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Prospective Study of Swallowing Disorders, Chest Infection, Fluid

Balance and Outcome in Unselected Patients with Acute Stroke

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Dedicated to my wife, Margaret and my children, Daniel, Leah, Luke and Micah

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ABSTRACT

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Published data suggests that the bedside assessment of swallowing has limited usefulness. There is a paucity of information on the natural history of dysphagia following stroke and the relationship of dysphagia to outcome.

This thesis examines the above areas.

One hundred and twenty one consecutive acute stroke patients have been prospectively studied. Their swallowing was assessed, using standardised proformas, by both a doctor and a speech and language therapist, nutritional status was also documented (anthropometric and haematological parameters). Assessments were repeated at 7, 28 and 180 days. Where feasible a videofluoroscopy was conducted within 3 days of admission and at day 28. A CT scan was also performed. Outcome measures were the presence of chest infection, mortality, length of stay, disability and institutionalisation.

On admission, 31% (32/104) of those assessed by the speech and language therapist, and 50% (61/121) by the doctor had dysphagia, 21% (20/94) were aspirating on videofluoroscopy. Detailed assessment by the speech and language therapist gave a sensitivity of 47%, specificity of 86%, positive predictive value of 50% and a negative predictive value of 85% for aspiration. Multiple logistic regression analysis identified a weak voluntary cough, coughing on 5 mls of water and any alteration of conscious level as the best

predictors of aspiration. Patients with dysphagia had a higher incidence of chest infection (p<0.01), poor nutritional state (p=0.038), mortality (p=0.022), disability (p=0.02), length of stay (p<0.001), and institutionalisation (p<0.05). Dysphagia, but not aspiration, was an independent predictor for mortality and chest infection. Dysphagia was still present in 27% (28/110) at day 7 and 11% (8/73) at six months, 3% (2/73) had developed dysphagia. Twelve patients were aspirating on videofluoroscopy at day 28.

In conclusion, dysphagia is common following stroke, may be transient, persist or develop at a later date. The poor reliability of the bedside assessment is confirmed. Dysphagia at the time of stroke is associated with a worse outcome, but the routine use of videofluoroscopy is questioned.

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DEFINITIONS

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Aspiration: The passage of food and or liquid into the airway below the level of the true vocal cords.

BSA: Bedside swallowing assessment.

DOC: Refers to a physician conducting the bedside assessment of swallowing.

Silent aspiration: Aspiration with no outward signs of difficulty.

SLT: refers to a speech and language therapist conducting a bedside swallowing assessment.

Supraglottic/laryngeal penetration: Entry of food, liquid into the larynx, but remaining above the level of the true vocal cords, and not passing into the trachea..

Unsafe swallow (or dysphagia): Refers to an abnormal swallow which may result in aspiration.

Videofluoroscopy: A modified barium swallow, using small volumes of barium to examine the oro-pharyngeal swallow.

PREFACE

Swallowing impairment (dysphagia) following stroke is considered to be associated with a particularly high early mortality. Workers have noted dysphagia to be an independent indicator of prognosis (Gordon et al., 1987; Barer, 1989), with 35-40% of those with any swallowing abnormality dying (Wade and Hewer, 1987), resulting in a four fold increase in mortality in the first month (Barer, 1987).

The importance of this has been recognised only recently, previously most textbooks (Mushet, 1867; Rheberger, 1926; Bickerstaff, 1980; Walton, 1985; Tallis, 1989) and literature (Meadows, 1973) mention it only as a complication of brain stem stroke or bilateral hemisphere lesions.

What is not known is the true incidence of dysphagia or swallowing difficulties following strokes, as some patients will have sub- clinical problems. It has been stated that up to 69% of those with acute stroke have some form of clinical swallowing difficulty (O'Neill,P.A. personal communication), Gordon et al. (1987) reported an incidence of 45% in all strokes and 43% of unilateral cerebral hemisphere strokes admitted to hospital, whilst Barer (1989) has reported an incidence of 28% in patients seen within 48 hours of stroke onset. Unfortunately, he excluded those who could not swallow tablets and did

not use videofluoroscopy to assess clinical accuracy. The problem of determining the incidence and prevalence partly relates to the varying methods of assessment used and their timing, which is usually weeks to months (Veis and Logemann, 1985; Gordon et al. 1987; Horner and Massey, 1988) after stroke.

There have not been any large studies on patients seen and assessed within 24 hours of the onset of symptoms, which is important as many swallowing problems may clinically improve after this time (Gordon et al., 1987; Kidd et al., 1993). There is also a paucity of studies that have attempted to validate a simple bedside swallowing assessment against videofluorosocpy.

The relationship between dysphagia, its complications (aspiration, infection, malnutrition and dehydration) and functional outcome require detailed investigation.

The studies described here were designed to answer the following questions.

1. What is the occurrence of clinical swallowing problems following acute stroke?

2. Does a bedside swallowing assessment of swallowing have the sensitivity and specificity to accurately detect aspiration, as assessed on videofluoroscopy, during the acute phase of stroke?

3. Is there a relationship between the side or site of the cerebral lesion and the presence of aspiration?

4. What is the natural history of swallowing post stroke?

5. Is there a deterioration in hydration and nutritional status of patients with swallowing difficulties when compared to those with a normal swallow?

6. How are dysphagia and aspiration related to outcome?

Is there an increase in :

a] chest infection

b] mortality

c] length of hospital stay and functional impairment

in those patients with swallowing difficulties?

CHAPTER 1

INTRODUCTION

DEFINITION

Stroke has been given many definitions over the years. In 1867 apoplexy or cerebral haemorrhage was described as a loss of consciousness, feeling and voluntary action - a suspension of brain function (Mushet, 1867). More recently, stroke has been defined as "a rapidly developing episode of focal or global neurological dysfunction lasting >24 hours or leading to death and being presumed to be due to a disturbance in the vascular supply to the brain, either cerebral infarction or intracranial haemorrhage" (WHO, 1978).

EPIDEMIOLOGY

Stroke is common, afflicting one person every five minutes in the United Kingdom (Consensus, 1988), and is the third commonest cause of death in most developed countries (Viitanen, et al., 1987). The incidence varies by the hour (Wroe et al., 1992), by the day (Barer et al., 1984), and by season - cold stress (Bastin, 1898; Keatinge, 1986) may play a part.

There are approximately ≈99 000 first ever strokes per year in England and Wales (Bamford et al., 1988), and half of these occur in those over 75 years

of age (Stevens and Ambler, 1982; Consensus, 1988). The incidence of stroke is 1-2 per thousand population (OCSP, 1983; Bamford et al., 1988), increasing with age (Warlow, 1983) from 0.1-0.6/1000 in those aged <40 years (Warlow, 1983) and rising exponentially (Bonita, 1992) to 13-33/1000 in those >75 years (Warlow, 1983).

There has been a decline in stroke incidence (Ebrahim, 1992): between 1945 and 1971 the reduction was 2% per year in Rochester, USA (Garraway et al., 1979). The largest decrease in incidence was in those aged over 85 years of age (Garraway et al., 1979). Between 1980-4 there was a 17% increase in stroke incidence, in this population, probably related to increased investigation, including CT scanning (Broderick et al., 1989; Ebrahim, 1990). This decrease has not been mirrored in Central and Eastern Europe.

MORTALITY, OUTCOME and PROGNOSIS

Many of the studies looking at the incidence and causes of death following stroke are either based on hospital (O'Brien, 1987) or autopsy series (Brown and Glassenberg, 1973; Silver et al., 1984; Viitanen et al., 1987), with only a few studies looking at all (hospital and community) cases (Stevens and Ambler, 1982; OCSP, 1983; Garraway, 1983). Consequently data on

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outcome, and on incidence and prevalence of complications, need to be treated with caution as they may not be applicable to the whole population. Only 60% (Bamford et al., 1988) to 80% (Bonita et al., 1987) of stroke patients are admitted to hospital, the others are managed at home. The elderly are admitted for nursing care and rehabilitation and the young for investigation. Other groups more likely to be admitted are males of all ages, and those with a severe motor deficit (Bonita et al., 1987).

Mortality

There is a high mortality rate following stroke within the first year (a thirteen fold increase), the majority of the deaths occurring within the first month (Ebrahim, 1990), Deaths then continue at a consistent age-dependent but increased rate compared with the general population (Viitanen et al., 1987; Ebrahim, 1990). Half the deaths following stroke will be due to heart disease, only one third being due to stroke itself (RCP, 1989).

The mortality from stroke is decreasing. Since 1900 in the USA and since 1964 in UK there has been an increase in the one month and seven year survival (Garraway et al., 1979). There was a 20%-28% fall in stroke mortality between 1970/2 and 1986 (Shaper, 1991; Balarajan, 1991). This

decline in the UK has been noted, in a retrospective study, to occur at different rates in different ethnic groups (Balarajan, 1991). The decrease in mortality may be due to reduced case fatality (particularly so in the case of early deaths) rather than reduced incidence (Ebrahim, 1990).

Early death after cerebral infarct, in unselected (community) populations is low - 10% after 30 days (Sandercock et al., 1992; Sandercock and Willems, 1992), which has been reduced from 25% in 1945 (Garraway et al., 1979).

There has been a similar decline in mortality following primary intracerebral haemorrhage. Scheutz et al. (1992) in a retrospective study (diagnosis was made prospectively, details collected retrospectively) have noticed a decrease in early mortality following cerebral haemorrhage which appears to be related to a reduction in the number of comatosed patients (which may be related to the treatment of hypertension); and also that there was a lower case fatality rate which may be due to scanning detecting small bleeds previously diagnosed as ischaemic, or may be due to demographic changes. These changes may be due to changes in the age of the population or the ethnic mix of the population. This may result in a population either at a different risk of intracerebral bleed, or where the severity of the haemorrhage might be less.

Both of these would lead to an apparent change in occurrence and mortality following intracerebral haemorrhage.

Mortality at thirty days is related to haematoma volume (Fogelholm et al., 1992), and has decreased from 49% to 31% between 1978 and 1989. A similar decrease has been noted in those on anticoagulants at the time of the haemorrhage (71-35%) (Garraway et al., 1989; Scheutz et al., 1992); but the quality of life in the survivors being poor (Scheutz et al., 1992).

The clinical signs present at the time of presentation, following acute stroke, are markers of prognosis and eventual outcome. These can be divided into early and late prognostic signs.

Markers of Early Mortality

There are clinical and laboratory (biochemical and haematological), which are predictive of eventual outcome.

There are many markers, which if present on admission, are predictive of early mortality. Those that carry the greatest relative risk are the presence of an impaired conscious level, haemorrhagic stroke, extensor plantars (Ebrahim,

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1990), abnormal respiratory pattern (Rout et al., 1971), pyrexia (Hindfelt, 1976) and age over 70 years (Wade et al., 1985; Gray et al., 1988; Fogelholm et al., 1992; Granger et al., 1992).

Markers of late Mortality

Persistent incontinence (Wade and Hewer, 1985; Barer and Mitchell, 1989; Fullerton et al., 1988), poor functional recovery (Wade et al., 1985), age over 70 years (Wade et al., 1985) are all markers of late mortality. The presence of an impaired conscious level may not only be predictive of short term mortality, but have an effect on mortality at one year (Christie, 1981).

Biochemical and haematological parameters

Haematological signs

A raised haematocrit and an elevated white cell count (Gray et al., 1988) have been reported to be poor prognostic indicators.

Biochemical signs

A raised blood glucose (Power et al., 1988; O'Neill et al., 1991; Oppenheimer and Hachinski, 1992), especially if there is no history of diabetes (Candelise et al., 1985), and a rise in catecholamine levels (Feibel

et al., 1977) which may be secondary to the rise in stress hormones, are markers of poor prognosis (O'Neill et al., 1991). The association between hyperglycaemia present on admission and outcome has been questioned by Matcher et al. (1992), but with a 'young' study group.

Cause of Death

During the first week many deaths are due to the stroke itself. In the second week deaths are more often due to infection, pulmonary embolism or cardiac disease (Brown and Glassenberg, 1983). Bronchopneumonia is a common cause (Mulley, 1982), the aetiology of which may be aspiration (Brown and Glassenberg, 1983), or immobility. The association between dysphagia, aspiration and pneumonia is discussed in greater detail later.

Pulmonary embolism may be secondary to deep vein thrombosis which usually occurs in the paralysed leg of which 50% are asymptomatic (Mulley, 1982; Oppenheimer and Hachinski, 1992). Fatal events often occur in ambulant patients (Viitanen et al., 1987), the risk continuing into the rehabilitation period particularly in those less mobile (Lowe et al., 1992; Oczkowski et al., 1992).

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Prognosis

Prognosis, following stroke, is dependent on stroke pathology and site (Bamford et al., 1991). For every person who dies following a stroke, there are almost four who survive with varying degrees of disability (Consensus, 1988). In 24% of all severely handicapped people in the community, stroke is the main cause of the handicap (Harris et al., 1971).

A typical health district will provide care for $\approx$ 1500 survivors of stroke at any one time (Consensus, 1988). In a population of one million there will be 1250 first ever strokes per year and 350 recurrent (Bonita et al., 1984); 880 will survive 6 months, 640 will be in private residence, the rest being heavily dependent and in long term care (Bonita, 1992), 15% of all patients, suffering an acute event, in a one year period will have residual difficulty caring for themselves (Bonita, 1992).

Around 20% of those who survive the initial three week period never improve enough to go home and many die with intercurrent infection, heart disease or recurrent stroke (RCP, 1989). The others have a reduced life expectancy; the male 3 year life expectancy is only around 54% as against 88% in age matched controls (RCP, 1989).

Outcome

From the time of admission, the nutritional status of the patient should be monitored, decubitus ulcers, and contractures should be looked for and prevented. The nutritional state of a patient may relate to the presence or absence of dysphagia. A poor nutritional status may be associated with a worse outcome, but this may be difficult to separate out from the direct effects of the stroke. A more in depth discussion is presented in a later chapter.

The severity of the stroke, premorbid mobility (Ebrahim, 1990), the presence of perceptual difficulties and somatic problems (Wade et al., 1988) all affect the rate of recovery and increase length of hospital stay. Does the presence of dysphagia have an independent relationship to the length of hospital stay? This question remains to be answered.

Dysphagia

Given the association between dysphagia, resulting aspiration and the occurrence of complications such as pneumonia, malnutrition and mortality, there is a need to detect the presence or absence of aspiration at the bed side as it is not a feasible proposition to subject all patients to videofluoroscopy. Many of the studies performed to investigate dysphagia and the presence of

aspiration have been performed many months or even years after the stroke (Linden and Siebens, 1983; Veis and Logemann, 1985). Consequently the correlations between the clinical findings and the presence of aspiration during the acute phase of stroke may not apply. Only one study has examined dysphagia acutely both clinically and using videofluoroscopy. They found that the absence of pharyngeal sensation was associated with aspiration (Kidd et al., 1993). At present there have not been any other studies undertaken to validate a simple bedside assessment of swallowing, against videofluoroscopy, during the acute phase of swallowing. This area needs further critical evaluation, and this work attempts to do this.

CHAPTER 2

MECHANISM OF SWALLOWING

MECHANISM OF SWALLOWING

The act of swallowing is extremely complex involving six cranial nerves and 55 muscles of the face, mouth, pharynx and oesophagus (Elliot, 1988; Groher, 1984; Donner, 1986), the entire act being the most complex "all or none" reflex obtainable by peripheral nerve stimulation (Doty, 1951). The pharynx is pivotal in the act of swallowing, speech and respiration (Clarke, 1920; Linden and Siebens, 1983; Selly et al., 1989a).

There are three phases to swallowing (Ekberg and Nylander, 1982), which are synchronous, co-ordinated and symmetrical, though the pattern may be unique and specific for each individual (Ekberg and Nylander, 1982). As swallowing is a continuous process each phase overlaps with the one before it.

Before the mechanism and the neurological control of swallowing is elucidated, we will follow the bolus from the mouth to the oesophagus via the pharynx.

BOLUS PASSAGE

Food is initially introduced to the oral cavity, mechanically broken down by the teeth, and then formed into separate boluses by the tongue tip (Ramsey et al., 1955). The tongue then pushes the bolus backwards past the faucial

arches, at the same time as the soft palate elevates. Leaving the back of the tongue, the bolus momentarily enters the paired open mouths of the valleculae (between the tongue and the epiglottis) (Ardran and Kemp, 1952; Curtis, 1982). The bolus will then either pass over or round the epiglottis, depending on its size and consistency, being directed to the hypopharynx (Jones and Donner, 1988), and the inverted "pyramids" of the pyriform fossae (Ardran and Kemp, 1961), the orifices of which are below the level of the laryngeal entrance (Curtis, 1982). These two channels then unite at the top of the oesophagus and direct the bolus down into the oesophagus. The anatomical relationships of the mouth, pharynx and oesophagus are shown in figure 1.

The bolus is given an initial push by the tongue (McConnel et al., 1988). Subsequently, whilst the vocal cords and laryngeal vestibule remain closed (Shaker et al., 1990), peristaltic waves (stripping wave (Logemann, 1988)) beginning at the palatopharyngeus muscle sweep down the entire pharynx taking <1 second (Hellemans et al., 1981), moving the bolus from the hypo-pharynx to the oesophagus (Elliot, 1988). These continue down the oesophagus at 3-4 cm /sec (Hellemans et al., 1981). This general "pattern" is modified depending on the nature (consistency and volume) of the bolus (Dantas et al., 1990).

This whole process is dynamic; the anatomical relationships are varied and

Figure 1

The important anatomical relationships for swallowing.

Bolus

Pharynx

Epiglotis

Larynx

Larynx

Oesophagus

Cricopharyngeus
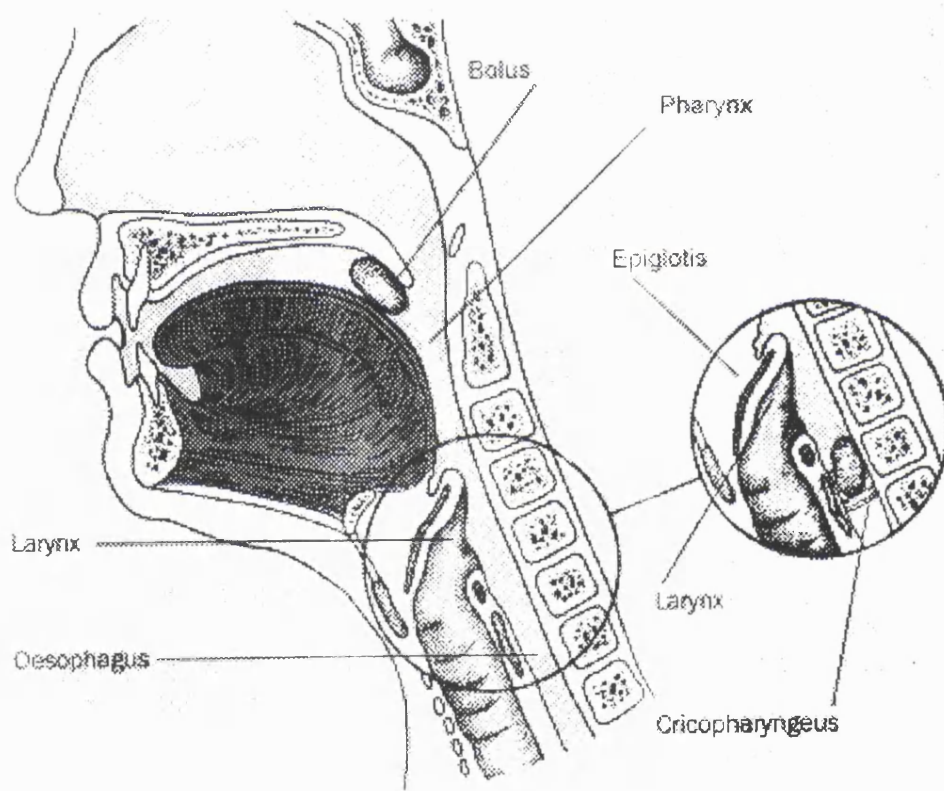
altered by the subconscious movement of the head and neck, to adapt to the bolus characteristics (Ekberg and Nylander, 1982; Bucholz et al., 1985).

ORAL STAGE

During this stage food, placed in the oral cavity is broken down by the mechanical action of the teeth and tongue movement. Once this is complete, a bolus is formed by rolling lingular movements (Hellemans et al., 1981; Elliot, 1988), pressing against the hard palate (Miller, 1986). The bolus is trapped between the tongue and soft palate (Logemann, 1983; Miller, 1986), that to be swallowed separated from the rest by the tongue tip (Ramsey et al., 1955) and is propelled backwards towards the pharynx (Hellemans et al., 1981; Elliot, 1988). As the bolus passes the sensitive bases of the anterior faucial arches the swallow reflex is triggered (Logemann, 1983).

As food or liquid is put into the mouth, prior to the initiation of deglutition, partial vocal cord closure followed by arytenoid approximation to the base of the epiglottis occurs (Shaker et al., 1990). The airway remains open during the oral stage, (Ardran and Kemp, 1961), but respiration ceases as the swallow commences (Selley et al., 1989a).

Later, prior to the initiation of the pharyngeal phase, vocal cord adduction is

completed, followed by the leading swallowing complex of the tongue base and superior hyoid movement (with submental myoelectrical activity) occurs (Shaker et al., 1990).

PHARYNGEAL STAGE

Three simultaneous processes occur during this stage: cessation of respiration (Ardran and Kemp, 1952; Elliot, 1988), airway protection and transport the bolus to the oesophagus (Elliot, 1988). For swallowing to occur safely, all three openings (mouth, nose and larynx) into the pharynx must be closed (Barclay, 1930). Also there needs to be [1] complete bolus transport and [2] precise coordination (Miller, 1986).

The mechanical protection of the airway is ensured by the following mechanisms:

A) Nasopharynx

Contraction of the superior pharyngeal constrictor (Elliot, 1988) and approximation of the pharyngeal pillars (Hellemans et al., 1981) aid the soft palate in closing off the nasopharynx, hence preventing nasal regurgitation. This should be completed before the head of the bolus reaches the valleculae

(Ardran and Kemp, 1961). At this time muscles of the posterior tongue are active as part of the ventral wall of the pharynx (Miller, 1986) closing off the oral cavity.

B) Tracheobronchial Tree

Protection of the tracheobronchial tree commences at the start of the pharyngeal stage, and has five primary protective mechanisms (Butcher, 1982), which rapidly follow each other:

1) Sphincteric closure of the laryngeal aditus

The larynx is closed from the bottom up (Butcher, 1982) by muscular contraction, involving seven of the eight intrinsic muscles (Curtis and Hudson, 1983), and adduction of the true vocal cords just before the swallowing process begins (Miller, 1986). This early closure of the larynx is considered by Shaker et al. (1989) to be a preprogramed event that is essential for protection of the airway (Logemann, 1983). The true vocal cords appear to remain apposed during the entire swallowing act, protecting the airway in case of mild incompetence or incoordination of laryngeal closure (Shaker et al., 1990).

2) Axial elevation of the larynx

The larynx is rotated upwards and forwards (Ardran and Kemp, 1952; Butcher, 1982), by the hypomandibular muscle complex acting in conjunction with the hyoid bone, by two vertebral body heights (Curtis, 1982), to rest against the base of the tongue (Ardran and Kemp, 1952). At the same time as the pharynx is elevated (Borgström and Ekberg, 1988) the cricopharyngeus muscle begins to relax (Logemann, 1983).

