful 2 = Thick or decreased 3 = Ropy or absent
Gingiva/oral mucosa – observe	1 = Smooth, pink, moist 2 = Generally pale, with small amount of reddened areas or ulcers, dry 3 = Bleeding, inflamed, multiple ulcers, very dry and oedematous
Teeth - observe	0 = Non-dentate 1 = Clean, no debris 2 = Plaque/debris in localised area 3 = Plaque/debris generalised, cavities visable

TABLE 2
Demographic characteristics of study participants

Demographics		Value
Age (months) Median (Min-Max)		11.5 (0.1-168)
Length of PICU stay (hours) Median (Min-Max)		107.5 (21-977)
Dentate status *n* (%)	Non-dentate	22 (47.8)
	Dentate	24 (52.2)
Participants receiving mechanical ventilation *n* (%)		35 (76.0)
Non-survivors of PICU *n* (%)		3 (6.5)
Primary Diagnosis *n* (%)	Trauma	4 (8.7)
	Respiratory failure	13 (28.3)
	Post-operative	8 (17.4)
	Neurology	4 (8.7)
	Haematology/ Oncology	7 (15.2)
	Sepsis	5 (10.9)
	Other	5 (10.9)
	Cardiovascular	0 (0.0)
Admission source *n* (%)	Hospital ward	5 (10.9)
	Hospital department of emergency medicine	9 (19.9)
	Outside retrieval	17 (32.6)
	Operating Theatre	15 (37.0)
PIM2 Median (Min-Max)		1.79 (0.23-31.58)
PELOD (day 2) Median (Min-Max)		1.0 (0.00-61.0) 61.00

TABLE 3
Frequency of oral dysfunction during critical illness

Oral dysfunction (*n*=65)	***n* (%)**
Nil dysfunction (OAS = 5)	14 (37.4)
Moderate dysfunction (OAS 6-10)	32 (62.6)
Severe dysfunction (OAS >10)	0 (0)

TABLE 4
Frequency of pathogenic oropharyngeal flora isolated from swabs

Pathogenic flora (*n*=65)	***n* (%)**
Candida sp.	30 (46.1)
Staphylococcus aureus	11 (16.9)
Haemophilus influenzae	6 (9.2)
Enterococcus sp.	3 (4.6)
Pseudomonas aeruginosa	3 (4.6)
Escherichia coli	2 (3.1)
Acinetobacter sp.	2 (3.1)
Stenotrophomonas maltophilia	2 (3.1)
Klebsiella pneumoniae	2 (3.1)
Enterobacter cloacae	2 (3.1)
Serratia marcescens	2 (3.1)

TABLE 5

Oral hygiene received by participants

	n (%)
Oral cleansing solution used (day two)	
Nil	5 (12.5)
Water	31 (77.5)
Chlorhexidine mouthwash	1 (2.5)
Toothpaste	2 (5.0)
Sodium bicarbonate mouthwash	1 (2.5)
Oral cleansing implement used (day two)	
Nil	5 (12.5)
Foam swab	28 (62.5)
Cotton swab	4 (10.0)
Toothbrush	3 (7.5)
Frequency of oral hygiene (day two)	
Nil	5 (12.5)
Every 4 hours	9 (22.5)
Every 6 hours	16 (40.0)
Every 12 hours	7 (17.5)
Once daily	3 (7.5)
Received oral or systemic antifungal therapy during PICU admission	6 (13.0)
Dentate patients who had their teeth brushed within 48 hours of PICU admission	2 (8.3)
Participants who received oral care using a cotton swab during PICU admission	16 (34.8)

TABLE 6

Oropharyngeal sampling results and PICU-related HAI

Day No.	Oropharyngeal colonisation	Type of HAI	Pathogen in HAI	Day of HAI
0	Commensal flora & *Haemophilus influenzae*	Pneumonia (ETT aspirate)	*Haemophilus influenzae*	2
2	Commensal flora & *Acinetobacter baumannii*			
4	Commensal flora & *Acinetobacter baumannii*			
0	Commensal flora & *Escherichia coli*	Pneumonia (ETT aspirate)	*Pseudomonas aeruginosa*	2
2	Commensal flora & *Candida sp.*			
4	*Candida albicans*			
6	*Pseudomonas aeruginosa* & *Candida albicans*			
8	Commensal flora			
0	Commensal flora & *Haemophilus influenzae*	Pneumonia (ETT aspirate)	*Candida* sp.	2
2-6	Commensal flora & *Candida sp.*			
8	Commensal flora, *Haemophilus influenzae* & *Candida* sp.	Pneumonia (ETT aspirate)	*Staphylococcus aureus* & *Haemophilus influenzae*	8
10	Not done			
12	Commensal flora & *Candida albicans*			
14-18	Commensal flora			
20	*Klebsiella pneumoniae, Enterobacter cloacae* & *Candida albicans*			
22-24	Commensal flora			
26	Klebsiella pneumoniae, Enterobacter cloacae & commensal flora			
28	Not done			
30-32	Commensal flora			

Day No.	Oropharyngeal colonisation	Type of HAI	Pathogen in HAI	Day of HAI
34-36	Nil flora			
38	Commensal flora			
0	Nil	Blood-stream infection (Blood culture)	*Enterococcus faecalis*	4
2-4	Commensal flora & *Staphylococcus aureus*			
0-6	Commensal flora	Bacteraemia (Blood Culture)	*Escherichia coli*	6
0	*Staphylococcus aureus* & commensal flora	Pneumonia (ETT aspirate)	*Candida* sp.	10
2	Not done			
4	*Staphylococcus aureus, Candida* sp. & commensal flora			
6-18	*Candida* sp.			
20	Nil			
0-4	Not Done	Pneumonia (ETT aspirate)	*Candida* sp.	14
6-8	Nil			
10	Commensal flora & *Candida sp.*			
12-14	Nil			
16	*Candida albicans*			
18-22	Nil			
0-4	*Candida albicans*	Blood-stream infection (Blood culture)	*Stenotrophomonas Maltophilia*	18
6-10	Nil			
12-14	*Pseudomonas aeruginosa*			
16	*Stenotrophomonas maltophilia*			
18	Nil			
20	*Stenotrophomonas maltophilia*			
22-24	*Serratia marcescens* & commensal flora			