As the larynx and pharynx rise, the epiglottis is tipped down (Hellemans et al., 1981) in two stages due to upward laryngeal movement (Dodds et al., 1990) and bolus weight (Hellemans et al., 1981). Closure, which may not be 100% (Ardran and Kemp, 1961; Hellemans et al., 1981), is not complete until early in the pharyngeal stage of the swallow (Feinberg et al., 1990), the final downward tilt being due to contraction of thyroepiglottic and aryepiglottic muscles (Ardran and Kemp, 1961; Borgström and Ekberg, 1988).

3&4) Relaxation of the superior pharyngeal constrictor and opening of the cricopharyngeus

These final two stages occur as the swallow nears completion. As the larynx moves forwards opening the oropharynx (McConnel et al., 1988; Logemann

and Kahrilas, 1990), and the bolus enters the pharynx, touching momentarily the posterior pharyngeal wall (Donner, 1974), the cricopharyngeus muscle relaxes. The opening of the cricopharyngeus is aided by the pharyngeal stripping wave and bolus weight. Maximum cricopharyngeal opening occurs at the time of maximum elevation and anterior displacement of the larynx (Logemann and Kahrilas, 1990), resulting in the commencement of the oesophageal stage (Hellemans et al., 1981). Both laryngeal elevation and cricopharyngeal relaxation are essential for normal opening of the pharyngo-oesophageal segment to allow bolus passage (McConnel et al., 1988).

5) Cessation of Respiration

When the pharyngeal stage commences, respiration stops, irrespective of the stage of the cycle. On the completion of the swallow, exhalation occurs. This is vital to protect the airway during swallowing. Inspiration following a swallow, may result in inhalation of any material still in the pharynx (Selley et al., 1989b).

OESOPHAGEAL STAGE

The oesophageal phase commences soon after commencement of the pharyngeal stage (Miller, 1986). Peristalsis commenced in the pharynx continues

across the cricopharyngeus and down the oesophagus at a slower rate, of 3-4 per second (Hellemanms et al., 1981).

For unimpeded bolus passage the oesophago-pharyngeal junction must relax and completely open. Any failure of this or prominence of the cricopharyngeus may be due to pharyngeal paresis (McConnel et al., 1988). Once the bolus has passed through the cricopharyngeus the resting state is presumed and the act of swallowing ceases (Fisher et al., 1978).

NEURAL CONTROL OF SWALLOWING

The act of swallowing, although predominantly a reflex and hence automatic, is modulated by the characteristics of the bolus passing through the various stages.

Bolus volume and viscosity affect different phases of swallowing in different ways. Dantas et al. (1990) suggest that bolus volume modulates the central swallowing program by inducing earlier receptive pharyngeal expansion and upper oesophageal sphincter opening to accommodate larger boluses. As bolus size increases there is an increase in hyoid and laryngeal movement, and bolus velocity (Fisher et al., 1978), though the amplitude of peristaltic waves in the

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pharynx remains constant (Dantas et al., 1990). An increase in viscosity acts mainly to slow bolus transit giving later sphincter opening.

The size of the bolus does not alter the sequence of events during swallowing but will result in alterations to the timing of each part of the swallow (Curtis, 1982; Logemann, 1988). For instance, as the bolus size increases (1-20 cm^3) pharyngeal transit time increases by 0.1 seconds, as do laryngeal closure and elevation (Logemann, 1988), cricopharyngeal opening increases by 0.3 seconds and takes up a larger part of the pharyngeal swallow (33.3% for 1 cm^3 and 75% for 20 cm^3) (Logemann, 1988). The larger the bolus the higher and more anteriorly the larynx elevates (Logemann, 1988).

To enable these changes to occur during swallowing, there is a complicated neurological feedback system which will now be elaborated (see figure 2).

NEUROLOGY

Activation of deglutition is either voluntary (Cerebral) or involuntary (Peripheral receptors in the mouth and pharynx) (Hellemans, 1981).

Swallowing is a sequential, semiautomatic contraction and relaxation discharge

of the muscle groups of the oropharyngeal, laryngeal and oesophageal regions

(Miller, 1986), requiring both cortical and cranial nerve input (Elliot, 1988).

The neural control of swallowing involves four major components:

1) Cerebral control.

2) Paired swallowing centres in the medullary reticular formation (Miller,

1972a; Hellemans et al., 1981; Robbins and Levine, 1988; Dodds et al.,

1990) midbrain.

3) Afferent sensory fibres - cranial nerves.

4) Efferent motor fibres - cranial nerves and ansa cervicalis.

To describe the control of swallowing two major hypotheses are proposed:

1) Reflex chain: This theory suggests the system is dependent on continuous

feedback (Miller, 1986): a bolus stimulates the sensory receptors, with

sequential triggering of the next step.

2) Central pattern generator (One in each half of the brainstem (Miller,

1986)): Regardless of the bolus characteristics, the same preprogrammed

stereotyped swallow will occur.

Figure 2

A schematic diagram depicting the neurological control and feedback

mechanisms of swallowing.

NEUROLOGICAL CONTROL OF SWALLOWING

CORTEX

AMYGDALA

CEREBELLUM

PONS

DORSAL

MEDULLA

VENTRAL

RESPIRATORY CENTRE

V VII IX X XI

XII

X

ORAL CAVITY

PHARYNX

OESOPHAGUS
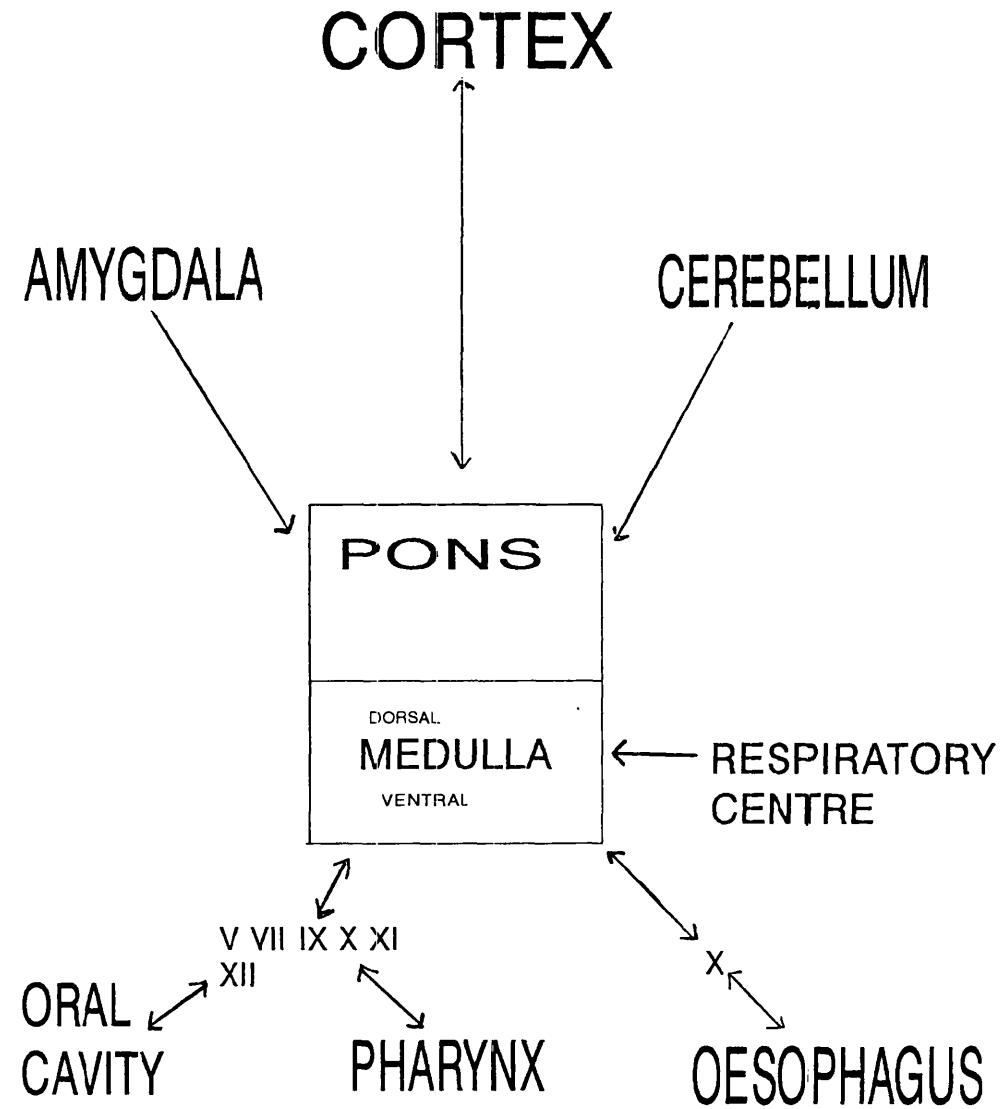
Dodds et al. (1990) consider that it is a combination of 1 and 2: a basic medullary program altered by bolus volume and consistency. Peripheral feedback is important in positioning the bolus on the teeth, preventing tongue injury during chewing (Logemann, 1983), repeated swallowing (Hellemans et al., 1981) to modify the pharyngeal response (Robbins and Levine, 1988).

A more recent suggestion is that the control of swallowing does not reside in discrete areas but is diffuse throughout the brainstem and cortex (Alberts et al., 1992). This may explain the diversity of lesions causing swallowing problems following stroke.

Cerebral control

The voluntary swallow and its integration with other responses requires cortical and subcortical areas. The cortex, though not required for swallowing, is essential for learning motor responses to different bolus characteristics (Miller, 1986). Some parts of the swallow continue regardless, other parts have feedback loops (Morrell, 1984).

The cortical representation of swallowing is probably situated anterolateral to the orbital gyrus of the frontal lobe (Meadows, 1973) receiving information

from the contralateral frontal lobe, oropharyngeal and laryngeal receptors (Hellemans et al., 1981; Miller, 1982; Robbins and Levine, 1988).

Stimulation of the premotor cortex is critical for the sequencing the interaction of the tongue, facial movements and mastication with swallowing (Woolsey et al., 1952; Miller, 1986). As it has been suggested (Robbins and Levine, 1988), but not confirmed (Chen et al., 1990), that different hemispheres control separate phases of deglutition, the right hemisphere- the pharyngeal stage and the left -the oral stage, connections between both hemispheres would essential.

The cortex can only exert effect through the brainstem. The amygdalohypothalamic region with the midbrain tegmental field is thought to integrate and control deglutition with visceral and somatic responses; modifying the reflexively evoked swallow, and facilitating visceral response (Miller, 1986).

Brainstem

Both the pons and medulla are involved with the control of swallowing.

Pontine Level

There are two regions in the pons:

1) Reticular formation dorsal to the motor nucleus of the trigeminal nerve, receiving input from peripheral receptive fields relaying on to the ventroposteromedial nucleus in the thalamus (Car et al.,1975).

2) Ventral to Motor nucleus of the trigeminal nerve which is part of the descending pathway, stimulation of which causes swallowing and mastication.

Medullary Level

Paired swallowing centres

These are not discrete areas, but a collection of interneurons within in the reticular formation (Miller, 1972a; Hellemans et al., 1981; Dodds et al., 1990) between the posterior pole of the facial nucleus and the rostral pole of the inferior olive (Miller, 1972a; Butcher, 1982).

Afferent sensory fibres

a) Cortical

The cortex modifies the swallowing reflex both by inhibition and facilitation

(Bucholz et al., 1985; Miller, 1986; Dodds et al., 1990). In particular the amygdolothalamic region facilitates visceral function, this action may be modified by dopamine agonists (Miller, 1986).

b) Cranial Nerves

The orderly progression of swallowing is dependant on peripheral feedback (Miller, 1972b; Logemann, 1983). Sensory input from the oral cavity and pharynx, via the fifth, seventh, nineth, tenth and twelfth cranial nerves, first and second cervical nerve roots and vagal input from the oesophagus (Miller, 1986), provide information regarding bolus characteristics and the stage of the swallow. Sensory receptors are found in the fauces, tonsils, soft palate, tongue base and posterior pharyngeal wall (Donner, 1974). Those in the oropharyngeal region and larynx are dominant (Miller, 1986). Absence of stimulation or anaesthesia of these receptors in the oral, pharyngeal and laryngeal mucosa significantly impairs the swallow reflex (Hightower et al., 1957; Mansson and Sandberg, 1975; Hellemans et al., 1981; Miller, 1986). Water in the hypopharynx and laryngeal aditus, and light touch at the bases of the anterior faucial arches, are the most effective stimuli for triggering swallowing. Repeated stimulation and continuous feedback enhances the swallow (Miller, 1986). The cessation of speech and respiration are

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coordinated with the swallow via their own control centres (Miller, 1972a; Butcher, 1982), relaying information to the swallowing centres.

c) Efferent motor fibres - cranial nerves and ansa cervicalis.

The efferent outflow is via the cranial nerves supplying the pharynx and larynx, and the ansa cervicalis (C1-C2), which innervate the musculature control tongue, laryngeal and hyoid movement (Dodds et al., 1990).

SUMMARY

The normal swallow has three phases: oral, pharyngeal and oesophageal. These are coordinated, synchronous and continuous throughout the three phases. The control of swallowing involves six cranial nerves and 51 muscles of the face, oral cavity, pharynx and oesophagus. The swallow is considered to be a reflex action, controlled via a pair of medullary 'swallowing' centres. Peripheral feedback and descending afferents from the cortex modify the swallowing mechanism. The only phase of the swallow that is under voluntary control is the early part of the oral phase (bolus preparation).

CHAPTER 3

AGEING AND SWALLOWING

EFFECTS OF AGEING ON SWALLOWING

As stroke is primarily a disease of the old, with approximately 50% occurring in those over 75 years of age, it is worth considering how the swallow changes with age. Knowing the changes that are expected with increasing age enables the investigator to determine the normal variants of the swallow from the abnormal, ie those caused by disease.

How does ageing affect the swallowing mechanism? What changes occur with age? That is, what changes are due primarily to ageing and not secondary to disease? The "normal swallow" is a symmetrical and synchronized act. This occurs in ≈80% of young individuals (mean age 52 years) (Ekberg and Nylander, 1982a). Ekberg and Feinberg (1991) in a study of elderly people (72-93 years) without a history of dysphagia found that only 16% showed this behaviour, but only 31 of the 56 subjects were free of neurological disease. In those, where there was no evidence of neurological disease, 3% had sensorimotor dysfunction and 26% had dissociation between the oral and pharyngeal stages, but aspiration did not occur. It is suggested, therefore, that there is a greater variability in the swallowing process as one gets older due to changes in nerve conduction velocity, tongue muscle strength and reduced compliance in the pharyngoesophageal segment of the pharynx (Tracy et al., 1989). With these changes and the fact that the elderly people take longer to process complex information regarding the bolus (Ekberg and Feinberg, 1991)

they swallow more slowly (Robbins et al., 1993) and have less room for error.

Though "abnormalities" of the swallowing process may occur,

Blonsky et al. (1975) found studying "normal" elderly people and those with

Parkinson's Disease, that ageing itself was not associated with swallowing

difficulties. This has been supported by Logemann (1983) and Elliot (1988).

PREVALENCE OF SWALLOWING DIFFICULTIES

The prevalence of dysphagia or swallowing problems will depend on the

population under investigation. The greater the level of dependence the greater

the prevalence of dysphagia.

Bloem et al. (1990) using a community based questionnaire, in the

Netherlands, found a prevalence of dysphagia of 16% in those over 87 years

of age. A Swedish study, again community based, found an increasing

prevalence of swallowing problems with increasing age (Tibbling and

Gustafsson, 1991).

WHY DO PROBLEMS WITH SWALLOWING OCCUR?

These may be related to primary sensorimotor abnormalities, altered cognition,

mood disorders, or alertness of the elderly person.

Changes in the constitution of connective tissue and cells, do occur with age,

e.g. progressive atrophy of musculature, and prolongation of neural conduction time (Ekberg and Wahlgren, 1985), but actual muscle innervation is less affected by age (Jones and Donner, 1991). These involutional and degenerative changes result in a compensated swallow (Feinberg et al., 1990), but despite these changes co-ordination of the swallow remains the same (Light and Spirduso, 1990).

The mouth, pharynx and oesophagus act as functional unit. Loss of bolus control (resulting in either the early presentation or an inappropriately large bolus being presented to the pharynx), improper timing or sequencing, inadequate propulsion or peristalsis, faulty valving, shortened response and delayed pharyngeal initiation, cricopharyngeal dysfunction (shorter response and opening times), and luminal compromise (internal obstruction or external compression) can all lead to bolus misdirection, and consequently risk of aspiration (Feinberg et al., 1990; Jones and Donner, 1991). Many of these problems are the result of primary sensorimotor abnormalities occurring in the presence of an otherwise normal oropharynx (Feinberg et al., 1990).

Feinberg et al. (1990) also suggest that social isolation and reduced oral intake may lead to disuse de-conditioning and an increased risk of aspiration, though they offer no evidence to support this. Subsequent malnutrition including protein calorie malnutrition may lead to or worsen any dysphagia that might

be present (Veldee and Peth, 1992).

Considering the general changes above, it is worth considering the various aspects of change according to the swallowing phase.

ORAL PHASE

Mechanical Changes

Desiccation of oral soft tissues with consequent reduction in elasticity, impaired repair of tissue damage, and altered cell membrane chemistry occur with ageing (Feldman et al., 1980). This reduction in the elastic tissue of the face and alteration in skin and muscle composition may lead to alterations in lip posture, reduced oral continence and oral spillage with advancing years (Jones and Donner, 1991).

Ageing of the hard tissues leads to attrition of the occlusal surface of the teeth, increasing the surface area and probably decreasing their pulverisation ability (Feldman, et al., 1980). This alteration in dentition (poor dentition or dentures, being edentulous, or altered tooth contact (Cleall, 1965)), as well as reduced saliva production (Sonies et al., 1989), may lead to a prolonged oral stage in elderly people. Feldman et al. (1980) found, using pieces of carrot,

that the absence of teeth significantly decreased the swallowing threshold performance and increased the particle size which the subject was willing to swallow. Also if dentition was complete, the formation of a bolus from ingested food in older people required an increased number of chewing strokes.

The bolus formed also tends to be smaller (Sheth and Diner, 1988) and require more tongue movements (Sonies et al., 1988) due to atrophy of the tongue (muscle being replaced by fat and connective tissue) (Logemann, 1990). Added to this, Tracy et al (1989) have noted that older subjects hold the bolus more posteriorly in the oral cavity and accomplish transit of the bolus head more rapidly, presenting it to the pharynx early. This increases the risk of aspiration if the pharyngeal phase is in anyway compromised (e.g. stroke). The characteristics (consistency and volume) of the bolus affect the timing of the swallow and consequently any difficulties experienced by elderly people may be volume or consistency specific (see section on neurological control of swallowing)

In elderly people with poor dentition, poor oral hygiene leading to gingival inflammation or dentures, anaerobic organisms may colonise the oropharyngeal secretions with a risk of aspiration pneumonia, especially if the swallowing mechanism is in any way compromised. This is important, as inhalation of

oropharyngeal secretions is felt to be the major cause of pneumonia (Penza, 1989).

Neurological Changes

Mastication and bolus formation are reliant on continuous sensory control, consequently any increase in conduction time may compromise this stage of swallowing (Feldman et al., 1980).

This poor masticatory performance is compounded by a decrease in chemosensory perception with age such that taste and smell are diminished (Ekberg and Feinberg, 1991). Due to degeneration of the neuroepithelium with increasing years the ability to detect odours at low concentrations is diminished by a factor of 16 (Koopman, 1991). As taste is predominantly 'smell', these changes may make food less appetizing and as a consequence lead to poor nutrition.

PHARYNGEAL STAGE

Structure

With age there may be changes in the pharyngeal structures. The occurrence of pharyngeal diverticula increases with age. Reduction in pharyngeal sensation and the subsequent dyscoordination may lead to the formation of diverticula

(Hightower, 1957), which may be aided by thinning tissues, but in themselves,

are not the cause of dysphagia (Negus, 1950). Borgström and Ekberg (1988)

state that, in the elderly, diverticula of the Killian - Jamison type may be

more common than Zenker type diverticula, only causing a problem if there

is a degree of pharyngeal dysfunction. Aspiration secondary to diverticula is

due either to obstruction or reflux of contents back into the pharynx after the

completion of the swallow (Feinberg et al., 1990).

Function - Neurological

The bolus may enter the pharynx early due to poor oral / lingual control, but

the swallow usually triggers in time to prevent airway penetration (Jones and

Donner, 1991). Shaw et al. (1990) have stated that pharyngeal peristaltic trans-

it and its coordination with the cricopharyngeus is stereotyped and unmodified

by age or bolus volume. Borgström and Ekberg (1988) have commented that,

ageing per se influences striated muscle (atrophy or hypertrophy) and nerve

conduction velocity and consequently coordination of muscle activity is

impaired, even if individual muscles perform properly. Some fibres

hypertrophy in response to the atrophy of others, but the resultant contraction

force these muscles is weak affecting the quality of pharyngeal function. Tracy

et al. (1989) found that peristaltic velocity and amplitude with in the pharynx

diminish with age, and that there is a delay in the initiation of the pharyngeal

swallow and consequent pooling in the valleculae (between the epiglottis and

the tongue) prior to the onset of laryngeal elevation. Although vallecular

retention has been seen to occur in elderly people (Blonsky et al., 1975) in the

absence of any other pathology, is felt to be of no consequence (Donner,

1974). In fact barium may enter the airway in the first one or two swallows,

but is rapidly cleared (Dodds et al., 1990). Feinberg and Ekberg (1991)

noticed that some people (in a heterogeneous population, the majority with a

neurological deficit), have a delay of up to 1.5 seconds between the

presentation of a bolus to the pharynx and the swallow being triggered; they

have attributed this delay to ageing! This delay in the initiation of the swallow

reflex, together with neurological impairment of pharynx (including reduced

sensation (Mansson and Sandberg, 1974)) will interfere with coordinated

movement of the pharynx and larynx leading to erratic deglutition (Borgström

and Ekberg, 1988).

Function - Mechanical

Laxity of connective tissue surrounding the larynx and trachea may lead to

downward and forward laryngeal shift, and anterior buckling of the upper

oesophageal sphincter, with bolus retention in the valleculae and pyriform

sinuses (Jones and Donner, 1991). Over the age of 60 years maximal

laryngeal elevation may be longer, and return to resting position more slowly (Logemann, 1990). Decreased muscular tonicity causes delayed clearing of the pharynx, especially the lateral food channels (Zanio et al., 1970).

There may be a delay in the onset of cricopharyngeal relaxation and opening when referred to the onset of laryngeal motion (Curtis et al., 1984; Shaw et al., 1990). Tracy et al. (1989) have found that the oldest subjects exhibit shorter cricopharyngeal opening times compared to younger age groups, which they considered could be due to poor oral control leading to the bolus entering the pharynx earlier, but Shaw et al. (1990) found cricopharyngeal closure to be similar in all age groups, and concluded that the differences were due to a prolonged oral phase which would agree with other authors (Cleall, 1965; Feldman et al., 1980).

With increasing age there may be reduced secretions bathing the vocal cords (Sonies et al., 1989), with thinning and bowing of the cords resulting in a post epiglottic gap (Koopman, 1991). This may be compounded by difficulty in folding over the epiglottis (Ramsey et al., 1955). The cough reflex is the final protection of the airway and between the second and eighth decade the threshold for the cough reflex increases six fold between the second and eighth decade, these changes could potentially increase the risk of aspiration if the swallow was in any way compromised (Pontoppidan and Beecher, 1960;

Horner and Massey, 1991).

OESOPHAGEAL STAGE

Structure

Little work has been done on the structural changes, though grey-white plaques (glycogenic acanthosis) on the mucosa of the aged oesophagus have been found (Egrun and Miskovitz, 1992).

Function

Most recent work has focused on the upper oesophageal sphincter (cricopharyngeus). Fulp et al. (1990) studying young (mean age 37 years) and old subjects (mean age 66 years) noted a lower resting pressure and a delay in relaxation of the upper oesophageal sphincter in the older age group; they considered that this indicated an increased resistance to flow across the upper oesophageal sphincter. Curtis et al. (1984) noticed increasing abnormalities occurring in the cricopharyngeus with increasing age.

As the musculature in the oesophagus weakens with age there is often a perceptible break in the peristaltic wave at the junction of the striated muscle and smooth muscle, near the aortic arch, leaving some barium above the arch

after passage of the primary wave, retroperistalsis frequently occurs in this striated portion, but this is usually of no significance (Sheth and Diner, 1988).

Soergel et al., (1964) suggested that there was slowing of oesophageal transit with increasing age, these changes were particularly marked in those >90 years. As their study group included those with diabetes and dementia, underlying neurological disease may have accounted for these changes. This appeared to confirm earlier work in 1959 (Piaget and Follet (1959), quoted in a review by Zboralske (1965)) where degenerative motor function in the aged oesophagus was considered to be present on cineradiography. Zborlske (1965) states that total muscular activity of the oesophagus in the aged is not normally decreased, but activity is largely uncoordinated. Tertiary contractions (non propulsive) being almost universally present in elderly patients and infrequent in younger patients, are the most common abnormality in "presbyoesophagus". The prevalence of non-peristaltic contraction, segmental tertiary waves, delay of oesophageal emptying and oesophageal dilatation increases in the older person (Jones and Donner, 1988). Yet, others have reported that in "healthy" elderly men (aged 80-90 years) there was reduced peristaltic pressure and magnitude of oesophageal "squeeze" compared to a younger control group, but the duration, speed of contraction (peristaltic) waves and the onset of contraction after swallow initiation was the same (Hollis and Castell, 1974) but may be less efficient (Mandlestam and Lieber, 1970).

This reduction in "squeeze", or peristaltic amplitude (Egrun and Miskovitz, 1992) may be due to reduced muscle tone (Koopman, 1991), secondary to a reduction in the number of ganglion cells/cm^2 in the aged oesophagus when compared to young controls (Eckardt and LeCompte, 1977). Consequently disordered oesophageal function may be caused by disease, and the aged oesophagus has a normal, but inefficient peristalsis.

SUMMARY

Swallowing is complex, and changes in structure and function may occur in any one of the three phases (oral, pharyngeal or oesophageal) with increasing age. Co-ordination remains intact, but as neural control is slower synchronization between the stages of swallowing may not be exact. Consequently there is an increase in the variability of swallowing with less margin for error. If swallowing problems and / or aspiration do occur, a disease process should be suspected.

CHAPTER 4

STROKE AND SWALLOWING

STROKE and SWALLOWING

Swallowing problems frequently occur following stroke. Studies, mainly clinical, have put the occurrence at between 25-62% (Veis and Logemann, 1985; Gordon et al., 1987; Barer, 1989; Gresham, 1990; Horner et al. 1991; Robbins et al.,1993).

In the last century Bastin (1898) identified a case of dysphagia associated with dysphasia, suggesting a cortical aetiology for the swallowing dysfunction. In 1973, Meadows (1973) reviewed the literature and concluded saying that dysphagia following unilateral stroke was the exception rather than the rule. Until relatively recently the literature (Meadows, 1973) and major textbooks (Bickerstaff, 1980; Walton, 1985) stated that swallowing problems, or dysphagia, following stroke, occurred following bilateral hemispheric, or brainstem lesions. Other major textbooks do not even mention dysphagia as a complication of unilateral hemisphere stroke (Tallis, 1989). It is now accepted, on the basis of clinical and radiographic studies, that unilateral hemisphere strokes can result in dysphagia (Gordon et al., 1987; Robbins and Levine, 1988; Barer, 1989).

PREVALENCE OF SWALLOWING PROBLEMS

The ability to swallow has been assessed both clinically, at the bedside and

with videofluoroscopy. These studies have often been conducted at different times following stroke and by using different assessments.

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Clinical studies

Only four studies (Gordon et al., 1987; Barer, 1989; Gresham, 1990; Kidd et al., 1993) have been carried out to look at the occurrence of dysphagia within the first 24-48 hours following the stroke. They were all conducted prospectively, and used various bedside assessments of different complexities.

Barer (1989) studied the ability of patients, recruited into a study of a β receptor antagonist following acute stroke. Twenty eight percent of patients with unilateral hemisphere stroke were found to have dysphagia (difficulty swallowing 90 mls of water). Patients with severe dysphagia were excluded from the study, as were unconscious patients. Speech and language therapists did not assess the patients.

Gresham (1990), studied patients admitted acutely to a local hospital in Australia. Dysphagia was found to be present in 54% of those patients referred to the speech and language therapists. Only 61% of patients admitted with acute stroke were referred, and the referring physician may have missed minor swallowing problems. The assessment used by the medical staff was not reported.

Gordon et al. (1987) looked at an unselected cohort of patients admitted consecutively to hospital with a diagnosis of stroke. Ninety one patients had their swallow assessed by their ability to swallow 50 mls of water. Forty five percent were found to have dysphagia with swallowing (coughing and choking on swallowing). Unfortunately 29% of patients were admitted to the study after 48 hours, and none of the assessments were conducted by a speech and language therapist.

Kidd et al. (1993) studied 60 patients admitted with a diagnosis of acute stroke using both a bedside assessment and videofluoroscopy. These patients were all conscious and had no significant underlying medical condition. Patients were studied within 72 hours of admission, therefore a proportion of patients with transient dysphagia may have been missed, as Gordon et al. (1987) had previously found that many swallowing problems resolved in the first 48 hours. Forty two percent were noted to have dysphagia and 42% were aspirating on videofluoroscopy, 50% silently.

Radiological studies of Swallowing

Radiographic studies of swallowing have been conducted since the 1950s (Ardran and Kemp, 1952,1961,1967; Ramsey et al., 1955), but more recently videofluoroscopy has been used, and accepted as the 'gold standard'

(Logemann, 1983; Bastian, 1991) both to look at the normal swallow and that following stroke (Logemann, 1983; Robbins and Levine, 1988; Jones and Donner, 1991; Horner et al., 1991, Linden et al., 1993).

With the exception of studies conducted by Robbins and co workers (1988, 1993), these studies have been conducted months to years after the stroke, often on patients with persistent swallowing difficulties (Horner and Massey, 1988; Splaingard et al., 1988; DePippo et al., 1992).

Robbins and co workers (1988, 1993) have conducted two prospective studies, comparing the swallow of patients with their first hemispheric ischaemic stroke with that of age matched controls. Videofluoroscopy was carried out 3 weeks after the stroke. All stroke patients were found to have delays in one or more stages of the swallow.

Whereas Robbins (1988, 1993) studied stroke patients, following their first ischaemic stroke, Horner and co-workers (Horner and Massey, 1988, 1991; Horner et al., 1990) have studied various groups of patients (all strokes, bilateral strokes, brainstem strokes) with persistent swallowing problems referred for swallowing assessment. They have, in these select groups, found up that to 91% had swallowing difficulties, predominantly a delay in the swallowing reflex.

The problem with many of the stroke studies are that they often study a small select group of patients (Horner and Massey, 1988; Robbins and Levine, 1988; Chen et al., 1990; Alberts et al., 1992), from a hospital based population, and often study them a long time after the ictus (Horner and Massey, 1988; Splaingard et al, 1988). This is of particular importance as the majority of swallowing problems may resolve spontaneously within the first two weeks (Gordon et al., 1987). No studies have been conducted looking at the occurrence of swallowing difficulties in the entire stroke population.

STROKE and SWALLOWING

Dysphagia may result from disease of the cerebral cortex, brainstem, or cranial nerves (Bucholz, 1987). Severe intracranial lesions, whether focal or diffuse (Mrazek, 1969), loss of consciousness for more than 24 hours and increased intracranial pressure (Lazarus and Logemann, 1987) are associated with moderate or severe swallowing difficulties and increased incidence of aspiration.

Brainstem Lesions

Acute focal brainstem infarct may produce acute dysphagia with little or no other neurological deficit (Jones and Donner, 1988). Paresis of the nineth to twelfth cranial nerves (Butcher, 1982) results in pharyngeal dysmobility and

asymmetry (Horner et al., 1991), incomplete laryngeal closure, vallecula pooling (Nathadwarawala et al., 1992), and incomplete relaxation (Borgström and Ekberg, 1988; Horner et al., 1991) or spasm (Miller, 1972a; Bevan and Griffiths, 1989) of the cricopharyngeus.

Horner et al. (1991), found that 62.5% of brainstem stroke patients aspirated, most had lesions involving the medulla or pons. The risk of aspiration was greatly increased if there was bilateral involvement, and aspiration may be silent (Horner et al., 1990).

Cortex

Majority of cortical lesions, that result in dysphagia, will affect the precentral gyrus or internal capsule (Curtis, 1982). Lesions here will affect the voluntary actions of pharyngeal and laryngeal support musculature on the contralateral side, with spasticity and peristaltic dyscoordination which may, in turn, lead to aspiration.

Levine et al. (1992) found that the presence of asymptomatic periventricular white matter lesions, in other wise normal subjects, were associated with prolongation in the duration of swallowing, but that this was not related to lesion side. Similarly, Johnson et al. (1992) found that there was a prolongation of swallowing time in a small number of patients following stroke.

SIDE OF LESION

In 1973, Meadows (1973) suggested that left cortex damage results in decreased oral motor activity and apraxia, whereas right cortical damage is associated with delayed triggering (Robbins and Levine, 1988), pharyngeal pooling, airway penetration and aspiration. Work by Robbins and Levine (1988) and Robbins et al. (1993) appear to confirm this. Robbins and Levine (1988) suggested that the delay in the oral phase of swallowing was due to altered tongue control. Chen et al. (1990) were not able to confirm a sidedness to the swallow and more recent work, using MRI (Alberts et al., 1992) suggests that stroke size and location does not correlate with the presence of aspiration. Alberts et al. (1992) considered that their results supported a concept that swallowing function might be diffusely distributed throughout the brain and may not be located in any specific site within the brainstem or cortex. The major fault with these studies is that they have studied less than 100 patients between them, and those with more severe stroke were not included.

Lesions in the lower part of the inferior precentral gyrus or posterior portion of the inferior frontal gyrus in either hemisphere show a delay in the initiation of pharyngeal response, suggesting a problem with feedback (Miller, 1982). Robbins and Levine (1988) suggest that the left premotor cortex is the dominant cortical area for volitional swallowing .

HOW IS SWALLOWING AFFECTED?

Stroke may affect either the oral and / or pharyngeal phase of swallowing (Robbins and Levine, 1988; Robbins et al., 1993). Irrespective of the site of the neurological lesion and the consequent clinical signs, any patient badly positioned at the time of swallowing will be at risk of aspirating. If the conscious level of a patient is reduced then the risk of aspiration is higher, as the ability to protect the airway is less (Groher, 1984).

Veis and Logemann (1985) studying 38 patients referred with a swallowing difficulty, up to 4 months after the event, found that 82% had delayed triggering of the swallow reflex, 58% reduced pharyngeal peristalsis and 50% reduced lingual control. Stroke patients are more likely to have a longer and more variable oral phase (Robbins and Levine, 1988). Majority of stroke patients have a combination of oral and pharyngeal problems (Robbins and Levine, 1988), and those with pharyngeal palsy were found only likely to aspirate if the oral stage was also slow (Horner and Massey, 1988). More recently Kidd et al. (1993) have suggested that the presence of an impaired pharyngeal sensation will result in aspiration. Feinberg et al. (1990) have noted that aspiration appeared to occur more frequently during or after the pharyngeal stage.

A delayed oral phase with late elevation of the cricoid, and hence larynx, or

premature descent results in a compromised airway (Bachman, 1959), especially as the vocal cords on their own may not fully protect the airway (Elliot, 1988). If the cords are also affected closure will not be complete and the cords may not align (Curtis, 1982).

Aspiration is dependent on the impairment present and the characteristics of the bolus (Linden and Siebens, 1983; Logemann, 1983; Jones and Donner, 1991). Paralysis of the tongue and/or soft palate makes the control of a liquid bolus difficult, as it does not possess the cohesiveness of paste (Linden and Siebens, 1983; Logemann, 1983). Consequently the liquid bolus will enter the pharynx early with the risk of entering the unprotected airway before the swallow has triggered (Linden and Siebens, 1983). Therefore oral stage problems and delay in triggering of the swallow should be managed with thickened fluids or puree (Logemann, 1991).

Conversely if pharyngeal weakness, pooling and bolus retention are a problem, liquids would be preferable to thick puree and solids (Logemann, 1991). Liquids pass through the pharynx with a shorter transit time (Dantas et al., 1990).

Damage to the parietal lobe of the brain with unilateral neglect may result in pocketing of food in the paralysed cheek (Miller, 1984). This may result in

food entering the pharynx during inspiration, when the airway is wide open.

Those patients with pseudobulbar palsy have loss of voluntary control over their swallow, which is uncoordinated. This is compounded by lack of perception of food put into the oral cavity, poor judgement of bolus size, the amount of chewing required, distractibility, and attempting to eat and talk at the same time (Miller, 1984).

Disturbance in organization of the motor sequence with right sided cortical lesions (praxia of eating), results in the inability to move food from the plate to mouth and perform the oral phase (Miller, 1984).

Stroke may affect the respiratory pattern (Rout et al., 1971) and alter the relationship of breathing with eating. During a normal respiratory cycle, breathing ceases during swallowing and expiration occurs, following a stroke the patient may breath in at the end of a swallow increasing the risk of aspiration (Selley et al., 1988) occurring especially if the pharynx has not been cleared (Elliot, 1988).

HOW IS SWALLOWING ASSESSED ?

The assessment of the ability to swallow is paramount if the presence of any

of the above swallowing difficulties are to be managed appropriately. A full assessment consists of a clinical bedside assessment (BSA) and, where indicated, further more complex investigative techniques.

The Clinical Bedside Assessment

As the importance of dysphagia following stroke has become increasingly recognised, the need for an accurate BSA has become more prominent. Traditionally the clinical assessment of some ones ability to swallow has been the domain of the speech and language therapist, but with increasing work loads there is need for a BSA that can be used by medical and nursing staff.

The assessment conducted by speech and language therapists is thorough, including examination of the anatomy and neurology of swallowing, and the swallowing mechanism itself (Logemann, 1983, Groher, 1984). This takes time, and even then may not be accurate (Splaingard et al., 1988). Smithard et al., (1993) and Splaingard et al. (1988) have noted that nearly half of those aspirating are not detected.

Many workers have relied on a simple swallow test of a fixed volume of water to assess the ability to swallow (Gordon et al., 1987; Barer, 1989; Horner and Massey, Horner et al; Horner et al. DePippo et al. 1992). Those UK based studies (Gordon et al., 1987; Barer, 1989) did not use videofluoroscopy to

confirm their clinical findings and asses the sensitivity and specificity of their assessment. Other workers have studied stroke patients both clinically and with videofluoroscopy (Veis and Logemann, 1985; Horner and Massey, 1988; Horner et al., 1988, 1990) and have noted that the commonest indicators of aspiration following a swallow of water were a wet voice, dysphonia, cough on swallowing and a prolonged swallowing time (Nathadwarawala et al., 1992). Kidd et al (1993), in the only study to use videofluoroscopy during the acute phase of stroke, recently suggested that the absence of pharyngeal sensation had a 100% sensitivity at predicting aspiration on videofluoroscopy. This result may be spurious as 24 of 60 patients had dysphasia.

The gag reflex is often considered to be involved in the swallowing process. Some of the earlier studies appeared to support this (Horner et al., 1990; Linden and Siebens, 1983), but these studies were conducted in patients many months following their stroke. Studies assessing stroke patients ability to swallow during the first few days and weeks following stroke have not confirmed this evidence (Logemann, 1983; Horner and Massey, 1988 Stanners, 1993; Bleach, 1993). More recently work done by Davies et al (1995) demonstrated that the gag reflex was absent in half of fit elderly people and a third of young volunteers despite them all having a normal and safe swallow. Despite this evidence many doctors in training and nursing staff rely on the gag reflex as a proxy for a safe swallow (Smithard et al., 1996).

Linden et al. (1993) studied the ability to correctly predict aspiration at the bedside. They published their BSA which was 7 pages long (Linden et al., 1993). If the assessment is to be conducted quickly, safely and accurately at the bedside a simple, short and sensitive test, that has ben validated against videofluoroscopy, needs to be developed. So far this tool does not exist.

Radiological assessment of the swallow

The investigative methods available to look at the swallowing mechanism include manometry, electromyography, scintigraphy, ultrasound and fluoroscopy. Manometry and electromyography have their place in the assessment of the swallow, but are not part of routine clinical practice.

Scintigraphy

Scintigraphy involves the use of radioisotopes. It is a technique that allows one to examine the whole swallowing process in one go. It will give information regarding the presence of aspiration and oesophageal reflux (Espinola, 1988). It does not give any information with regards to structure of the system and can only examine one swallow. It will allow a degree of quantifiable assessment of the amount aspirated. Scintigraphy can be used to assess changes in aspiration with time (Hamlet et al., 1992) and Shaw et al. (1990) have stated that its use is simple, reliable and sensitive. Unfortunately the results of only 9 patients were included in this study.

Ultrasound

Ultrasound is useful, safe, portable and hence could be used at the bedside. It is able to assess a dry swallow as well as swallows using water. Its main draw back is that cannot image the whole swallowing mechanism at any one time (Sonies, 1991), this is particularly marked in men. Ultrasound images in real time, but the true cords can only be visualised 50% of the time.

Nasal endoscopy

This technique, often used by the ENT surgeon as an adjunct to examine the nares, pharynx and larynx, has in recent years been harnessed to study the swallow (Bastian, 1991; Langmore et al., 1991). It has the advantage of being able to be used at the bedside, and repeated examinations can be conducted without radiation exposure. If the endoscope is harnessed to a video recorder, the study can be viewed and reported on at a later date. Although it gives good views of the pharynx and larynx, which are three dimensional, it offers no information with regard to the oral stage (Langmore et al., 1991). Aspiration has to be summized from residue seen in the larynx and pharynx (Bastian, 1991) as views of the larynx are obscured during the swallow. It is accepted that the endoscope will give good information on structure and vocal cord kinetics (Shaker et al., 1989), some workers consider that it is unable to give any information regarding pharyngeal motility (Jones and Donner, 1988). Unfortunately no information can be gathered concerning the function of the

upper oesophageal sphincter or the oesophagus Bastian, 1991).

Fluoroscopy

Oesophageal dysphagia over the years has been assessed by the 'formal barium

swallow', until the increasing use of gastroscopy. During this examination

large volumes of barium are administered, which may result in aspiration of

large quantities of barium if pharyngeal dysphagia is present and the results

may be fatal (Gray, 1988). Videofluoroscopy (a modified barium swallow) has

been developed (Logemann, 1983, 1986, 1991), to replace the barium swallow

in the investigation of the pharyngeal swallow. Cineradiography gives images

with better definition, but with the risk of higher radiation exposure and more

expense (Logemann, 1983).

Videofluoroscopy enables all three phases of the swallow to be studied in one

examination, the recording onto video tape allows analysis at a later date by

both the speech and language therapist and radiologist (Logemann, 1991). The

examination should be conducted both in the lateral and anteroposterior

projection (Logemann, 1983; Horner et al., 1988). The lateral view allows

imaging of the oral, pharyngeal and oesophageal phase. Pharyngeal

distensibility, function and relaxation of the upper oesophageal sphincter, and

the presence or absence of aspiration can be assessed. Symmetry is observed

best in the anteroposterior plane.

Videofluoroscopy allows for dynamic imaging (Bucholz, 1987), the accurate diagnosis of swallowing difficulties and hence allowing treatment decisions to be made. The examination is safe, using only small volumes of high density barium (Logemann, 1983,1986; Splaingard et al., 1988), which can be thickened, thinned or added to differing food consistencies to stress the swallow (Logemann, 1983, 1991; Robbins et al., 1987). The examination will enable even the smallest amount of aspiration to be detected (Fienberg et al., 1990; Miller, 1984), and is considered the most useful examination for the investigation of oro-pharyngeal dysfunction (Shaw et al., 1990; Logemann et al., 1986, 1991). During the examination the speech and language therapist is able to conduct trials of compensatory manoeuvres or swallowing techniques (Logemann, 1983, 1991) to enable the patient to swallow safely. For the above reasons videofluoroscopy has been considered the 'gold standard' for the examination of the swallowing mechanism.

Unfortunately the examination is limited for technical reasons. It has to be conducted in the X-Ray Department, and unless special chairs are available the patient needs to be able to stand. Some screening apparatus will allow the porter's trolley between the table and camera, permitting the examination to proceed, where a chair is unavailable.

Videofluoroscopy and nasal endoscopy are complementary to the clinical examination, adding information to that already gathered at the bedside. They should not replace the clinical assessment, and not all patients will require to undergo videofluoroscopy.

At present nasal endoscopy should be reserved for those patients unable to go to the X-Ray Department. When the patient has medically improved, further consideration should be given to the requirement for a videofluoroscopy examination.

SUMMARY

Swallowing problems following stroke are common occurring in up to 63% of all stroke patients. No specific site or side of lesion has been consistently associated with dysphagia or aspiration. This may be due to the timing of those studies so far conducted, none have examined the swallow in the first 48 hours. Is the presence of a specific hemisphere side or site time dependent? Is it a function of the studies so far conducted? Examining the same cohort at two different time points may help to give the answer.

Difficulties may occur in any phase of the swallow. Difficulties with swallowing may not be due to an abnormal swallowing mechanism, but due

to an abnormal respiratory pattern, poor positioning or reduced conscious level.

A universally accepted clinical bedside assessment of swallowing has not been developed. Most investigators use their own assessment tool. Only one study has examined the role of a clinical bedside assessment in the assessment of swallowing during the acute phase of stroke, using videofluorosocpy to help validate the findings. There remains a paucity of studies in this area. As the prevalence of dysphagia is high at the time of admission, and given that there is a risk of aspiration there is a need for a tool that can detect the difference between a normal (safe) and an abnormal and unsafe (at risk of aspiration) swallow, preventing many patients being put nil by mouth unnecessarily. A simple assessment tool that can be used by all staff needs to be developed.

Videofluoroscopy is the 'gold standard' for the radiological investigation of swallowing, but only examines the swallow for a short period of time. The reliability of the report following videofluoroscopy is examiner dependent, is their consistency between reporters?

At the present time studies have not examined the natural history of swallowing. All studies have assumed that a safe swallow always remains safe and that aspiration does not occur late in the history of stroke. Does a safe

swallow always remain so?

CHAPTER 5

MORBIDITY

ASPIRATION

Aspiration is a generic term referring to material penetrating the larynx and entering the airway below the true vocal cords (Logemann, 1983), whilst silent aspiration is defined as penetration of saliva or food below the level of the true vocal cords, without cough or any other outward sign of difficulty (Linden and Siebens, 1983; Horner and Massey, 1988).

For aspiration to occur, impairment of the closing mechanism or a pathological communication between the nasopharynx and the trachea is needed (Donner, 1974). Entry into the supraglottis i.e. below the epiglottis may be common, but the degree of penetration is important. Entry above the false vocal cords may be a normal phenomenon (Blonsky et al., 1975; Curtis and Curess, 1984; Curtis et al., 1985), and often results in the clearance of the larynx as the swallow progresses.

It is believed that aspiration, per se does not lead to serious consequences in normal people (Bartlett and Gorbach, 1975). Huxley et al. (1978), using indium[111] chloride showed that up to 45% of normal people aspirate whilst asleep and the lack of any ill effects following aspiration had been reported earlier by Amberson in 1937 (Amberson, 1937).

Complications following aspiration are related to the frequency of aspiration,

volume of aspiration and the nature of the aspirate (Donner, 1974). Aspiration may be bolus specific; liquids may penetrate and solids not (Linden and Siebens, 1983).

WHY DOES ASPIRATION OCCUR ?

Feinberg et al. (1990) found that in their experience aspiration is more commonly associated with depression of conscious level rather than specific neuromuscular disorders. The most common mechanical reasons for aspiration are incomplete laryngeal elevation and closure; and weak or absent pharyngeal swallow (Veis and Logemann, 1985; Horner and Massey, 1988).

Barium or food may enter the airway as a result of muscular or neurological dysfunction / dyscoordination (Curtis and Hudson, 1983; Feinberg et al., 1990), which may be secondary to debilitation, or dementia (Feinberg et al., 1992). Paralysis of the muscles of the floor of the mouth or altered functions of the recurrent laryngeal nerve, may suppress important safety mechanisms leading to aspiration (Donner, 1974). Problems lower down e.g. with the cricoarytenoids or cricothyroid muscle would lead to larger volumes being aspirated, and in the case of the cricothyroid asymmetrical elevation of the larynx, and reduced vocal cord closure would occur (Curtis and Hudson, 1983). As the severity of dysphagia increases, there is an increasing risk of

aspiration and hence pneumonia (Kasprisin et al., 1989).

WHEN DOES ASPIRATION OCCUR ?

Aspiration will occur:

1)   Before swallow: Prior to the swallow reflex occurring, the bolus is controlled by the tongue. Early, uncontrolled presentation of the bolus to the pharynx prior to the swallow commencing will result in a bolus being presented to the pharyngeal phase whilst the airway is unprotected (Logemann, 1986).

2)   During swallow: A delayed swallow reflex or poor laryngeal closure and/or elevation during a swallow, may result in the airway being unprotected at the time of bolus passage with the concomitant risk of aspiration (Logemann, 1986).

3)   After swallow: If there is poor pharyngeal peristalsis, cricopharyngeal dysfunction (Logemann, 1983), and consequently, poor pharyngeal clearing, residual solid or liquid remains in the valleculae and pyriform sinuses. This residue may enter into the airway after the swallow (Lazarus and Logemann, 1987), when inspiration begins (Selley et al., 1989a; 1989b).

Aspiration only tends to occur at one phase of the swallowing sequence, most frequently during the pharyngeal stage (Feinberg et al., 1990). In 1985, Veis and Logemann, noted that following stroke, of those that were aspirating, 82% did so due to delayed swallowing reflex. Horner et al. (1988) noted that, of those aspirating 91% had a delayed swallow with increased pharyngeal residue in 75%. Though later work found that 39% of those without aspiration had pharyngeal abnormalities (Horner et al., 1990).

HOW DOES ASPIRATION OCCUR?

The pathogenesis of laryngeal aspiration or penetration of a swallowed bolus, frequently observed in patients with oropharyngeal dysphagia, may have several causes:

a] Premature spill of liquid from the mouth into the pharynx (Logemann, 1986).

b] Abnormal coordination of oropharyngeal motility and glottic closure.

c] Abnormal glottic closure mechanism (Logemann, 1986).

d] Ineffective bolus transport, resulting in large amounts of post-swallow pharyngeal residue (Shaker et al., 1990).

Patients with delayed reflex with/without tongue control frequently aspirate

liquids. The more liquid/fluid a substance, the more likely it would be aspirated and travel further into the respiratory system (Feinberg et al., 1990). After swallowing thicker consistencies there is more residue in the pharynx (Lazarus and Logemann, 1987).

Cineradiography may show stasis of barium in the pharynx, pooling in pyriform fossae or frank aspiration (Merlo and Cohen, 1988). Pooling may lead to aspiration as the larynx lowers and inspiration occurs. Weak vocal cord adduction would compound this and increase the risk (Elliot, 1988). A frozen (paralysed) larynx can result in significant aspiration (Jones and Donner, 1988).

PREVALENCE OF SWALLOWING PROBLEMS

The prevalence of swallowing problems in stroke patients is high (Barer, 1987; Gordon et al., 1987; Elliot, 1988; Gresham, 1990). How many of these patients aspirate? The answer to this question is not known, few of the studies looking at the presence of aspiration have been conducted earlier than three weeks post stroke (Horner and Massey, 1988; Robbins et al., 1988; Horner et al., 1991; Kidd et al., 1993; Robbins et al., 1993).

There are no studies examining the prevalence of swallowing problems in an

unselected cohort of stroke patients, many have only studied those with persistent swallowing problems (Veis and Logemann, 1985; Splaingard et al., 1988; Horner et al., 1990; Horner et al., 1991; DePippo et al., 1992). Horner and co workers (1988, 1990, 1991) in a series of studies, found that approximately half the patients aspirated and half of these were silent aspirators. Unfortunately, these results only tell us that half of those patients with swallowing difficulties are aspirating at times between a few months and 2 years post stroke.

PREDICTING ASPIRATION

The commonest signs suggested for the detection of aspiration are a weak cough (Horner and Massey, 1988; Horner et al., 1990, 1991; Coelho and Ferranti, 1991), dysphonia (Linden and Siebens, 1983; Horner and Massey, 1988; Horner et al., 1988), wet voice (Linden and Siebens, 1983; Horner and Massey 1988), and an impaired pharyngeal gag (Linden and Siebens, 1983; Horner et al., 1990).

None of these signs on its own has been shown to be sensitive or specific enough to predict the presence of aspiration. For instance a wet voice which may indicate pharyngeal paresis, with pooling of secretions in the valleculae and pyriform sinuses (Ardren and Kemp, 1952) occurs only in one third of

those aspirating, and where dysphonia occurs in 91% of those aspirating it is also seen in 60% of those not aspirating (Linden and Siebens, 1983; Horner et al., 1990). Horner found, in patients with bilateral strokes, that a weak voluntary cough was suggestive of aspiration. This was not supported in a study with a more heterogenous population of stroke patients (Horner et al., 1988). The gag reflex is there to prevent material entering the pharynx and has a role completely different to that of swallowing (Logemann, 1988), and on its own an impaired pharyngeal gag does not indicate the inability to swallow (Davies et al, 1995). Recently Kidd et al. (1993) have suggested that the absence of pharyngeal sensation predicts aspiration with a sensitivity of 100%.

Studies investigating the use of a combination of these signs were only able to predict the presence of aspiration in 42% (Splaingard et al., 1988) or more recently 67% when using an extensive proforma (Linden et al. 1993)

MORBIDITY

The morbidity associated with dysphagia may be insignificant or may cause major problems with repeated infection, poor nutrition and embarrassment (Gustafsson and Tibbling, 1991).

The swallowing literature has been concerned with the site of the neurological

lesion (Robbins and Levine, 1988; Alberts et al., 1992) and the occurrence and detection of aspiration (Horner and Massey, 1988; Splaingard et al., 1988; Gresham, 1990; Horner et al., 1991). The related complications, for example, dehydration and malnutrition have had little attention, consequently their true prevalence and their importance are uncertain.

Complications of aspiration

The complications resulting from aspiration include: persistent cough, fluctuating hoarseness, recurrent pulmonary infections, and severe under nutrition (Hellemans et al., 1981). Ekberg and Hilderfors (1985), studying patients after oesophagectomy, found that most patients with severe swallowing dysfunction appear to be remarkably unaffected during daily living.

Chest infection

How often does chest infection occur in dysphagic stroke patients with aspiration? Elliot (1988), has stated that aspiration pneumonia accounts for 6% of deaths acutely following stroke. Gordon et al. (1987) found a 19% incidence of bronchopneumonia in those with dysphagia as opposed to 8% of those without. Ekberg and Hilderfors (1985) found an incidence of 13% in ambulatory patients, with various causes of dysphagia, that had been

discharged from in-patient care. Post mortem studies show an incidence of aspiration in 16% of stroke patients (Silver et al., 1984) and Brown and Glassenberg (1973) found the presence of bronchopneumonia and bronchitis in 98/209 cases and 11/209 had aspiration pneumonia. Mrazek (1969) noted during a postmortem study that in 2/3 of cases of bronchopneumonia and in 63/65 patients with documented dysphagia there was evidence of aspiration.

Kaldor and Berlin (1981) noted that 55/71 had pneumonia on the side of paresis, though this was not supported by Mulley (1982). Normal defence of the lung is by the mucocilary 'staircase', coughing, polymorphonucleocytes, alveolar macrophages and interstitial lymphocytes (Pennza, 1989), this may be compromised by poor nutrition (Chandra, 1990), and the risk will be compounded where there are abnormal chest wall movements (De Troyer, 1981).

Aspiration pneumonia has been documented to be a common sequel to aspiration (Gordon et al., 1987; Schmidt et al., 1994) and aspiration of oropharyngeal secretions is considered to be the most important mechanism in the pathogenesis of bacterial pneumonia (Huxley 1978). Aspiration of oropharyngeal bacteria is implicated as the most common route of entry for both community acquired and nosocomial pneumonia (Pennza, 1989). If, following stroke, poor oral care is given there will be multiplication of the

usual organisms eg E.Coli and an increase in the presence of anaerobes (Bartlett and Gorbach, 1975). Most commonly pneumonia occurs from aspiration of upper airway pathogens. If a chest radiograph of good quality fails to reveal new pulmonary infiltrates then the diagnosis is suspect (Gleckman and Bergman, 1987). Predisposing factors for infection, which are often present following stroke, include a reduced level of consciousness, presence of dysphagia and mechanical disruption of the cardiac sphincter eg nasogastric tube (Bartlett and Gorbach, 1975; Harkness et al., 1990).

The presence of pneumonia and other respiratory sequelae depends not only on the above factors but also on the nature of the aspirate (chemical, physical, bacteriological), the amount and frequency of aspiration and the status of the host defence. Direct inhalation of vegetable matter leads to granulomatous changes; animal fats to necrotizing lesions and acid severe inflammation (Feinberg et al., 1990).

The diagnosis of aspiration pneumonia can be difficult, but important due to its high fatality rate (Cameron et al., 1973; Gleckman and Bergman, 1987; Harkness et al., 1990). Prandial aspirators sometimes develop febrile illness that last 24-72 hours without respiratory symptoms and fleeting infiltrates on chest radiograph (Ekberg and Hilderfors, 1985; Fienberg et al., 1990). Pneumonia is defined as due to aspiration when confirmed by radiographic and

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/or cytologic studies (Kasprisin et al., 1989).

Dehydration

Any one will become dehydrated (water deplete) if their water loss exceeds their intake. The risk of dehydration is increased if there is an inability to fend for themselves, and they have poor mobility or are immobile. Older people report that their mouth feels less dry with dehydration, consequently reduced oral sensation may play an important role in hypodypsia with age (Morley, 1986). This may contribute to the raised osmolality associated with those with most disability (O'Neill et al., 1989; 1990).

Dehydration will lead to death as a consequence of azotemia, hypernatraemia, hyperkalaemia and hypercalcaemia, all of which will produce sedative and therefore an anaesthetic effect on the body (Groher, 1990).

Various workers (Gordon et al., 1987; Splaingard et al., 1988; Barer, 1989) have suggested that dehydration occurs in those with dysphagia following acute stroke, but their results did not reach statistical significance. These groups (Gordon et al., 1987; Barer, 1989) measured urea and haematocrit but not osmolality. In stroke patients fluid balance is generally managed badly. Those patients with an intravenous infusion are overhydrated and those allowed to drink may not drink enough (O'Neill et al., 1990).

Nutrition

As people grow older there is a change in body composition; decreases in water, cell mass, and bone mineral result in decreased density (Yearick, 1978). With increasing age there is an increased risk of a poor nutritional status often secondary to a poor diet. Those aged >79 years are at most likely to be under nourished (Larsson et al., 1990). Larsson et al. (1990) noted that, of those admitted to hospital, up to 30% will have a degree of malnutrition. Not only the presence or absence of fat but, also its distribution is important as a health risk (Morley, 1986).

Being in an undernourished state is of far greater prognostic importance than obesity (Morley, 1986). Protein energy malnutrition impairs several aspects of the immune system, cell mediated immunity, ability to kill ingested bacteria and fungi, several components of the complement system, mucosal secretory IgA and antibody affinity. These changes lead to an increased risk of infection further immune suppression and worsening malnutrition (Chandra, 1990; Larsson et al., 1990).

Despite the association with age, this decline in immunity is not inherent with ageing, as 20-25% of older people have a normal response (Chandra, 1990).

Nutrition and Stroke

The nutritional status in patients with dysphagia and stroke is poorly documented. If all stroke patients are hypercatabolic in the immediate post ictus period as haemorrhagic strokes appear to be (Touho et al., 1990), then starting from a poor base line will compound matters. There could be an increase in morbidity (McEvoy and James, 1982), with infection often being related to poor nutritional status in stroke patients (Viitanen et al., 1987; Axelsson, et al., 1988, 1989).

Splaingard et al. (1988) found no statistical difference between those with dysphagia and those without using hydration, total protein and albumen as markers. How hydration was measured is not clear. Work by Axelsson et al. (1988, 1989) in Scandinavia has shown, using biochemical and anthropometric indices, that following stroke there tends to be a decrease in nutritional status of patients in the first few months. They noted that patients with more than one marker for malnutrition failed to improve their nutritional status, and would in fact deteriorate nutritionally during their stay in hospital, and have a greater length of stay in hospital. This was worse in those patients with dysphagia.

Those patients with the worse strokes tend to a low affect and be introspective, with consequent anorexia and confusion leading to a vicious circle perpetuating

the malnutrition (Axelsson et al., 1989). Veldee (Veldee and Peth, 1992) also postulates that protein calorie malnutrition may affect the muscles of swallowing, therefore with dysphagia leading to malnutrition, thence weakness of the deglutitive musculature and worsening of the dysphagia. Arora and Rochester (1982) suggest that weakness of the respiratory muscles and reduced ability to cough will occur with malnutrition and this will increase the chances of infection. The ability to cough may be impeded by recurrent small volume aspiration leading to suppression of the cough reflex (Arora and Rochester 1982).

OUTCOME

Those patients with dysphagia are considered to have a worse prognosis. Wade and Hewer (1987) found no increased fatality rate in those who choked as compared to those who were simply slow in eating; but 43% of conscious patients seen within the first week of the acute stroke had swallowing difficulties, and they were noted to have a higher fatality rate (35-40%), and survivors had reduced function. Barer (1989) suggested that patients with difficulty swallowing have a worse outcome. But is this due to the dysphagia and its complications or the stroke?

SUMMARY

Swallowing difficulties following stroke occur and may be complicated by the presence of aspiration. Aspiration may occur with problems in any phase of the swallow, but may be bolus specific and can be silent.

Not all incidences of aspiration result in complications, but poor nutrition, dehydration, chest infections and even death may result. The occurrence of complications appears to be related to the character, volume and frequency of the aspirated material.

Patients developing dysphagia after stroke have an increased occurrence of chest infection, mortality rate and poorer functional status, compared to stroke patients without problems. Is this a function of the severity of the stroke or is dysphagia an independent factor?

In the first few months following acute stroke there is a decline in the nutritional status of those persons admitted to hospital, particularly if dysphagia is present. Is this marked decline in nutritional status due to dysphagia or aspiration?

The prevalence of complications of these complications following stroke and their relationship to dysphagia has not been thoroughly examined in a

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prospective manner. A prospective study is required to examine the role that dysphagia has on outcome following stroke, and to investigate its relationship to the stroke itself.

CHAPTER 6

QUESTIONS TO BE ANSWERED

QUESTIONS TO BE ANSWERED

The preceding review of the literature has revealed the situation regarding dysphagia following stroke as it stands at the time of this work. Many papers have been published looking at the physiology and timing of swallowing both during a normal swallow and following stroke. Many have attempted to localize the control of swallowing to one cerebral hemisphere or the other, but a consensus still does not exist. Other workers have reported the prevalence of dysphagia (swallowing difficulties), and a few have looked at the nutritional status of stroke patients. Non have examined prospectively the relationship between dysphagia and outcome following stroke. Similarly no one has been able to devise a simple bedside assessment of swallowing, validated against videofluoroscopy, that can be used easily and quickly at the bedside.

Following the review of the literature, the following questions remain unanswered.

1. How common is dysphagia (swallowing difficulties) at the time of admission to hospital and what proportion of patients are aspirating during the acute phase of stroke?

2. Can a bedside assessment be used to detect the presence of aspiration? Is it possible to devise a bedside swallowing assessment to be used during the

acute phase of stroke, that has been validated against videofluorosocpy, and what elements of this bedside assessment can be best used to reliably detect or exclude the presence of aspiration?

3. Is there a relationship between the side of the stroke and the occurrence of aspiration? Does the timing of the investigation matter?

4. What is the natural history of swallowing following stroke, and does a safe swallow always remain so?

5. Do stroke patients with dysphagia or aspiration become dehydrated? What happens to the nutritional status of patients following acute stroke?

6. Is aspiration or dysphagia an independent predictor of the occurrence of chest infection, mortality and length of stay, or are they just a function of stroke severity?

NULL HYPOTHESIS

The null hypothesis regarding these studies is:

1: That it will not be possible to detect the presence of aspiration at the bedside.

2: That dysphagia or aspiration do not exert an effect on outcome that is independent of the stroke itself.

The following studies aim to answer these preceding questions, reject the null hypothesis and at the same time suggest the next research questions that need answering.

CHAPTER 7

GENERAL METHODS

METHODS

PATIENT RECRUITMENT

Over a one year period, patients admitted to the Departments of General

Medicine and Geriatric Medicine of the University Hospital of South

Manchester and the Department of Medicine, Manchester Royal Infirmary and

the Robert Barnes Medical Unit, Barnes Hospital were recruited into the study.

INCLUSION CRITERIA

All patients admitted to these hospitals within 24 hours of the onset of the

symptoms of an acute stroke.

EXCLUSION CRITERIA

1. Admission to hospital more than 24 hours after the onset of stroke.

2. Failure to obtain consent from the patient or a significant other (next of kin

or carer). Consent was obtained from the significant other when the patient

was unconscious or severely dysphasic/ aphasic.

3. The presence of other severe illness. This included metastatic carcinoma,

septicaemia with or without shock.

CONFIRMATION OF DIAGNOSIS

The diagnosis of stroke was confirmed by clinical examination, as soon after

admission as possible. The definition of stroke and criteria for diagnosis were that of the World Health Organisation (WHO, 1978). Information was recorded on a simple proforma. The diagnosis of stroke was further confirmed by the use of unenhanced CT scans.

The methods involved with each study are documented within the ensuing chapters.

DATA POINTS

Data was recorded at 4 data points. These were Days 0 (admission), 7, 28 and 180 (6 months).

Medication

As certain classes of medication can have central depressant effects, the type of drugs being taken by the patients at each assessment point were recorded.

END POINTS

These included: 1] The completion of the study at six months.

2] Death.

3] Further stroke(s).

4] Surgery: Fracture neck of femur; Carotid

Endarterectomy.

OUTCOME MEASURES

The following measures were used to assess outcome:

a) Functional outcome was assessed on the following lines

    i) Barthel score at 6 months

    ii) Place of discharge

b) Presence of chest infection

c) Survival (Mortality rate).

d) Length of stay

e) Nutritional status

f) Hydration

STATISTICAL ANALYSIS

Analysis on the data was performed by the Department of Medical Statistics

at the University Hospital of South Manchester. The statistical package SSPS

PC+ and Minitab V8 were used. The following statistical methods were used

during the different studies: chi square, Kruskal-Wallis test, Fisher's exact test

and Mann Whitney U test were used for non-parametric data and Students t-test for the parametric data. For comparison within groups a paired t-test was used, and between groups analysis of variance was performed. Chi square and Kruskal-Wallis were used when comparing the presence/absence of a variable between groups eg the presence of chest infection. To compare categorical data (Barthel scores), Fisher's exact test or Mann Whitney U were used.

Multiple logistic regression analysis was also performed when looking for those elements of the bed side swallow assessment that were best at predicting the presence of aspiration.

Multiple analysis of variance and multiple regression analysis were performed to ascertain the role of dysphagia or aspiration on outcome.

ETHICAL APPROVAL

Ethical approval was sought from and granted by both Central and South Manchester research ethics committees.

CHAPTER 8

GENERAL RESULTS

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PATIENT NUMBERS

One hundred and fifty three patients were recruited during the time of the study. A further 29 were not eligible for inclusion: consent was refused in three cases, 1 patient had previously been entered into the study and presented after 6 months of follow up with a second stroke and was therefore ineligible for the study and a further 23 patients presented to the hospital more than 24 hours after stroke on set. Two patients were not entered into the study due to the presence of other significant pathology that would affect their immediate prognosis.

Four patients were withdrawn from the study at an early stage due to a change of diagnosis. It was considered that 1 patient had had a transient ischaemic attack. The other 3 had cerebral tumours diagnosed by CT scanning (2 primaries and one metastatic). The remaining 149 patients were included in the following studies and the results are presented in the following chapters.

DEMOGRAPHY

Median recruitment time was 14 hours (mean 16, range 0.5 - 50) following the onset of stroke. Th subject recruited late was withdrawn at day 8 due to an extension of their stroke and the data has been used for the validation of the bedside assessment as a bedside assessment was performed within 24 hours of

the videofluoroscopy.

Of the 149 patients studied with a median age of 79 years (range 40-93), 86 (58%) were female. Forty two (28%) had had previous transient ischaemic attacks and 49 (33.0%) previous strokes. Other risk factors present were a history of hypertension in 50 (33.6%), hypotensive drug usage in 38 (25.5%), diabetes 20 (13.4%), and smoking (current or previous) 74 (50.0%). The majority of patients were right handed 132 (88.5%).

Of those patients that had suffered previous clinical strokes 31 had some residual disability, but were independent in activities of daily living, and a further 5 remained dependent in activities of daily living.

Medication used during the study that might cause alterations in conscious level (including drowsiness) included; β-blockers (10 patients), benzodiazepines (mainly temazepam) (38), neuroleptics (12), antidepressants (21), opiates (mainly mild opiates) (29) and antiepileptic therapy (9).

The following chapters describe the various studies conducted within the aegis of the main study.

CHAPTER 9

BEDSIDE SWALLOWING ASSESSMENTS

BEDSIDE SWALLOWING ASSESSMENTS

AIMS

1. To assess the incidence of swallowing difficulties (dysphagia) and aspiration in patients admitted to hospital with acute stroke.

2. To develop a bedside swallowing assessment, validated against videofluoroscopy, that is simple, straight forward and could be easily used by junior medical and nursing staff, during the acute phase of stroke.

METHODS

Patient recruitment and criteria for entry into the study were described earlier.

A bedside clinical swallowing assessment was performed during the first week by both physicians (DOC1, DOC2) and speech and language therapists (SLT1, SLT2). The use of two assessors from each speciality allows the assessment of agreement, using the Kappa statistic (Spitzer et al., 1967), both between and within specialities. DOC and SLT used standardised proformas. The DOC proforma was devised by the author with help from Ms R Wyatt (Chief SLT, Withington Hospital, South Manchester). This was modified on advice from Professor Barer and Dr Ellul. The prime criteria were that it should be simple, straight forward and confined to one side of A4. Elements included within the bedside assessment were those that were considered most relevant by Ms Wyatt,

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and those that were commonly indicated to be predictors of aspiration in the swallowing literature (Veis and Logemann, 1985; Gordon et al., 1987; Horner and Massey, 1988; Splaingard et al., 1988). The assessment was divided into two stages (see appendix). If a patient was unable to swallow a 5 ml spoonful of water (coughing / choking on more than one occasion out of three attempts and or a wet voice (weak laryngeal function)), then stage two was not attempted (swallowing 60 mls of water within 2 minutes). Failure to go on to stage 2; coughing / choking during stage 2; or the presence of a wet voice indicated an unsafe swallow.

The SLT assessment was developed by Ms R Wyatt. The SLT proforma was a condensed version of the accepted practice for the assessment of swallowing as practised at the Withington Hospital. The final decision as to whether a patient's swallow was unsafe or not may be coloured by the speech and language therapists clinical judgement.

DOC1 assessed the ability of patients to swallow, daily for the first four days (Days 0-3), at the end of the first week (Day 7) and then on days 28 and 180 (6 months). DOC2 carried out an assessment within the first 3 days, and within 24 hours of the videofluoroscopic examination.

The speech and language therapist (SLT1) assessed the patient at least 3 times

during the first week, twice before videofluoroscopy and at the end of the first week, then at day 28 and 180. A second speech and language therapist (SLT2) assessed the patient's swallow within 24 hours of videofluoroscopy.

Videofluoroscopy

Videofluoroscopy (modified barium meal) is a simple procedure to perform, and often only involves a short exposure to radiation (1 minute of screening being equivalent to the radiation dose of one chest radiograph). At present the videofluoroscopy is considered the gold standard radiological assessment for determining the presence of aspiration and to examine the structure and function of the swallow (Bastian, 1991).

Small volumes of high density barium, EHZD 250% w/v, were used in differing volumes and consistencies (Logemann, 1983, 1986) to look for the presence or absence of aspiration. A specific objective of the protocol was that all patients should have videofluoroscopy performed within 3 days of admission, unless they were medically unfit, or had a reduced conscious level (Glasgow coma scale (Teasdale et al., 1979) < 10). If the patient was unable to stand for the procedure it was performed with the patient sitting in a special chair (designed by the medical physics department at the University Hospital of South Manchester) or on a patient's hospital trolley.

All the videofluoroscopy examinations were stored on Sony U-matic videotape

and reported at a later date. The results of the videofluoroscopy were reported

as aspirating (barium entering the airway below the true vocal cords (Horner

and Massey, 1988)) or not aspirating. Reporting of the videofluoroscopy was

done blind to the clinical assessments.

All swallow assessments (clinical or radiological) were performed blind to the

other results of the other swallowing assessments.

RESULTS

SWALLOWING ASSESSMENTS

Bedside swallowing assessment

All 149 patients were assessed by a doctor, the majority on the day of

admission, of these, 28 patients never regained consciousness. This left a total

of 121 patients for assessment of the swallow, of which all were assessed by

DOC1 and only 104 were assessed by SLT1. SLT1 did not assess all patients

with a reduced conscious level.

DOC1 considered that 61 (50%) of the 121 patients had a safe swallow, and 60 (50%) had dysphagia. If those with a reduced conscious level are also considered to have an unsafe swallow, 88/149 (59%) patients were assessed to be unsafe. Seventy five patients had their swallow assessed at the bedside by a second doctor, independently of the first medical assessment (DOC2).

Of the 104 assessed by SLT1, 72 (69%) were considered to have a safe swallow and 32 (31%) to have dysphagia. If those with a reduced conscious level are included then a total of 60/132 (46%) were considered to have unsafe swallows. A second speech and language therapist (SLT2) assessed 72 patients at the bed side.

Excluding those with a significantly reduced conscious level, 104 patients were seen by both DOC1 and SLT1.

Videofluoroscopy results

Ninety eight (81%) patients underwent videofluoroscopic examination within a median time of 2 days (interquartile range 1-4). 49 patients did not undergo videofluoroscopy due either to a reduced conscious level or being deemed medically unfit, 2 other patients refused. Only 94 examinations were available

for analysis as one was accidently erased from the video tape, and two were uninterpretable for technical reasons, and one patient had a videofluoroscopy examination but was later too drowsy for a bedside assessment of their swallow. All videofluoroscopy examinations were stored on Sony 100 U-matic video tape and reported at a later date.

Twenty (21%) patients had aspirated, a further 44 (47%) had supraglottic penetration (barium entered the larynx but did not go below the true vocal cords) of barium. The remaining 30 (32%) had neither. As supraglottic penetration is considered a variant of the normal swallow, these patients have subsequently been included in the 'normal' (safe) group.

Clinical and videofluoroscopy findings

All 94 patients undergoing videofluoroscopy examination were assessed by DOC1, 65 by DOC2, but only 83 by SLT1 and 72 by SLT2. DOC1 assessed the patients within a median time of 0 days (Interquartile range 0-1) and SLT1 a median of 1 days (Interquartile range 0-1) of the videofluoroscopy examination.

Only 83 patients were assessed by DOC1, SLT1 and videofluoroscopy. Of these 83 patients assessed by DOC1, SLT1 and videofluoroscopy, 19 (23%)

were seen to aspirate during videofluoroscopy. Only 9 (47%) of these were detected clinically by SLT1 and 13 (68%) by DOC1. The 10 that were not detected by SLT1 (and 6 by DOC1) were deemed to be silently aspirating.

Sixty four patients were judged not to be aspirating on videofluoroscopy. Nine of these were categorised as being unsafe by SLT1, and 21 by DOC1. These were false positive results.

BEDSIDE ASSESSMENT

Assessor accuracy

The sensitivity (true positives / true positives + false negatives), specificity (true negatives/ true negatives + false positives) and predictive values of the bedside assessment were determined by using the VF result as the 'gold standard'.

The DOC1 assessments were analysed twice, firstly compared with the videofluoroscopic result and secondly only analysing the same 83 results as those for the SLT1 assessments (table 1). Looking at the results in this way allowed more direct comparison between DOC1 and SLT1 assessments using their bedside assessments.

These results confirm that the sensitivity of the bedside assessment for the detection of aspiration is low. The specificity of the speech and language therapists' bedside assessments was higher than the physicians (86% vs 66%). As the sensitivities and specificities of a test do not all ways give a guidance as to the clinical usefulness of the test, the positive (PPV) and negative predictive values (NPV) have been calculated. The PPV and NPV for SLT1 were 50% and 85% respectively (table 1).

Assessor agreement

Agreement both between and within professions was assessed using the Kappa (κ) statistic. Kappa is the proportion of agreement over and above that due to chance. The Kappa statistic takes in to account both positive and negative agreements, giving a result between -1 (agreement less than that expected by chance) and 1 (perfect agreement). A result between 0 and 1 suggests that the agreement is better than chance.

There was very good agreement between the speech and language therapists (κ=0.8, agreement 91%). There was only moderate agreement between specialities (DOC1 vs SLT1 κ=0.51) and between the doctors (κ=0.52). The detailed results and 95% confidence intervals are given in table 2.

	SENSITIVITY (%)	SPECIFICITY (%)	PPV (%)	NPV (%)
SLT1 N = 83	9/19 (47)	55/64 (86)	9/18 (50)	55/65 (85)
DOC1 N = 94	14/20 (70)	49/74 (66)	14/39 (36)	49/55 (89)
DOC1 N = 83*	13/19 (68)	43/64 (67)	13/34 (38)	43/49 (88)

*NB To match the same set of 83 patients assessed by SLT1 and VF

Table 1:   Sensitivities, specificities and predictive values for the detection of aspiration.

ASSESSORS	N	AGREEMENT (%)	KAPPA	95% CI
DOC1 v DOC2	65	49/65 (75)	0.5	0.26 - 0.73
DOC 1 v SLT1	83	61/83 (73)	0 .41	0.21 - 0.6
SLT1 v SLT2	74	69/74 (93)	0.79	0.55 - 1.00
DOC2 v SLT1	61	46/61 (75)	0.42	0.17 - 0.67
DOC1 v SLT2	80	53/80 (66)	0.24	0.05 - 0.43
DOC2 v SLT2	57	42/57 (74)	0.35	0.10 - 0.60

Table 2: Agreement between and within specialities

Forty two of the videofluoroscopy results were reported by two separate radiologists. These were performed independently, at different times. Both were blinded to the clinical information. There was moderate agreement, for the reporting that aspiration was present (76% , $\kappa=0.48$).

Predictors of aspiration

The independent value of the elements of the SLT and DOC bedside assessments in predicting the VF result was assessed. This was to determine which parts of the study assessments carried most 'weight' in predicting the presence of aspiration seen during the VF examination.

Many of the elements within the first phases of the bedside assessments of the DOC and SLT were statistically significant (see tables 3 and 4), though no statistical difference was found in the time taken to drink 60 mls of water nor the number of sips to drink it. Consequently we have been unable to confirm the results of other Nathanderalwalla et al. (1992) or DePippo et al. (1992).

A multiple logistic regression analysis was used to determine the optimum subset of the bedside assessment variables for predicting aspiration. The presence of aspiration was the dependent variable, with the other elements

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of the bedside assessment being independent variables. This showed that the independent predictors for aspiration within the SLT1 bedside assessment were: any impairment of conscious level (χ^2 [1]=15.4 p=0.0001); and a weak voluntary cough (χ^2 [1]=5.2 p=0.023). The presence of these criteria were able to predict aspiration with a sensitivity of 75%, specificity of 72%, positive predictive value (PPV) of 41% and negative predictive value (NPV) of 91%.

The optimum predictors within the DOC1 bedside assessment were; conscious level (χ^2 [1]=11.7 p= 0.0006); cough on swallowing 5 mls of water (χ^2 [1]=4.9 p=0.027); and voluntary cough (χ^2 [1]=6.4 p=0.011). When compared to videofluoroscopy, the presence of any of these variables gave a sensitivity of 62% and a specificity of 80% for the presence of aspiration (PPV = 42%, NPV = 90%).

DOC

| ASSESSMENT | ASPIRATING ON VF | | p - value |
	Yes n = 20 (%)	No n = 74 (%)	
*Abnormal sitting balance	10 (50)	13 (18)	0.006
*Conscious level (not alert)	10 (50)	6 (8)	< 0.0001
Abnormal breathing	3 (17)	5 (7)	0.19
Abnormal lip closure	2 (11)	2 (3)	0.17
*Abnormal tongue movement	8 (50)	7 (10)	0.0007
Asymmetrical palate movement	4 (27)	18 (25)	1.0
*Abnormal laryngeal function	7 (41)	7 (10)	0.005
Absent gag	9 (53)	23 (32)	0.17
*Weak/absent cough	9 (53)	7 (10)	0.0003

Stage 1: Teaspoon of water

*Dribbles more than once	5 (29)	6 (8)	0.030
*No laryngeal movement	2 (12)	0 (0)	0.034
*Repeated movement > once	5 (29)	4 (6)	0.011
*Cough on swallowing > once	8 (47)	5 (7)	0.0002
Stridulous on swallowing	2 (12)	2 (3)	0.16
*Weak laryngeal function	9 (56)	19 (26)	0.043

Stage 2: 60 ml of water

Not able to finish	1 (11)	8 (13)	1.0
Cough with/after swallow	1 (12)	8 (13)	1.0
Stridor during/after swallow	1 (12)	10 (16)	0.22
Weak laryngeal function	2 (25)	10 (17)	0.62
*Aspiration present	14 (70)	25 (34)	0.008

Table 3: Value of individual elements from the Doctors' bedside assessment in predicting aspiration. Items marked * were significant at the 5% level.

(not all patients underwent every assessment, which will result in different percentages)

ASSESSMENT	SLT ASPIRATION ON VF		p - value
	Yes n = 19 (%)	No n = 64 (%)	
*Abnormal head posture	7 (39)	8 (13)	0.034
*Abnormal trunk control	12 (67)	20 (32)	0.016
*Drowsy	7 (39)	3 (5)	0.0007
*Abnormal communication	13 (72)	24 (38)	0.022
Abnormal respiration	5 (28)	10 (16)	0.30
Lip closure:			
*Abnormal at rest	12 (67)	19 (30)	0.011
Abnormal eating/drinking	10 (56)	22 (35)	0.19
Abnormal during speech	6 (40)	18 (29)	0.54
Tongue movements:			
Abnormal protrusion	8 (57)	17 (27)	0.056
*Abnormal lateral movement	10 (67)	19 (31)	0.022
*Abnormal velar movement	7 (50)	12 (21)	0.041
Gag reflex:			
Affected side absent	10 (71)	21 (41)	0.09
Normal side absent	9 (64)	19 (36)	0.12
Palatal function:			
Abnormal speech	3 (20)	5 (9)	0.35
Nasal regurgitation	0 (0)	1 (2)	-
Tongue function:			
Abnormal eating (tongue)	5 (36)	15 (24)	0.50
*Abnormal drinking (tongue)	6 (38)	6 (10)	0.012
Drooling	4 (24)	8 (13)	0.27
Abnormal jaw movement	2 (15)	8 (14)	1.0
FLUID Laryngeal function:			
*Abnormal voluntary cough	10 (62)	16 (27)	0.017
Abnormal phonation pre swallow	7 (44)	14 (23)	0.12
Abnormal involuntary cough	13 (76)	53 (85)	0.46
Abnormal phonation post swallow	8 (47)	15 (25)	0.15
*Abnormal swallow reflex	9 (53)	14 (22)	0.031

Pharyngeal function:

Regurgitation	1 (6)	1 (2)	0.38
*Pooling in pharynx	7 (41)	9 (14)	0.035
*>1 swallow to clear	4 (24)	3 (5)	0.034

Aspiration:

*Tracheal penetration	7 (41)	9 (14)	0.035
*Laryngeal penetration	9 (53)	14 (22)	0.031
*Fluids	9 (47)	9 (14)	0.004
Soft solids	3 (23)	2 (5)	0.093

Table 4: **Value of individual elements from the Speech and Language Therapists' bedside assessment in predicting aspiration.**
Items marked * were significant at the 5% level.

(not all patients underwent every part of the assessment, this results in differing percentages)

DISCUSSION

Dysphagia is common following a stroke (Gordon et al., 1987). In this study over half of our patients were identified as having an unsafe swallow, following DOC assessment (31% following SLT) , which is comparable to the 45% reported by Gordon et al. (1987), 28% in the BEST study (1989) and 42% in the paper by Kidd et al. (1993). The next question is how these bedside assessments relate to the presence of aspiration on VF, but only the study by Kidd et al. (1993) has previously done this during the acute phase of stroke. Like them, this study found that a significant number of patients were aspirating, though the proportion was lower (21% v 42%).

If aspiration is the important clinical abnormality, it follows that the value of the bedside instrument is in screening for this, though a secondary consideration is identifying those patients who are definitely not aspirating and who can be allowed to eat and drink normally. Ideally, for a screening tool, a sensitivity approaching 100% is required to ensure that all true positive cases are identified. In this study, the sensitivity of the DOC bedside assessment was 70% and that of the SLT was 47%. Thus, neither assessments were satisfactory as a screening instrument with 30% of aspirators being missed by the doctor and 53% by the speech and language therapists. This high proportion of silent aspiration is much greater than the 20% reported by Kidd et al. (1993). Combining a weak voluntary cough and any impairment of

consciousness increased the sensitivity slightly to 75%, which is similar to that

attained by Linden et al. (1993) but still fell short of the 100% attained by

Kidd et al. (1993) where those subjects with normal pharyngeal sensation could

be reliably categorised as not aspirating and, as such, could be allowed to eat

and drink normally. In this study, pharyngeal sensation was not recorded and

none of the items in the detailed bedside assessment were of similar value. The

presence of the gag reflex seems to be the most common proxy for a safe

swallow, yet these results and those of others (Horner and Massey, 1988,

1991) suggest that it is of no value in the assessment of dysphagia. This is

reinforced by recent work showing that many healthy elderly and a significant

minority of young people have an absent gag (Davies et al., 1995).

This study hinges on the role of videofluoroscopy as the 'Gold standard'. At

present it is accepted as the 'Gold standard' for the assessment of swallowing

and the diagnosis of aspiration, but it must be accepted to have limitations. The

mean filming time for a modified barium swallow is 3 minutes (Wright,

1991). Consequently it only reflects function over one brief period of time,

with the patient optimally seated and may not reflect function outside the

radiology department (Stanners et al., 1993). In this study, there were a

significant number of patients who were identified as having an unsafe

swallow, a proportion of these may have had a false negative videofluoroscopy

examination. Furthermore, swallowing, like many other impairments, is in a

state of flux following a stroke and the proximity of the bedside assessment and radiographic evaluation may be crucial. The usefulness of videofluoroscopy is also dependent on the accuracy of the report and the experience of the radiologist reporting. In this study two radiologists reported a proportion of the videofluoroscopy examinations, but only agreed in 76% of occasions. This further casts doubt onto the claim of videofluoroscopy to be a 'gold standard', this may be improved by the use of a check list so that examination and reporting of the video could be standardised. Further research needs to be done to look at this question.

This study confirms that bedside assessment of swallowing following acute stroke is of limited value in identifying those patients who aspirate on videofluroscopy examination. If this was the most important abnormality to identify then it would have major radiological resource implications. However it may be that the presence of an unsafe swallow that is the most important problem, though even here the lack of agreement between specialists is a cause for concern. This may be improved by simplifying the screening instrument and the combination of conscious level, voluntary cough and coughing on 5mls of water may allow those patients not at risk to be identified by admitting medical staff and only those at risk being placed nil by mouth. The scarce expertise of the SLT concentrated on the management (The College of Speech and Language Therapists 1990) of these at risk patients.

CHAPTER 10

ASPIRATION FOLLOWING A STROKE: IS IT RELATED TO THE SIDE OF THE STROKE?

ASPIRATION FOLLOWING A STROKE: IS IT RELATED TO THE SIDE OF THE STROKE?

AIM

To examine the relationship between the presence of aspiration, as demonstrated on videofluoroscopy, following acute stroke and the site, side and pathology of the stroke.

METHODS

Patient recruitment and eligibility for the study has been described earlier.

An unenhanced Computed tomograph (CT) scan of the brain was performed. If the patient was too unwell to move from the medical ward a CT scan was not performed. All CT scans were reported by consultant radiological staff at the Withington Hospital.

Patients underwent videofluoroscopy, modified from that of Logemann (1983) both within 3 days of admission and again at one month, where feasible, as described earlier.

STATISTICS

All data were analysed using Minitab V8. Chi square analysis was performed to look at the differences between relevant groups.

RESULTS

One hundred and thirteen (76%) of the 149 patients had CT scan. The CT scans were performed within a median time of 3 (interquartile range 2-4) days of admission. Eighty two (73%) had a recent lesion demonstrated on CT scan, 37 (33%) had left sided lesions, 40 (36%) right, 2 (2%) bilateral, and 2 (2%) were brain stem lesions. In one case there was no report of the side of the lesion, and the films could not be traced. Fifty nine (53%) patients had an infarct on CT and 13 (12.0%) had a haemorrhage (a further three patients were found to have had a haemorrhage at necropsy). Nine (8%) patients had had an haemorrhagic infarct.

Eighty seven patients (50% female) had both a CT scan and underwent videofluoroscopy. The median age was 79 years (range 40-93). Of these, forty four (52%) of patients had suffered a cerebral infarction, 8 (9%) a haemorrhagic infarction, 11 (10%) a primary intracerebral bleed. In a significant proportion (21 - 24%) a lesion was not evident on their scans. Three CT reports did not document the side of the lesion, and the films could not be traced for reporting. Thirty four (52%) had left sided lesions, and 23

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(36%) evidence of previous lesions.

Videofluoroscopy was conducted with in a median of 2 days (interquartile range 1-4) after admission, 17 (19.5%) of whom were aspirating. There were no significant relationships between the side of the neurological lesion and the presence of aspiration (ASP) on videofluoroscopy ($\chi^2(1)=1.536$, p>0.5) (table 1). Most lesions were in the parietal lobe, but there was no significant difference between this (6 ASP, 35 N) and all other areas combined (7 ASP, 15 N) ($\chi^2(1)= 2.58$, p>0.1). In 21 instances no lesion was identified on CT, 4 patients were aspirating. A relationship between site and side of the lesion and ASP could not be demonstrated when first ever strokes (66/87) were assessed alone (5 aspirators - left sided lesion, 5 aspirators - right sided lesion).

Videofluoroscopy was repeated in 69 patients, at a median time of 29 days (interquartile range 26-45, range 22-85 days) after stroke onset. Eighteen patients (5 ASP, 13 N) were withdrawn between the 2 assessments due to recurrent strokes, needing surgery or death. Nine (13.5%) patients had aspiration documented on the second videofluoroscopy examination. Of those where a lesion was identified on CT and were aspirating, 7 had right hemisphere lesions, (table 1), $\chi^2(1)=8.736$, p<0.01. In patients without a prior history of stroke and only a single lesion on CT scan (45/69), 5 were

aspirating on videofluoroscopy, four were right hemisphere strokes. Most

lesions were in the parietal lobe, but this was not statistically significant

$\chi^2(1)=0.056$, p>0.5.

CT Scan	1st VF (n=87)		2nd VF (n=69)	
Side / site of lesion	ASP	N	ASP	N
No Lesion seen	4	17	2	13
Left	9[a]	25	0[b]	27
Right	4	25	7	19
Parietal lobe	6[c]	35	5[c]	31
Other lobes	7	15	2	15
Not Recorded	0	3	0	1

a: p>0.1   b: p<0.01   c: p>0.5

VF: Videofluoroscopy
ASP: Aspiration
N: No aspiration

Table 1.   Side and site of stroke compared to the presence or absence of aspiration.

DISCUSSION

Over recent years many workers (Robbins and Levine, 1988; Chen et al., 1990; Alberts et al., 1992; Robbins et al., 1993) have studied the relationship between the presence of aspiration on videofluoroscopy and site or side of the neurological lesion. Until quite recently the dogma was that a brainstem or bilateral cortical lesions were required to produce dysphagia. A review of the literature in 1973 (Meadows, 1973) suggested that damage to the right cortex would result in aspiration more frequently than damage to the left. Others (Robbins and Levine, 1988; Robbins et al., 1993) have suggested that different phases of the swallow may be represented in different hemispheres, the oral phase on the left and the pharyngeal on the right. Robbins et al. (1993) more recently reported that all stroke patients, 21 days post stroke, have swallowing difficulties, but that those with right sided lesions were more likely to aspirate.

The only workers that have consistently claimed sidedness for swallowing are Robbins and co workers (Robbins and Levine, 1988; Robbins et al., 1993). Certainly their studies have used patients with first ever strokes and discrete lesions. Other studies have used a more heterogeneous population, and have often studied patients selected due to the presence of persistent swallowing difficulties (Horner and Massey, 1988; Splaingard et al., 1988; Alberts et al., 1992). This is the first time patients have been studied repeatedly and

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prospectively.

These results have not been able to confirm the presence of an association between aspiration and stroke side when patients are studied early (within the first week). But when the patients were studied at a median time of 29 days following their stroke, there appeared to be an association between aspiration and the side of the stroke.

Why does there appear to be sidedness at the second assessment time and not initially? At the time of the initial assessment more patients were examined, and examined earlier. Many of those demonstrated as aspirating may have had a transient problem, others may not have been aspirating on that day but were on the next. As the neurological lesion settles down, for example resolution of oedema, many problems resolve, some are unmasked and others persist. The other possibility is that swallowing ability following stroke fluctuates (Logemann, 1983).

Videofluoroscopy is also an artificial scenario for the assessment of the swallow. It only allows assessment of the patient's swallow for a small period of time whilst the patient is sitting in the ideal position.

It may be due to the fact that patients with more severe strokes died before the

second assessment and others may well have become more alert. The other possibility is that swallowing ability following stroke fluctuates (Alberts et al., 1992). Some patients who had problems during the early stages may have been wrongly classified, others with problems at the second assessment may have had silent problems. Another argument may be that patients with right hemisphere damage have persistent swallowing problems, whilst left hemisphere lesions lead to transient swallowing difficulties.

This study may have found a difference that does not exist or missed the presence of 'sidedness' during the acute phase because of the timing of the CT scan. Early CT scans will miss a significant proportion of infarcts. There were a significant proportion of scans (21 - 24%) revealing no lesion. It is recognised that CT is not very sensitive in detecting small infarcts and those in the posterior fossa or brainstem. Consequently small lesions in either of these areas may explain the difficulty in detecting a discrete area of the cortex that is responsible for aspiration.

All of these studies have examined the relationship between the presence of a recent stroke as demonstrated radiologically and the presence of aspiration. None have examined the relationship between the side of clinical weakness and the presence of aspiration, or even dysphagia. Unpublished data would suggest that an association does not exist (Queen Mary's NHS Trust Stroke Register).

This would suggest that the studies, including this study, that have relied on radiological data have found an association that may not exist in an unselected stroke population.

In conclusion, these results would suggest that there tends to be an association between the side of the stroke and aspiration that persists. The evidence for this is not completely convincing due to small numbers, and the reliance on radiological data. A different approach to this problem may be to look at the effect of magnetic stimulation of the brain (Hamdy et al., 1996). It has not been possible to demonstrate any relationship between the type of pathology and the presence of aspiration. Further studies are required to look specifically the neuroanatomy of swallowing, and the relationship between the side of the hemiparesis and aspiration and/ or dysphagia.

CHAPTER 11

NATURAL HISTORY

NATURAL HISTORY

AIMS

1.To examine the ability to swallow safely, with time, following acute stroke: does a safe swallow always remain safe?

2. To examine the changes with time regarding the presence of aspiration, silent aspiration and laryngeal penetration, following acute stroke.

METHODS

Recruitment and eligibility of patients for the study were described earlier.

CLINICAL ASSESSMENTS

On admission all patients had their diagnosis reviewed and their ability to swallow assessed by the same doctor (DOC), and then daily for 4 days, and on day 7, 28 and at 6 months. During the first week the same speech and language therapist (SLT) assessed the swallow up to 3 times, then again at day 28 and at 6 months. Both DOC and SLT used the standardised BSAs described earlier (see appendix).

Within 3 days of admission all patients underwent videofluoroscopic examination, which was repeated, where possible at day 28. The examination

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undertaken was described earlier. Patients did not undergo videofluoroscopy examination if their conscious level was reduced (Glasgow coma score < 10) or if they were medically unfit.

This study only documents the swallowing ability (presence or absence of dysphagia) of those patients able to have their swallow assessed, over a period of 6 months.

STATISTICS

Formal statistics were not conducted as this was an observational study.

RESULTS

One hundred and twenty one patients were assessed excluding those who remained unconscious (28 patients) through out the study and eventually died. The numbers at each assessment point differ due to deaths (25 patients, 18 stroke related) following stroke and withdrawals (14 recurrent stroke, 2 carotid endarterectomy, 3 hip surgery, 3 dementia, 1 at patients request).

The median time from stroke onset to assessment by DOC was 14 hours (range 0.5-50). At which time 61 of the 121 patients (50%) were considered to have dysphagia. Between day 0 and day 7, the number with dysphagia reduced from 50% to 27%. By day 28, 18 patients (17%) were still assessed as having dysphagia, 13 (12%) of these had dysphagia throughout, but 5 (5%) had been previously assessed as safe. Seventy three patients remained in the study at 6 months, of these 8 (11%) had an unsafe swallow, 6 (8%) had problems persisting from the time of the assessment on day 28, 2 (3%) had problems not previously identified. For the full results see table 1.

The speech and language therapist examined 104 (87%) of the patients. The initial assessment took place with a median time of 1 (range 0-5) day following stroke onset. At this point, which was within a median time of 24 hours (interquartile range 1-2 days) of the videofluoroscopy, 32 (31%) were considered to have dysphagia. By day 7, 22 patients (33%) were considered

Day	N	Unsafe Swallow(%)	New	Persistent
0	121	61(50)	--	---
1	113	33(39)	4	29
2	111	35(31)	6	28
3	105	43(39)	6	35
7	110	28(27)	1	27
28	105	18(17)	5	13
180	73	8(11)	2	6

Table 1. Number of patients with an unsafe swallow at each assessment point as assessed by the doctor (DOC).

to have dysphagia, of these only 11 had previously been identified. At day 28, only 2 (3%) were considered to be unsafe, these were persistent problems. By 6 months one of these had resolved, but another patient was considered to be at risk, having been assessed as safe at day 28. The full results are documented in table 2.

Analysis was carried out to determine whether any clinical markers predicted whether a patient would have a clinically unsafe swallow at day 28. Gender, side of weakness, presence of neglect, incontinence, abbreviated mental test score, Barthel ADL (BAL) and sidedness of stroke were examined. No significant difference in any of these parameters were noted between those with new or persistent swallowing problems. This may be a real effect, but numbers were small and hence a real difference may have been missed. Full details of the results are given in table 3.

A total of 95 (78.5%) of patients underwent videofluoroscopy within a median time of 2 days (range 0-9, interquartile range 1-4) following admission. The remaining patients (26) were considered to be too ill to be moved from the ward. Eighty one (88%) VF examinations were repeated at a median of 29 days after the stroke onset, (range 22-85 Interquartile range 27-33).

Twenty one (22%) patients were noted to be aspirating on the initial

Day	N	Unsafe Swallow(%)	New	Persistent
3	104	32(31)	---	----
7	66	22(33)	11	11
28	62	2(3)	0	2
180	64	2(3)	1	1

Table 2: Number of patients with an unsafe swallow at each assessment point as assessed by the Speech and Language Therapist (SLT).

SWALLOWING DIFFICULTIES AT DAY 28

	NEW	PERSISTENT	P
	N=5	N=13	
Age (mean years)	76.8(60-82)	72.5(60-85)	NS
Gender (M:F)	1:4	9:4	NS
Median MTS	6 (3-7)	5(4-10)	NS
Median Barthel	10(0-17)	5(0-17)	NS
Presence of neglect	2/5	1/13	NS
Incontinence of Urine	2/5	1/13	NS

Table 3:   Differences between patients with a persistently unsafe swallow at day 28 compared to those with a new swallowing difficulty.

assessment, 44 (46%) had supraglottic penetration (entry of barium into the larynx, but remained above the true vocal cords) and 30 (32%) had neither. At the second videofluoroscopy examination 12 (15%) were still aspirating, of these only 4 (5%) had been previously identified. Forty patients (49%) had evidence of supraglottic penetration, 24 of whom had persistent problems. Twenty nine (36%) had no swallowing problems. The full results are shown in table 4.

At the time of the second videofluoroscopy the SLT did not identify any patients who were aspirating on videofluoroscopy (12 patients), DOC detected 2 patients.

There was no clinical difference between those aspirating at the time of the second assessment compared to the rest of the study cohort.

Assessment I		Assessment II			
		Aspiration	S. Penetration	Neither	No VF
Aspiration	21	4	8	3	6
S. Penetration	44	4	24	10	6
Neither	30	2	6	12	10
No VF	8	2	2	4	0
Totals	103	12	40	29	22

S. Penetration: Supraglottic Penetration
VF: Videofluoroscopy

Table 4: Videofluoroscopic findings at assessment I (median time 2 days) and II (median time 29 days).

DISCUSSION

This study has not only confirmed that swallowing problems following stroke are common and that 22% of patients are aspirating (when examined by videofluoroscopy), but that swallowing difficulties apparently fluctuate with time; some patients being identified as having problems much later in the course of their illness. One reason for this may be lack of precision of the bedside assessment with patient categorisation varying between the DOC and the SLT. The results suggest that DOC was over cautious, tending to over categorise patients as unsafe. Furthermore, most patients with aspiration on videofluoroscopy were not correctly classified at the bedside, though earlier it was shown that it is the clinical not the radiological assessment which has the strongest association with outcome and complications.

Teasell et al. (1994) in a retrospective series of 52 patients who had undergone repeated videofluoroscopy for clinical indications, reported that 11% were still aspirating at a mean time of 83 days post stroke onset. This study showed that 12/81 (15%) were aspirating at a median time of 29 days post stroke, but that only 4 of these were also recorded to be aspirating at a median time of 2 days post stroke onset. It is clear both from these results and those of Teasell et al. (1994) that problems of aspiration persist for a long time. Also that aspiration may occur late into the course of the stroke.

It is clear that some of the changes were due to false positive and false negative errors in classification, but is known that the neurological deficit does vary in the first week, often worsening (Fullerton, 1991) before it improves. The changes in swallowing are probably a reflection of this and are being detected by careful repeated assessment by a trained observer.

An alternative argument would be that there has been no change in the patient's ability to sallow, but that the person assessing his/her ability to swallow has come to a different conclusion given the same information, i.e there may be a test-retest reliability problem with the BSA. This may be particularly marked where the assessor has undergone formal training and a clinical judgement biases the findings of the BSA. This would need to be examined in more detail.

This study has quantified the number of patients with persistent problems in the late phase of stroke with around 1 in 10 people still having detectable abnormalilities. A proportion of this 10% could be false positive given that the SLT identified a much smaller number, but on videofluoroscopy at one month 15% of patients were shown to aspirate, which is not a normal finding (Blonsky et al., 1975; Tracy et al., 1989). The patients identified late could have been missed earlier (false negatives), but, although a number of people were excluded with recurrent stroke, it is possible that cerebrovascular disease

accounted for late dysphagia.

This is the first study to examine the evolution of dysphagia over a long period following acute stroke using both videofluoroscopy and clinical assessments. It has been confirmed that in the majority of patients, dysphagia resolves, but a significant proportion appear to continue to have clinical problems or aspiration. Other patients apparently develop new problems. Further investigation is required to examine the test-retest variability of the BSAs and the best management of dysphagia, not only in the acute phase but also for the those with ongoing difficulties (Park and O'Neill, 1994).

CHAPTER 12

NUTRITION and HYDRATION

NUTRITION and HYDRATION

AIM

To examine the relationship between the presence of dysphagia and hydration and nutritional status following acute stroke. Does the presence of dysphagia matter?

METHODS

Patient recruitment and eligibility for the study was documented earlier.

Patients underwent videofluoroscopy as described earlier.

Nutritional status

Clinical, biochemical and haematological parameters were used to assess each subject's nutritional status. These were examined at four assessment points (Days 0 (admission), 7, 28 and 180 (six months). The groups referred to here are the same as those documented earlier.

Clinical assessment

Skinfold thickness (Yearick, 1978) was measured at four sites to provide a good overall picture of nutritional status. The sites measured were subscapular, triceps (midway between olecranon and acromium, on the non paralysed arm), mid arm circumference, suprailiac, and periumbilical. Skinfold thickness was

measured by raising a fold of skin between the thumb and forefinger, then measuring using skinfold callipers (Crymch, Wales). Arm muscle circumference (AMC) was calculated from the mid arm circumference (MAC) and triceps skinfold thickness (tsf) using the following equation AMC = MAC - (tsf x π / 10) (McEvoy and James, 1982). The calculated AMC is a measure of lean body mass. Anthropometric measurements have a large error component, both between and within observers. In an attempt to minimize this each measurement was done three times and the mean taken. Also, all measurements were performed by a single assessor.

Body mass index was also assessed being calculated from height (length) in metres and weight in kilograms (Elia and Jebb, 1990). Subjects were not weighed if they were considered too unwell.

Biochemical parameters

Biochemical parameters were used to assess the nutritional status. These included serum albumen (Axelsson et al., 1988), globulin and total protein levels. Albumen is a recognised marker of nutrition, but its serum concentration can be affected by severe acute illness. As albumen has a relatively long half life it is a useful marker of long term nutrition. The plasma concentration of albumen is affected by body posture. Therefore, where ever

possible, all samples were taken with the patient rested and sitting down.

Haematological parameters

The following parameters were assessed: haemoglobin (Chandra, 1990) concentration, lymphocyte count (Chandra, 1990) and haematocrit (Ciocon et al., 1988).

Hydration

Hydration was assessed both biochemically and haematologically. Hydration was assessed at the four assessment points.

Haemoglobin, haematocrit (Gordon et al., 1987); serum sodium, serum urea (Gordon et al., 1987; Barer, 1989), and osmolality (O'Neill et al., 1989), (using depression of freezing point, Camlabs, Cambridge, UK) were all assayed.

All the biochemical (except osmolality) and haematological assays were performed in the laboratories of the University Hospital of South Manchester. Osmolality was assayed by the author.

STATISTICS

The change in these indices have been analysed within groups. Changes in the nutritional indices have been taken with reference to the admission value. Paired t-tests were then performed within groups.

It has not been possible to analyse the results allowing for stroke severity due to the resulting small numbers within each group.

RESULTS

The following results are based on 121 patients who had their swallow assessed by DOC, and 104 patients assessed by SLT. Ninety four (78%) underwent videofluoroscopy examination within a median time of 2 days (interquartile range 1-4). Twenty patients aspirated and 74 did not. SLT assessed the ability to swallow within a median time 1 day (interquartile range 0-1 days) of the videofluoroscopy. DOC assessed patients within a median time of 0 (same) day (interquartile rang 0-1 days) of videofluoroscopy.

NUTRITIONAL MARKERS

There were significant changes in some of these markers over the six months of follow up. This was more marked in those with swallowing difficulties.

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Suggesting, therefore, that nutritional decline is more likely to be associated with the presence of an unsafe swallow. This decline was also noted for those aspirating on videofluoroscopy.

Anthropometric indices

Weight and BMI

There was no change in the weight of the stroke patients over the study period. When patients were divided by their swallowing ability, those with a safe swallow increased their weight, whereas those with unsafe swallows showed no significant changes in their weight. There was no significant changes in BMI either in the whole study group or any sub group.

DOC

Patients assessed as safe by DOC showed an increase in mean weight at day 7 (1.16 kg, p=0.006), which was present at day 28 (1.64 kg, p=0.014). This did not continue over the ensuing 6 months of the study. There were no concomitant change in BMI. Those patients (60, 50%) assessed as having dysphagia did not lose or gain weight during the six months of the study.

SLT

Where the SLT had assessed a patients swallow as safe (72/104, 69%), there was a small but statistically significant increase in mean weight over the first week (0.75 kg, p=0.045)and month (1.54 kg, p=0.005). Those assessed as having dysphagia did not show any changes in weight during the time of the study.

Videofluoroscopy

Those patients without aspiration on videofluoroscopy, gradually increased their weight to a peak at day 28 (70.3 kg (+1.41kg), p=0.03). In those where aspiration was documented (20/94, 21%), there were no significant changes in weight.

Skinfold thickness

All strokes

There were no significant changes over time, but MAC, AMC and suprailiac skinfold thickness declined over the first month, picking up over the ensuing 5 months to that at the time of admission.

DOC

A safe swallow was associated with significant changes at two sites at different time periods. There was an increase in the mean periumbilical

skinfold (1.5 mm, p=0.018) and subscapular skinfold (1.0 mm, p=0.047) by day 7 and an increase in the mean suprailiac skinfold by day 28 (1.4 mm, p=0.032). There were fluctuations at other sites during the 6 months of the study, but none reached statistical significance (table 1).

In those patients with dysphagia nutritional indices deteriorated over the first month: tsf (-3.5 mm, p=0.001), MAC (-12.4 mm, p=0.007) and AMC (-10.9 mm, p=0.013) by day 28. The decrease in these had begun by day 7 (see table 2) but did not reach significance at this time. Although there were improvements by 6 months, AMC (-12.9 mm, p=0.011) and MAC (-19.4 mm, p=0.013) had deteriorated further.

SLT

In patients with out dysphagia, there were significant changes in skinfold thickness at different sites during the study: periumbilical (+1.3 mm, p=0.012) at day 7, suprailiac at day 28 (+1.4 mm, p=0.013), and a decrease of the tsf by day 28 (-1.2 mm, p=0.044) (table 3).

Nutritional indices deteriorated over the first month in those patients with dysphagia (table 4): with statistical significance being reached at tsf (-3.2 mm, p=0.007) and periumbilical (-2.7 mm, p=0.027) skinfold sites. Significant reductions were noted also in the MAC (-15.7 mm, p=0.005) and

DAY	0	7	28	180
n	41	35	31	31

Skinfold (mm)		mean difference (SEM)		
Triceps	14.9(1.2)	-0.3(0.4)	-0.01(0.6)	-0.3(0.6)
Mid arm circumference	288.5(7.9)	-1.0(1.7)	-1.7(3.2)	0.6(4.7)
Arm muscle circumference	283.8(7.5)	-3.0(2.7)	-1.8(3.1)	1.4(4.5)
Subscapular	14.6(1.2)	1.0(0.5)[a]	0.2(0.8)	-0.5(0.6)
Suprailiac	12.9(1.5)	0.7(0.7)	1.4(0.6)[b]	1.7(1.1)
Periumbilical	22.5(1.8)	1.5(0.6)[c]	-0.3(0.9)	-0.6(1.6)

a: p=0.047   b: p=0.032   c: p=0.018

Paired t-test: Differences are difference from baseline, Day 0

Table   1:   Patients assessed as having a safe swallow by the doctor.

DAY	0	7	28	180
n	28	26	21	19
Skinfold (mm)		mean difference (SEM)		

Triceps	16.7(1.2)	-1.2(0.8)	-3.5(0.9)[a]	-2.4(1.3)
Mid arm circumference	300.9(7.9)	-5.5(3.8)	-12.4(4.1)[b]	-19.4(7.0)[c]
Arm muscle circumference	295.3(7.6)	-9.0(5.8)	-10.9(4.6)[c]	-12.9(4.5)[d]
Subscapular	15.9(1.3)	0.0(0.8)	-1.2(0.8)	-1.0(1.4)
Suprailiac	14.3(1.2)	-1.1(0.9)	-0.5(1.0)	-1.4(1.7)
Periumbilical	24.5(1.7)	0.4(0.9)	-2.1(1.1)	-2.4(1.5)

a: p=0.001   b: p=0.007   c: p=0.013   d: p=0.011

Paired t-test: Differences are difference from baseline, Day 0

Table   2:   Patients assessed as having an unsafe swallow (dysphagia) by the doctor.

DAY	0	7	28	180
n	60	52	45	43
Skinfold (mm)		mean difference (SEM)		
Triceps	15.3(0.9)	-0.7(0.4)	-1.2(0.6)[a]	-0.8(0.7)
Mid arm circumference	286.3(5.8)	-2.0(1.8)	-0.8(3.2)	-1.0(3.7)
Arm muscle circumference	281.3(5.5)	-5.1(3.2)	-0.2(3.2)	-0.2(3.6)
Subscapular	14.7(0.9)	0.5(0.3)	-0.4(0.6)	-0.5(0.6)
Suprailiac	12.8(1.1)	1.0(0.6)	1.4(0.5)[b]	1.1(1.1)
Periumbilical	23.4(1.4)	1.3(0.5)[c]	-1.1(0.8)	-1.9(1.3)

a: p=0.044   b: p=0.013   c: p=0.012

Paired t-test: Differences are difference from baseline, Day 0

Table 3: Patients assessed as having a safe swallow by the speech and language therapist.

DAY	0	7	28	180
n	28	21	15	12
Skinfold (mm)		**mean difference (SEM)**		
Triceps	16.0(1.4)	-1.5(0.8)	-3.2(1.0)[a]	-3.2(1.3)[b]
Mid arm circumference	302.5(8.7)	-5.2(4.3)	-15.7(4.8)[c]	-20.4(11.1)
Arm muscle circumference	297.5(8.4)	-4.6(4.3)	-14.7(4.7)[a]	-11.1(6.9)
Subscapular	15.1(1.9)	0.2(0.9)	-1.4(1.0)	-0.5(1.9)
Suprailiac	14.0(1.5)	-1.6(1.1)	-1.0(1.1)	-2.0(1.8)
Periumbilical	23.4(1.4)	-0.6(1.4)	-2.7(1.1)[d]	-1.8(1.7)

a: p=0.007   b: p=0.038   c: p=0.005   d: p=0.027

Paired t-test: Differences are difference from baseline, Day 0

Table   4:   Patients assessed as having an unsafe swallow (dysphagia) by the speech and language therapist.

AMC (-14.7 mm, p=0.007) measurements. There were improvements in some but not all indices over the ensuing months, with values remaining lower at six months than at base line (day 0), though only the triceps skinfold thickness achieved significance (-3.2 mm, p=0.038).

Videofluoroscopy

In those patients without aspiration, there was a trend for nutritional indices to deteriorate, significance was only reached at day 28 at the periumbilical site (-2.0 mm, p=0.027) (see table 5).

In those patients where aspiration had been documented on videofluoroscopy, there was a deterioration in all indices over the first month (eg tsf, -3.5 mm, p=0.048; MAC, -11.8 mm, p=0.03; AMC, -10.9 mm, p=0.032), further details are included in table 6.

Laboratory indices

Biochemical indices

The serum concentration of albumen fell over the first week in all stroke patients studied (table 7). This was mirrored by an increase in the concentration of globulins. These levels had often increased at six months, towards baseline, whether swallowing problems were present or not.

DAY	0	7	28	180
n	64	57	47	40
Skinfold (mm)		mean difference (SEM)		
Triceps	15.4(1.0)	-0.7(0.4)	-1.0(0.5)	-1.2(0.7)
Mid arm circumference	294.4(5.9)	-1.1(1.6)	-4.7(3.0)	-4.2(3.9)
Arm muscle circumference	289.3(5.6)	-2.2(3.4)	-4.2(3.0)	-3.0(3.8)
Subscapular	15.1(0.9)	0.7(0.6)	-0.7(0.6)	-0.4(0.8)
Suprailiac	13.2(1.1)	0.1(0.6)	0.5(0.3)	0.9(1.1)
Periumbilical	23.8(1.4)	0.4(0.6)	-2.0(0.9)[a]	-1.3(1.4)

a: p=0.027

Paired t-test: Differences are difference from baseline, Day 0

Table 5:   Patients observed not to aspirate on videofluoroscopy.

DAY	0	7	28	180
n	19	16	12	10
Skinfold(mm)		mean difference (SEM)		
Triceps	14.4(1.1)	-0.5(0.9)	-3.5(1.5)[a]	-0.7(1.8)
Mid arm circumference	278.9(9.3)	-7.4(5.3)	-11.8(4.8)[b]	-18.4(13.6)
Arm muscle circumference	274.4(9.1)	-7.1(5.3)	-10.9(4.4)[c]	-8.2(8.2)
Subscapular	13.3(1.6)	0.5(0.8)	0.6(1.1)	-0.4(1.3)
Suprailiac	11.9(1.2)	0.4(1.1)	0.7(1.4)	-0.9(2.0)
Periumbilical	20.6(1.8)	1.7(0.7)[d]	-1.1(0.8)	-1.0(1.6)

a: p=0.048   b: p=0.030   c: p=0.032   d: p=0.039

Paired t-test: Differences are difference from baseline, Day 0

Table   6:   Patients observed to aspirate on videofluoroscopy.

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ALBUMEN

DAY	0	7	28	180
		mean difference g/l (SEM)		

DOC

	0	7	28	180
Safe	42.0(0.7)	-2.2(0.8)[a]	-0.8(0.7)[b]	-1.2(0.7)
Unsafe	41.0(0.7)	-2.6(0.8)[c]	-2.5(0.9)[d]	-0.7(0.7)

SLT

	0	7	28	180
Safe	41.4(0.6)	-2.1(0.5)[e]	-0.8(0.7)	-0.5(0.6)
Unsafe	40.3(1.0)	-3.9(1.3)[c]	-4.3(1.0)[e]	-3.5(0.9)[f]

VIDEOFLUOROSCOPY

	0	7	28	180
No Aspiration	41.5(0.6)	-2.2(0.6)[g]	-0.9(0.7)	-1.3(0.6)[d]
Aspiration	40.9(0.9)	-3.2(1.1)[b]	-3.2(1.5)[b]	0.0(0.6)

a: p=0.007 b: p<0.05 c: p=0.006 d: p<0.02 e: p<0.0001

f: p=0.002 g: p<0.001

DOC: Doctor
SLT: Speech and Language Therapist.

Paired t-test: Differences are difference from baseline, Day 0

Table 7: Changes in mean albumen concentration with time during the period of the study.

DOC

In those patients assessed as having a safe swallow, there was a significant reduction in the mean serum albumen by day 7 (-2.2 g/l, p=0.007). This situation had improved by day 28, but levels were still reduced from admission (-0.8 g/l, p<0.05). By the end of the study there was no significant difference from the albumen concentration found at admission (table 7).

Dysphagia was similarly associated with a decline in serum albumen concentrations, but this was more marked (-2.6 g/l, p=0.006) and more prolonged (day 28: -2.5 g/l, p<0.02). By 6 months the mean albumen concentration had returned to that of admission. There was an associated rise in globulins (2.0 g/l, p=0.036), which persisted at 6 months (4 g/l, p=0.015).

SLT

The serum albumen concentration was significantly reduced at day 7 in those patients with a safe swallow on bedside assessment (-2.1 g/l, p<0.0001). This decline was not significant at day 28, and albumen levels had returned to those on admission by six months post stroke (table 7).

The presence of dysphagia was associated with significant reduction in albumen concentration at all assessment points of the study (eg -3.9 g/l, p=0.006 at day 7). Globulins were increased at six months (4.2 g/l

p=0.031), though this increase was not mirrored earlier on in the study.

Videofluoroscopy

The absence of aspiration was associated with a small but significant decline in the serum albumen concentration (day 7: -2.2 g/l, p<0.001). This deterioration had not been fully reversed by six months after the stroke (-1.3 g/l, p<0.02). The concentration of globulins was observed to rise over the study period (3.0 g/l, p=0.008).

The presence of aspiration during the videofluoroscopy examination was associated with a decline in the serum albumen concentration (-3.2 g/l, p<0.05). Albumen concentrations at six months following stroke were not significantly lower than those on admission (table 7). There was a non significant rise in the globulin concentration during the study

Haematological indices

There were no changes in the haematological indices in the cohort as a whole. If the swallow had been considered safe at the bedside, no changes in haemoglobin , haematocrit or lymphocyte count were demonstrated during the period of the study.

Statistical significance was reached with reductions in haemoglobin concentration (-0.5 g/l, p=0.009) and haematocrit (-0.2, p=0.025) at one month, in those patients with dysphagia as assessed by the SLT.

There were no significant changes wether aspiration was present or absent on videofluoroscopy.

HYDRATION

Hydration was assessed by serum sodium, osmolality, and urea. There was no change in any of these indices over the study period. Patients with dysphagia were more likely to have parenteral fluids ($p < 0.001$), and these were continued for longer ($p < 0.0001$). These results are shown in more detail in table 8.

DAYS

	N	No (%) using parenteral fluids	Median	Interquartile Range	p value Length of use	Proportion use
DOC						
Safe	61	35(57)	2	1-3		
					<0.00001	<0.001
Unsafe	60	48(80)	6	3-8		
SLT						
Safe	72	50(69)	3	2-5		
					0.0002	>0.05
Unsafe	32	20(63)	8	5-8		
VF						
No ASP	74	48(67)	3	2-5		
					<0.001	>0.1
ASP	20	16(80)	8	2-8		

DOC: Doctor
SLT: Speech and Language Therapist
VF: Videofluoroscopy
ASP: Aspiration

Data was analysed using Chi square for proportions and Mann-Whitney U for median use

Table 8: Use of parenteral fluids during the initial phase of the study.

DISCUSSION

Previous studies have linked dysphagia following stroke to a worsening of nutritional status and dehydration (Gordon et al., 1987; Splaingard et al., 1988 Axelsson et al., 1989; Kidd et al., 1995), but none of these studies have attempted to differentiate the presence of dysphagia from the presence of aspiration as documented on videofluoroscopy.

Many workers have noted that patients admitted to hospital are malnourished (Bastow et al., 1983; Ek et al., 1990; Potter et al., 1995). Axelsson et al. (1988) confirmed that this was also the case in stroke patients. They also noted a gradual deterioration in nutritional status with time, but considered this to be a function of the stroke severity and patient's age rather than the ability to swallow. The presence of dysphagia may be a major cause in the deterioration of nutritional status, hydration. The presence of dysphagia may result in protein calorie malnutrition, a worsening of dysphagia and further deterioration (Veldee and Peth, 1992). This study has confirmed that the nutritional status of stroke patients declines with time, particularly if there are concomitant swallowing problems. This may be due to an increase in metabolic rate following the stroke similar to that demonstrated by Tuoho et al. (1990) in patients with a cerebral haemorrhage. Conversely, Weekes and Elia (1992) assessed 15 patients following their stroke (stroke pathology was not documented) and found no increase in resting energy expenditure, and suggest that only a moderate calorie intake would be required to maintain status quo. This

would contradict the argument put forward by Steffee in 1980, where he states that if nutritional input is not adequate following stress then protein is mobilised to provide glucose to obligate users (red blood cells, kidney and brain). Other factors may be the palatability of hospital food, or the inability of patients to take enough calories in before their food was removed by well meaning ward staff.

This deterioration continued until one month following the stroke, when after this time most of these markers began to improve. This improvement could have been artefactual, in that those with the worst indices in each group had died, or that as patients improved and their swallow resolved, the intake of food improved. The patients with the most marked deterioration were those with an unsafe swallow, whether aspiration had been found during videofluoroscopy or not. Those patients with silent aspiration tended to follow a similar pattern as those with a safe swallow. Recently Woo et al. (1994) have shown that despite supplementation of diets following chest infection it can take longer than six months to achieve the nutritional base line. Certainly those patients with an unsafe swallow had not reached base line at six months. Whether early feeding of stroke patients with oral or enteral supplements will improve outcome and shorten hospital stay, such as that shown by Bastow (1983) in patients with fractured neck of femur, is open to debate. It has been shown that nutritional deficiency results in immune deficits, and reduced immune response to infection (Chandra, 1990), further more a spiral decline may result with a worsening nutritional

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status (Steffee, 1980) and consequently increasing dysphagia (Veldee and Peth, 1992). Any effect that intervention may have will depend on whether the deterioration is due to the swallowing problems perse or as a function of the stroke.

Do patients with swallowing problems become dehydrated? Two prospective, clinically based studies (Gordon et al., 1987; Barer, 1989) have shown a tendency for this to be the case, and two studies (one prospective (Holas et al., 1994) and one retrospective series (Schmidt et al., 1994)) have not. Unfortunately, the study by Holas et al. was conducted 4 weeks after the stroke and is therefore not comparable to that of Gordon et al. (1987) or Barer (1989). This study did not find a deterioration in hydration following stroke. So why does dehydration not occur? In the early stages patients with swallowing difficulties will be hydrated with parenteral fluids, if anything there is a tendency to over hydrate them because of arginine vasopressin release (O'Neill et al., 1992).

In conclusion this study has shown that those patients with dysphagia are at risk of becoming under nourished. The study has also shown that information from videofluoroscopy adds little or no information to that determined clinically. It has not shown whether a poor nutritional status delays rehabilitation though this widely believed to be the case (Wanklyn et al., 1996). Further work is required to investigate the relationship between

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nutrition and rehabilitation. This work will need to determine whether

nutritional status is an independent factor affecting rehabilitation or whether

it is just a marker of stroke severity.

CHAPTER 13

OUTCOME

OUTCOME

AIMS

To examine the effect of dysphagia on outcome (chest infection, mortality [survival], length of hospital stay and place of discharge) following acute stroke.

METHODS

Recruitment and eligibility of patients for the study has been described earlier.

Outcomes examined were, in the short term the development of a chest infection with in the first 7 days, and in the longer term, mortality (or survival), functional outcome as assessed by the Barthel activities of daily living index (Mahoney and Barthel, 1965), length of hospital stay (LOS), and residence at the time of discharge.

Chest infection

The presence of chest infection was looked for in all subjects. Patients were examined on days 0 to 7 (pyrexia, tachypnoea >22 respirations per minute, inspiratory crackles, bronchial breathing and the use of antibiotics (Nahum et al., 1981; Bartlett and Gorbach, 1975; Sims, 1990)., diagnosis was based on the presence of two or more variables. The temperature was recorded twice

daily by the axillary route (under the unaffected arm, using an electronic 'B-D' thermometer [Boots plc.]). The presence or absence of a chest infection was determined by the author, who had also assessed the patient's ability to swallow safely.

A chest radiograph was performed on admission and at day 7 to look for radiographic evidence of infection and aspiration, this would have been performed earlier if clinically indicated. The chest radiographs were reported by staff in the X-ray department during normal reporting. The radiographs were reported blind to the results of the swallowing assessments, both clinical and radiological. The absence of any radiographic evidence of infection on the chest film did not preclude the diagnosis of chest infection. The chest radiograph was not required for the diagnosis of a chest infection.

Mortality

The occurrence of death was noted, either by review of the medical notes, hospital death certificates or on consultation with the Registrar of Births and Deaths.

Functional outcome

Functional outcome was assessed at 6 months using the Barthel activities of

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daily living index.

Place of residence and Length of stay

The total length of hospital admission was recorded. The place a patient was discharged to was noted. This was as either 'home' or 'Institution'.

STATISTICS

Significant associations between categorical outcome measures (chest infection, mortality and place of discharge) and the swallow assessment were determined using chi square or Fisher's Exact test.

For continuous outcome measures, length of stay had a log normal distribution and required $\log_e$ transformation prior to statistical analysis using two sample t-tests and one factor analysis of variance.

Barthel score had a non normal distribution and was assessed using Mann-Whitney and Kruskall Wallis tests. Multiple logistic regression analysis were carried out to determine the association between outcome and swallowing ability adjusting for other indicators of poor prognosis.

RESULTS

The results presented here refer to 121 patients who had their swallow assessed. Those patients (28) that had a reduced conscious level at the time of admission were not included in the study. The results are presented in table 1.

Chest infection

The occurrence of chest infection during the first week after the stroke was found to be 2 fold higher in those with dysphagia (DOC, 33% vs 16% $\chi^2(1)=3.9$, $p<0.05$; SLT 17% vs 43%, $\chi^2(1)=6.6$, $p<0.01$). Aspiration demonstrated on videofluoroscopy was not associated with an increased incidence of chest infection (Fisher's Exact test, $p>0.05$).

Twenty two patients were withdrawn early (after 1 week) from the study (14 further stroke, 3 hip surgery, 2 carotid endarterectomy, 2 dementia and 1 at the patients request). These have not been included in the analysis of outcomes documented below. There was an equal number in both groups (those with dysphagia and those with out).

Mortality

DOC

Allowing for those patients who were withdrawn due to a further stroke or a change in diagnosis, 99 patients had data recorded. No deaths occurred after

the first four weeks of the study. Fifty (50%) patients had a safe swallow. Mortality was lower in these patients, 6%(3/50) as opposed to 37% (18/49) in those patients who had dysphagia (p<0.001).

SLT

Of those that were assessed by SLT (86), 68% (58) were swallowing safely. Of these 7(12%) died as opposed to 10/28 (36%) of those with dysphagia (p=0.022).

Videofluoroscopy

The presence of aspiration on videofluoroscopy was not associated with an increase in mortality. The figures would suggest an increase in percentage mortality, but there were only a small number of deaths contributing to this finding.

Length of hospital stay

Patients with dysphagia remained in hospital longer than those with a safe swallow. Detailed information is provided below and in table 1.

DOC

Seventy eight patients survived until discharge, of which 31 had dysphagia.

These patients had a mean length of stay of 44.8 days (95% CI 32.1, 62.5) compared to 24.5 days (95% CI 17.9, 33.5) for those with a safe swallow (p<0.01).

SLT

Sixty nine patients survived in the SLT cohort. Eighteen had dysphagia. The length of stay for these patients was 68.4 days (95%CI 51.1, 91.7) compared to 24.7 days (95% CI 18.6, 32.9) for those with a safe swallow (p<0.001).

Videofluoroscopy

Seventy eight patients were alive at the time of discharge. Of these 14 were aspirating on videofluoroscopy. The mean length of stay was 36.4 days (95% CI 6, 147) compared to 28.1 (95% CI 4,180) for those with out aspiration (p>0.05).

Functional outcome

Functional outcome at six months was assessed in two ways. Disability, by the Barthel activities of daily living scale (BAL) where as place of discharge could be said to be a measure of handicap.

Disability

Those patients with swallowing dysphagia scored less on the BAL than those without difficulties (see table 1).

DOC

Those patients with dysphagia had a lower median BAL (15, IQ 7-19) compared to those with a safe swallow (18, IQ 13-20) (p=0.02).

SLT

The results in this group mirrored those above. Those with dysphagia had a lower median BAL (11, IQ 7-9) compared to those considered to be safe (18, IQ 15-20) (p=0.002).

Videofluoroscopy

Those patients with aspiration on videofluoroscopy had a lower median BAL (13, Interquartile range, 7-20) as compared to those who were not aspirating (18, Interquartile range, 15-20). This was not statistically significant.

Place of discharge

The type of residence patients are discharged to is a marker of their functional ability and could be thought of as a measure of handicap. Broad categories

were used: Institution (Nursing/Residential Home) and Home (table 1).

DOC

Seventy eight patients were discharged from hospital. Forty five percent of patients with dysphagia were discharged to institutionalised care compared to 21% of those patients who had been assessed as having a safe swallow (p < 0.05).

SLT

Fifty percent of those with dysphagia were discharged to institutionalised care compared to 22% of those assessed as safe (p < 0.05).

Videofluoroscopy

Twenty nine percent of those aspirating and 32% of those not aspirating were discharged to institutionalised care.

These results support the evidence that the presence of an unsafe swallow, within the acute phase of stroke as detected at the bedside is associated with a poor functional outcome, increased length of hospital stay and a placement in institutionalised care. This relationship appears to hold true whether aspiration (as demonstrated on videofluoroscopy) was present or not.

	CHEST INFECTION n(%)	DIED n(%)	INSTITUTIONAL CARE n(%)	LOS (Geometric mean) (95% Confidence limits)
DOC				
Safe	9/57(16)[a]	3/50(6)[b]	10/47(21)[a]	24.5 (18,33)[c]
Unsafe	20/60(33)	18/49(37)	14/31(45)	44.8 (32,62)
SLT				
Safe	12/69(17)[c]	7/58(12)[d]	11/51(22)[a]	24.7 (19,33)[b]
Unsafe	14/32(43)	10/28(36)	9/18(50)	68.4 (51-92)
VF				
No ASP	12/74(16)	6/68(9)	20/62(32)	28.1 (4,180)
ASP	7/20(35)	4/18(22)	4/14(29)	36.4 (6,147)

a: $p < 0.05$ b: $p < 0.001$ c: $p < 0.01$ d: $p = 0.022$

Numbers in each column vary due to deaths and withdrawals at different stages of the study.

DOC: Doctor   SLT: Speech and Language Therapist
VF: Videofluoroscopy   LOS: Length of hospital stay

Table 1:   OUTCOME FOLLOWING ACUTE STROKE

In an attempt to determine whether the association between aspiration or dysphagia with poor outcome (mortality, lower Barthel scores, Length of stay and occurrence of chest infection) remained after other accepted indicators of poor prognosis were taken into account (weakness, neglect, hemianopia, incontinence, apraxia, age and gender). The presence of a reduced conscious level is a major and accepted prognostic marker of outcome following stroke, but in this study these patients were excluded from the analysis of outcome. The presence of dysphagia remained a significant predictor of mortality (DOC, $x^2(1)=6.4$, p=0.01; SLT, $x^2(1)=4.4$, p=0.04), and for chest infection (SLT $x^2(1)=9.6$, p=0.002). The presence of aspiration as demonstrated on videofluoroscopy was not an independent predictor of mortality or for the presence of chest infection. Neither dysphagia or aspiration on videofluoroscopy were independent predictors of the length of stay or Barthel score.

DISCUSSION

Previous studies have linked clinical swallowing difficulties (dysphagia) to a poor outcome following stroke (Gordon et al., 1987; Wade and Hewer, 1987; Barer, 1989; Kidd et al., 1995), but only Kidd et al., (1995) included data from videofluoroscopy imaging. This study has examined the different and separate roles of the clinical assessment and the videofluoroscopy examination on outcome following stroke. The results have shown that dysphagia was significantly related to outcome.

The outcomes studied were mortality, development of chest infection, disability, length of stay and place of discharge.

Post mortem studies have suggested that aspiration pneumonia, following stroke is a significant cause of death within the first week (Brown and Glassenberg, 1973; Silver et al, 1984). Gordon et al. (1987) noted that when patients had dysphagia there was a two fold increase in the occurrence of chest infection. More recently Schmidt et al. (1994) noted a 7.6 fold increase in the risk of developing pneumonia following aspiration. Unfortunately the study was retrospective and only involved 52 patients. Holas et al., (1994) in a prospective study of 114 patients found an increased risk of pneumonia of between 7 and 8 fold in those patients aspirating on videofluoroscopy 4 weeks after their stroke. Martin et al. (1994) performed videofluoroscopy and clinical

assessments of the ability to swallow on patients with a diagnosis of aspiration pneumonia and non aspiration pneumonia, noting that those with aspiration pneumonia were more likely to have swallowing problems.

This study has confirmed that those with dysphagia are more likely to develop a chest infection. There is a possibility that this result was biased by the fact the person assessing for the presence of a chest infection was also assessing the patient's ability to swallow, but this is finding is supported by the fact that those patients assessed as having an unsafe swallow by the SLT also had an increased risk of developing a chest infection. The SLT was not informed as to the presence or absence of a chest infection. This study has not found a significant association between the development of a chest infection in the first week following stroke and the presence of aspiration on videofluoroscopy which confirms the work by Kidd et al. (1995).

What is not clear is the relationship between swallowing difficulties, chest infection and stroke severity. Using logistic regression analysis, this study has shown that dysphagia is independently associated with the occurrence of a chest infection, but aspiration documented on videofluoroscopy was not.

Other workers have suggested that the mortality following acute stroke is increased if swallowing problems are present regardless of conscious level

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(Wade and Hewer, 1987). This study confirmed that those patients with an unsafe swallow had an increase in mortality which was between three and six fold higher. The mortality in the unsafe groups whether assessed by the SLT or DOC was 36% and 37% respectively, the same order of magnitude as Wade and Langton Hewer (1987) in their study (42%). There was no relationship between aspiration on videofluoroscopy and mortality, though the number of deaths within these groups was small, which would confirm the work by both Schmidt et al. (1994) and Holas et al. (1994).

In the present cost conscious Health Service, LOS, functional outcome and place of discharge are important. Axelsson (1989) suggested that swallowing difficulties following stroke were associated with an increased LOS. Like others (Gordon et al., 1987; Barer, 1989) this study has shown an increase in LOS associated with the presence of dysphagia, but not with aspiration on videofluoroscopy.

It has also been suggested that the functional outcome of patients with swallowing difficulties is worse (Wade and Hewer, 1987; Barer, 1989). This study has confirmed that the presence of dysphagia is associated with a worse functional outcome, as assessed by the Barthel score.

Now that the access to institutionalised care is regulated by the local authority,

markers suggestive of eventual institutionalisation are being increasingly investigated. Those with dysphagia present at the time of admission were twice as likely to be admitted to institutionalised care which is in keeping with the findings of Kalra et al. (1993) where the presence of dysphagia at two weeks was predictive of admission to a nursing home.

In conclusion these results confirm that dysphagia, as detected at the bedside is an independent predictor of the development of chest infection and mortality (survival). The presence of aspiration on videofluoroscopy does not appear to add greatly to this risk assessment.

CHAPTER 14

DISCUSSION and CONCLUSIONS

DISCUSSION

The study of swallowing difficulties following stroke has aroused much interest over the recent years, both in the United Kingdom and in the United States (Horner and Massey, 1988; Barer, 1989; Gordon et al., 1987; Splaingard et al., 1988; Horner and Massey, 1991; Robbins et al., 1993; Kidd et al., 1993; Holas et al., 1994). In the United Kingdom patients have been studied acutely, with reliance being placed upon unvalidated bedside assessments (Gordon et al., 1987; Barer, 1989) performed by untrained medical staff. Conversely, in the United States much reliance has been placed on the videofluoroscopic examination, patients having been studied months to years after their stroke (Splaingard et al., 1988; Horner and Massey, 1991; Holas et al., 1994). Only the study by Kidd et al. (1993) has examined the ability to swallow following stroke during the acute phase, using both videofluoroscopy and a bedside assessment, as has been done in this study. None of these studies have examined nutrition in any detail nor the natural history of swallowing as presented here.

Many stroke and swallowing studies are hospital based, as were the studies presented here. Potentially the results of such studies may not be true of all patients suffering a stroke, as 20% or more of people suffering a stroke are not admitted to hospital (Bonita, 1992). As unselected consecutive patients were recruited, it can be assumed that the study population was representative

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of hospital stroke populations in the United Kingdom.

At present within many hospitals there are not the resources to permit immediate access to speech and language therapists when a patient is admitted to hospital following acute stroke (College of Speech and Language Therapists, 1991). Consequently, as the prevalence of swallowing disorders immediately following stroke may be as high as 50% there is a need for a bedside assessment that can be reliably used by all grades of staff, otherwise many patients may go without nutrition for an inordinate length of time with a possible adverse effect on their prognosis (Steffee, 1980; Dávalos et al., 1996).

Bedside Assessment

Many workers have attempted to compare clinical signs with the inability to swallow, suggesting that a wet voice and dysphonia are commonly associated with aspiration (Veis and Logemann, 1985; Horner et al, 1991; DePippo et al. 1992). DePippo et al. (1992) and Nathandwarawala et al. (1992) have both suggested that large volumes of water could be used to assess the swallow, with an abnormal swallow being associated with coughing, choking and taking longer to drink the set volume of water. In the present study no significant relationship was found between the volume of water drunk and the presence of aspiration, part of the reason may be that the population under study was

different, and that the numbers going on to drink the larger volume of water was small. DePippo et al. (1992) were studying patients a long time after stroke with known swallowing difficulties. Nathandwarawala et al. (1992) did not use videofluoroscopy to verify their findings.

Accuracy of the bedside assessment

More recently Linden et al. (1993) were only able to demonstrate a sensitivity of 66% for the detection of aspiration despite using an extensive proforma. The present study was only able to demonstrate a sensitivity of the order 47%-76% (depending on the assessor), confirming the limitations of the bedside assessment in detecting aspiration. The results, however, had a negative predictive value of 90% suggesting that if the swallow was considered to be safe, then there was a 90% chance that it was.

Assessor agreement

For a clinical test to be useful, different people must be able to use it reliably to get the same result. This study, therefore, compared assessments both between and within specialties. Agreement between assessors was moderately good as assessed by the Kappa statistic, but were higher within specialities, suggesting near perfect agreement. As the two doctors that took part in the study had no training in the assessment of the ability to swallow, the results would suggest that the assessment could be widely used by other clinicians.

This needs to be qualified by the possibility that they may have been a learning effect with time for the doctors. Further work is required in this area.

A clinical tool

In the attempt to develop a clinical tool to be used for the assessment of swallowing by untrained medical and nursing staff, it is necessary to have a reference swallowing assessment, which the new assessment must be at least as good as. The assessment by the speech and language therapist was accepted as our reference assessment despite its limitations (Splaingard et al., 1988; Linden et al., 1993), as at the present time they are the accepted experts. Speech and language therapists have to undergo post graduate training to be considered competent to assess the swallow (College of Speech and Language Therapists, 1991). Their presumed expertise allows them to advise the medical and nursing staff with regard to the patient's overall management.

The results of the present study suggest that a simple bedside assessment could be used to assess the swallow of a patient immediately following acute stroke. This assessment (a weak voluntary cough, dysphonia and coughing on 5 mls of water) has a high negative predictive value (90%), ie it detects that aspiration is absent, and the swallow is safe, with a high degree of certainty. Conversely its positive predictive value was low (47%). Due to the low occurrence of aspiration (20/94 patients seen to be aspirating on

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videofluoroscopy), prior to the swallowing assessment being conducted it would be reasonable to assume that the patient being assessed would have a safe swallow. Following the BSA if it was considered that the ability to swallow was unsafe (at risk of aspiration), then given the positive predictive value of 47%, the possibility that aspiration is present has increased from approximately 20/80 to 50/50, increasing the amount of uncertainty as to the patients ability to take things safely by mouth.

Therefore following a 'safe' assessment one could be fairly certain that a patient would be safe to eat and drink, with only 10% being misclassified as being safe, when in fact they were not. This would prevent a disproportionate number of patients being given 'nil by mouth' instructions when they had a safe swallow, and also the number of incorrect referrals being made to the speech and language therapists. Further work is required to validate this assessment in different stroke populations and also in patients with different neurological conditions. If the introduction of a simple bedside assessment is coupled to some elementary instruction on its use, then there may be improvements in the predictive values, sensitivity and specificity of the assessment, this needs to be investigated

Videofluoroscopy

Previous investigators have found the bedside assessment to not be accurate

enough for routine clinical use (Splaingard et al., 1988, Linden et al., 1993).

Martin and Corlew (1990) have stated that:

"Although oral-pharyngeal motility disturbances may be suspected during the initial bedside assessment, these problems and possible aspiration cannot be adequately diagnosed, quantified, and subsequently managed without videofluoroscopy".

The College of Speech and language Therapists (1991) have also stated that if speech and language therapists are going to provide a dysphagia service, then videofluoroscopy back up must be present.

Videofluoroscopy was the accepted 'Gold Standard' used during the present studies despite the limitation that videofluoroscopy only assesses the swallow for a discreet moment in time, subjects are studied in the 'ideal' situation (ie sitting upright with their head supported), and the detection of aspiration is dependent on the operator. Lof and Robbins (1990) have shown that the normal swallow is subject to variability both between people and repeated swallows by the same person, and that aspiration could be missed unless repeated swallows are made during the standard examination. When the swallow was assessed during the present study, each assessment was repeated at least three times whether it was clinical or radiographic, and during

videofluoroscopy examination the swallow was examined until the pharynx was clear of barium in order not to miss delayed aspiration. The detection of aspiration following ingestion of barium depends on the amount aspirated, and the radiologist reporting the examination. During this study there was only moderate agreement between two radiologists as to the presence or absence of aspiration.

Despite its limitations, videofluoroscopy is, at present, still considered to be the "gold standard" (Bastain, 1991; Langmore et al., 1991) for the study of swallowing problems. It provides information regarding both structure and function and allows the speech and language therapists the opportunity to study therapeutic intervention as patients swallow. Patients with a reduced conscious level cannot safely under go the procedure, but a reduced conscious level is a risk factor for an unsafe swallow (Barer, 1989), which has been confirmed in the present study. Other techniques such as nasendoscopy (with or without video recording) and ultrasound can be used at the bed side and they do not involve exposure to radiation (Langmore et al., 1991). At present these techniques are not widely used, and suffer from their inability to study the swallow as a whole. Nasendoscopy can only infer as to the presence of aspiration by the presence of food/liquid in the larynx after the swallow has been completed.

Side of the lesion

The cortical representation of swallowing is bilateral. Only Robbins et al. (1988, 1993) have been able to demonstrate an association between the side of stroke and aspiration. They studied first ever strokes 3 weeks after their stroke. This study examined all stroke patients and an association was demonstrated only 4 weeks post stroke, confirming the work of Robbins et al (1988, 1993). This work has not found an association between the presence of aspiration and the side of stroke acutely. This difference may be due to neural changes occurring over 4 weeks, those patients with the most severe strokes dying between the two assessments or a erroneous result because of the small numbers involved. Further work is required to examine the association of aspiration and the side of the stroke. There is little published work examining the association between the side of hemiparesis and aspiration, further work is required to confirm or refute any association.

Natural History

The natural history of the ability to swallow following stroke must be remembered. Once a swallow has been assessed as safe, this does not mean it is safe for ever. The results presented in the natural history phase of the swallow is the first study to examine the ability to swallow sequentially both clinically and with repeated videofluoroscopy examination. The study by Kidd

et al., (1995) did repeat the videofluoroscopy examination in those shown to have no abnormalities at the time of the first videofluoroscopy. The results presented earlier show that some patients alternated between being safe and unsafe with time. Some even became unsafe as time wore on. Whether this was due to changes within the patient or due to poor test retest reliability of the BSA remains an interesting question. At the time of the second videofluoroscopy assessment 13 patients were aspirating, all silently as assessed by SLT. What does this mean with regard to long term prognosis? Does silent aspiration affect outcome following stroke, this needs further investigation with a large number of patients.

Nutrition

The present studies have shown that there is a rapid deterioration in the nutritional status of dysphagic patients over the first 7-28 days, which confirms the work of Dávalos et al. (1996) who found a significant decrease in fat and visceral protein compartments. But, more importantly, base line was not regained by 6 months. Dávalos et al. (1996) suggest that acute stroke patients are hypercatabolic, as suggested by Touho et al. (1990) in patients following an haemorrhagic stroke. Others have suggested that their caloric requirement

is low (Weekes and Elia, 1992). This is compounded by the fact that patients unable to take nutrient orally have their nutritional requirements managed badly (Steffee, 1980). Often only intravenous fluids are offered. Enteral feeding is often considered late (Steffee, 1980) which may be too late as protein energy malnutrition occurs early (Wilmore, 1991; Dávalos et al., 1996), but may not be prevented by earlier enteral feeding (Dávalos et al., 1996). Protein calorie malnutrition that ensues will result in progressive loss of lean body mass, and could, in theory (Veldee and Peth, 1992), worsen any dysphagia that may be present. Consequently, if intervention is too delayed, there will be a need to hyperalimate patients, which at present is via a nasogastric tube. Recent studies, however, have shown that feeding regimens often fail with this method of enteral nutrition (Park et al., 1992; Wicks et al., 1992; Norton et al., 1996), and at present there is a reluctance to use percutaneous endoscopic gastrostomy therapeutically rather than as a last resort.

Poor nutritional status during illness will result in an increased risk of infection what ever the presentation (Chandra, 1990; Potter et al. 1995) due to anergy, which will also be associated with an increase in the risk of pressure sore occurrence and increased mortality (Ek et al., 1990). Consequently early supplemental nutrition may improve the prognosis of some of these patients, with a consequent reduction in the length of hospital stay. The use of early

enteral nutrition has been suggested by Nyswonger and Helmchen (1992), and Nydevick and Hulter-Asberg (1992), where in a retrospective study, those fed early where discharged earlier. Unfortunately they fail to elaborate on the swallowing status of their patients. This state of poor nutrition may persist for many months after an acute illness (Vellas et al., 1992). This study has confirmed the findings of others (Axelsson et al., 1982, 1988) that nutritional deterioration occurs following stroke, as is common with any severe illness. What is most interesting is that this deterioration is most marked in those with an unsafe swallow, whether aspiration was present or not. A recent study looking at the role of percutaneous endoscopic gastrostomy in very dependent stroke patients has showed an improved outcome (Norton et al., 1996), but the assessments of dysphagia were poor resulting in questions being asked about the patients assigned to the various groups (nasogastric tubes or percutaneous endoscopic gastrostomy).

The association between stroke severity and the deterioration of nutritional status has not been investigated here. Certainly Axelsson et al. (1982) considered that the deterioration could not be explained only by swallowing problems. Unosson et al. (1994) have suggested that protein energy malnutrition and the break down of body fat stores occurs independently of the functional ability following stroke. Further work will be needed to elucidate this and to see whether early feeding of stroke patients will improve

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functional outcome and reduce length of hospital stay. This would suggest the need for a multicentre study to ensure that the study had sufficient power to answer the question.

Outcome

Is it essential that aspiration is detected clinically or is it adequate to assess somebody as being unsafe to swallow regardless of their 'aspiration status'? What information do clinicians require to determine outcome? The studies presented here have attempted to answer this question. The answer has resource implications. If it is essential that presence of aspiration is determined, then all patients suffering acute stroke will have to undergo videofluoroscopy.

Wade and Hewer (1987) reported an increased mortality in those with clinically apparent swallowing difficulties, but the role of dysphagia as an independent predictor of outcome was not investigated. Using multivariate analysis, Barer (1989) identified that those patients with dysphagia following stroke had a decreased functional outcome compared to those without though this accounted for only 4% of the variance. Dysphagia may also be associated with specific complications, such as chest infection (Gordon et al., 1987, Schmidt et al., 1994; Kidd et al., 1995) and some groups have reported

dehydration to be more common in those patients with dysphagia (Gordon et al., 1987; Barer, 1989), others have not found a similar relationship (Holas et al., 1994; Schmidt et al., 1994). Others have reported an increased risk of nutritional deterioration and occurrence of chest infection, if aspiration is seen on videofluoroscopy. More recently in a retrospective analysis Schmidt et al. (1994) have reported a 7.6 fold increase in the development of pneumonia, a 9.2 fold increase in mortality, but no increase in the presence of dehydration in those patients found to be aspirating on videofluoroscopy, interestingly Holas et al., (1994) and Kidd et al., (1995) in separate prospective studies did not find any increase in mortality in those patients aspirating on videofluoroscopy.

Aspiration, as discussed earlier, may be clinically apparent or silent. Silent aspiration is common (Splaingard et al., 1988), and is known to persist for a long time (Splaingard et al., 1988; Horner and Massey, 1988) but its significance is unknown. It is possible that silent aspiration has no effect on prognosis. Holas et al. (1994) found that patients with silent aspiration had an increased risk of developing pneumonia, but no increased risk of death or dehydration. In this prospective series of patients, of which half of those patients aspirating were doing so silently, the presence of silent aspiration was not associated with an increased complication rate (pneumonia, dehydration) or worse functional outcome than those without aspiration on

videofluoroscopy.

The results have shown that patients with dysphagia at the time of admission have a worse outcome than those with out. These patients have a lower barthel score at 6 months following their stroke and were twice as likely to be resident in institutionalised care (residential/nursing homes). This has confirmed the work of Kalra et al., (1993) who have suggested that the presence of dysphagia at 2 weeks is an independent predictor of the place of discharge

Are the development of pneumonia, increased mortality, increased length of hospital stay and worse functional outcome related to the presence of dysphagia independent of the stroke or is the presence of dysphagia a function of stroke severity. The results presented in these studies have shown that dysphagia has an independent role to that of stroke in the development of pneumonia and death, but not on length of stay or eventual functional outcome. The presence of aspiration as demonstrated on videofluoroscopy was not independently associated with any of the outcome measures. The discrepancy in these results may be due to the small numbers of patients aspirating on videofluoroscopy, and in fact more patients aspirate on the ward than during videofluoroscopy. This difference is due to the care taken in positioning the patient during videofluoroscopy which may not occur on the ward, and that single swallows are being investigated in a controlled

environment. It may be more beneficial if future work investigates repeated

swallowing (a more natural process), aspiration and outcome

What is the role of videofluoroscopy?

What therefore is the role of videofluoroscopy in the acute phase of stroke.

The data presented here would argue strongly that videofluoroscopy should

not be used routinely to look for the presence of aspiration in the acute phase

of stroke. What is needed is a quick, reliable bedside assessment, such as the

simple bedside assessment. Videofluoroscopy should remain a tool in the

management of swallowing disorders, helping the speech and language

therapist decide which is the most appropriate compensatory strategy that will

enable the patient to swallow safely.

CONCLUSIONS

This series of studies investigated the occurrence, natural history and the effect of dysphagia on outcome following acute stroke. the null hypothesis being that it would not be possible to detect aspiration at the bedside, and that dysphagia or aspiration do not exert an independent effect on outcome.

1. How common are swallowing difficulties at the time of admission to hospital and what proportion of patients are aspirating during the acute phase of stroke?

Within the population under study a 31% (SLT1 assessment) -50% (DOC1 assessment) of patients had swallowing difficulties at the time of the initial assessment. Videofluoroscopy performed within a median time of 2 days found that 22% of those undergoing the examination were aspirating, half of them silently.

2. Can a bedside assessment be used to detect the presence of aspiration?

Is it possible to devise a bedside swallowing assessment to be used during the acute phase of stroke, that has been validated against videofluoroscopy, and what elements of this bedside assessment can be

used to reliably detect or exclude the presence of aspiration?

Simple bedside assessments were devised and used during this study. They were found not to be sensitive enough to enable a clinician to reliably detect aspiration. Sensitivities obtained in this study ranged from 47% (DOC1) to 68% (SLT1), this is similar to the results obtained by Linden et al., 1993. The use of an impaired conscious level, and a weak voluntary cough gave a NPV of 91% for SLT assessment, whereas from the DOC assessment the presence of any of impaired conscious level, cough with 5mls of water and a weak voluntary cough gave a NPV of 90%. Thus the test would allow a reasonable degree of accuracy in detecting those with a safe swallow during the acute phase of stroke. These results would suggest that using these markers of an unsafe swallow a physician would be able to predict a safe swallow and the DOC BSA is as useful as the SLT BSA. Consequently medical and nursing staff should be able to reliably predict a safe swallow and hence reduce the referral burden on SLTs. The new simple assessment needs to be validated on a different study population.

3. Is there a relationship between the side of the stroke and the occurrence of aspiration? Does the timing of the investigation matter?

The results of this study neither confirmed nor refuted the work of others (Robbins et al., 1988; Chen et al., 1990; Robbins et al., 1993; Alberts et al., 1992). The results suggested that in the early stages of stroke no association exists between the presence of aspiration and the site of the lesion. Information from the later assessment suggested that there might be an association between right hemisphere lesions and the presence of aspiration ($p < 0.05$), unfortunately numbers were small and the chance for a type II error large. Larger studies are required to examine this relationship further. A more detailed examination of both the oral and pharyngeal phases of swallowing needs to be done both in the acute and rehabilitative phases of swallowing to examine the changes that occur in different phases of swallow and their effect on outcome. A larger study examining first ever strokes both acutely and at three weeks may give a definitive answer as to whether there is an association between the side of the stroke and aspiration.

4. To examine the ability to swallow safely following acute stroke: does a safe swallow always remain safe?

Many previous studies looking at the natural history of swallowing, stopped assessing a patient's swallow as soon as the problems were deemed to have resolved (Gordon et al., Teasell et al., 1994, Barer 1989). None of the studies repeatedly assessed the swallow. Splaingard et al (1987) had shown that some patients had swallowing difficulties many months or years after their stroke. The results of the present study have shown that a patient's ability to swallow safely following stroke varies on a day to day basis. At each assessment point different people were being assessed as new problems. On a second VF examination a median time of 29 days following their stroke, 12 were noted to be aspirating, all of them silently. some patients were even assessed to have persistent problems at 6 months following their stroke. Consequently, is it misguided to presume that a safe swallow is always a safe swallow? Further work is needed to investigate the significance of late onset dysphagia.

5. To examine the relationship between the presence of dysphagia, aspiration and nutritional (including hydration) status following acute stroke. Does the presence of dysphagia with or without aspiration

matter?

The results have shown that those patients with dysphagia (unsafe swallow) at the time of admission underwent a deterioration in their nutritional status over the first 28 days as measured by their AMC, Tsf and albumen. A similar picture was found for those patients where aspiration had been documented on videofluoroscopy. Documenting the presence of aspiration did not add any significant information about the patient that had not been recorded clinically. No evidence was found to suggest that patients with dysphagia become dehydrated as a whole. This was due to the fact that parenteral fluids were used more frequently and for longer in those patients with dysphagia.

Further research is needed to investigate relationship between stroke, nutrition and outcome, this would include supplemental feeding either orally or enterally.

6. Is aspiration or dysphagia an independent predictor of the occurrence of chest infection, mortality and length of stay, or are they just a function of stroke severity?

Previous workers had suggested that those patients with dysphagia following stroke had an increased occurrence of chest infection (Gordon et al., 1987,

Schmidt et al., 1994), increased mortality (Wade at al., 1987; Barer 1989, Schmidt et al., 1994) and were functionally worse if they survived. The results from this study confirm this. Patients with swallowing problems and /or aspiration as seen on VF were up to five times more likely to develop a chest infection, twice as likely to die and more likely to be discharged to an institution (nursing or residential home).

EFFECTS ON CLINICAL PRACTICE

How do these results affect the way stroke patients are managed?

All patients should have their ability to swallow assessed. This could be done by a nurse or junior doctor observing conscious level, ability to cough and the ability to swallow a tea spoon of water on 3 occasions. The ability to swallow needs to be assessed early in an attempt to reduce the incidence of aspiration pneumonia, which may have an important role in the increased mortality associated with dysphagia independent of the stroke itself.

A simple bedside assessment should be used to assess the ability of a patient to swallow safely. If no problems are encountered then it can be assumed with

reasonable certainty that the swallow is safe, but it cannot be assumed that aspiration is not present. These results seriously question any role for videofluoroscopy during acute stroke, as the result of the examination added little to the knowledge gained clinically at the bedside. I would suggest that videofluoroscopy should be a second line investigation reserved for those patients where doubts exist regarding the type of swallowing difficulty and how to manage it.

Nutritional status deteriorates quickly in those with swallowing difficulties on admission to hospital. By 6 months this has not been fully regained. Medical and nursing staff should reconsider their policy regarding the feeding of patients with acute stroke and consider feeding earlier. The use of enteral feeding may increase and its role (which patients and when) in the management of dysphagia following stroke should be critically examined.

SUMMARY

These studies have shown that a simple bedside assessment can detect the ability to swallow safely, but cannot completely exclude the presence of aspiration. The presence of swallowing problems may persist for a long time,

resolve or occur for the first time later in the course of the stroke. Those patients with dysphagia (swallowing problems) on admission have an increased occurrence of chest infection, mortality, length of hospital stay, institutionalization and a significant decline in their nutritional status. dysphagia has been found to be independently associated with the occurrence of chest infection and mortality.

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APPENDIX

SWALLOWING ASSESSMENT

Name_____   Registration No._____

Date_____   Day [   ]   Doctor   1 / 2

Conscious level   (Alert=1, Drowsy but rousable=2,   [   ]
                    Response but no eye opening to speech=3, Responds to pain=4)

Head and Trunk control   (Normal sitting balance=1, Sitting balance not maintained=2   [   ]
                           Head control only=3, No head control=4)

Breathing Pattern   (Normal=1, Abnormal=2)   [   ]

Lip Closure   (Normal=1, Abnormal=2)   [   ]

Palate Movement   (Symmetrical=1, Asymmetrical=2, Minimal/Absent=3)   [   ]

Laryngeal Function   (Aah/ ee)   (Normal=1, Weak=2, Absent=3)   [   ]

Gag   (Present=1, Absent=2)   [   ]

Voluntary Cough   (Normal=1; Weak=2; Absent=3)   [   ]

Stage 1: Give a tea spoon (5 mls) of water 3 times

Dribbles water   (None/once=1; >once=2)   [   ]

Laryngeal movement on attempted swallow   (Yes=1; No=2)   [   ]

"Repeated movements" felt?   (None/once=1; >once=2)   [   ]

Cough on swallowing   (None/once=1; >once=2)   [   ]

Stridulous on swallowing   (No=1; Yes=2)   [   ]

Laryngeal function after swallowing (Normal=1; Weak/wet=2; Absent=3)   [   ]

Stage 2: if the swallow is normal in stage 1 (2 out of 3 attempts) try 60mls of water in a beaker.

Able to finish?   (Yes=1; No=2)   [   ]

Time taken to finish in seconds   [   ]

Number of sips   [   ]

Cough during or after swallowing   (No=1; Yes=2)   [   ]

Stridor during or after swallowing   (No=1; Yes=2)   [   ]

Laryngeal function after swallowing (Normal=1; Weak/wet=2; Absent=3)   [   ]

Do you feel aspiration is present   (No=1; Possible=2; Yes=3)   [   ]

Speech Therapy Assessment   Day   1 2 3 4 5 6 7 28   180

Therapist   1 / 2   Study Number   Hospital No.

Head Posture   Normal / Abnormal

Trunk Control   Normal / Abnormal

Alertness   Alert / Drowsy / Unconscious

Communication   Normal / Abnormal

Respiration   Normal / Abnormal

Lip closure

At rest   Normal/ Weak/ Absent

Eating/ Drinking   Normal/ Weak/ Absent

Speech   Normal/ Weak/ Absent

Tongue movements

Protrusion   Normal/ Weak/ Absent

Lateral movement   Normal/ Weak/ Absent

Velar movement   Normal/ Weak/ Absent

Gag Reflex   Stroke side   Present/ Absent

                                  Normal side   Present/ Absent

Palatal function

Speech   Normal / Abnormal

Nasal Regurgitation   Yes / No

Tongue function

Eating   Normal / Abnormal

Drinking   Normal / Abnormal

Drooling   Present / Absent

Jaw Movement   Normal / Abnormal

Laryngeal Function

   Voluntary cough   Normal/ Weak/ Absent

   Phonation pre swallow   Normal/ Abnormal/ Absent

   Involuntary cough   Normal/ Weak/ Absent

   Phonation post swallow   Normal/ Abnormal/ Absent

Swallow Reflex   Normal/ Delayed/ Absent

Pharyngeal function

   Regurgitation   Yes / No

   Pooling in Pharynx   Yes / No

No. of swallows to clear bolus from pharynx   1 2   3 4 >4

Tracheal penetration (cough)   Present / Absent

Laryngeal penetration   Present / Absent

Do you feel aspiration is present?   Yes / No