

Sites and sizes of fat deposits around the pharynx in obese patients with obstructive sleep apnoea and weight matched controls

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ABSTRACT: It has been suggested that deposition of fat in the soft tissues surrounding the upper airway may be an important factor in the pathogenesis of obstructive sleep apnoea (OSA) in obese subjects. We have used magnetic resonance imaging to determine the site(s) and size(s) of fat deposits around the upper airway in six obese patients with OSA (116–153% of ideal body weight) and five weight-matched controls without OSA (107–152% of ideal body weight). In all subjects, large deposits of fat were present postero-lateral to the oropharyngeal airspace at the level of the soft palate. Significantly more fat was present in these regions in the patients with OSA ($p=0.03$). Fat deposits in the soft palate were observed in 4 of the 6 patients with OSA but none of the controls. Fatty streaks were observed in the tongue in 2 of the 5 controls and 3 of the 6 patients with OSA. Fat deposits were observed anterior to the laryngopharyngeal airspace, in submental regions, in all obese subjects. This study shows that more fat is present in those areas surrounding the collapsible segment of the pharynx in patients with OSA, compared to equally obese control subjects without OSA.

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Anatomic abnormalities of the upper airway which can contribute to the development of obstructive sleep apnoea (OSA) include adenotonsillar hypertrophy [1], retrognathia and micrognathia [2], and macroglossia [3, 4]. However, the majority of patients with OSA have no such obvious abnormality. OSA is usually accompanied by obesity [5] and weight loss is beneficial in treatment [6, 7]. Although much work has focussed on the mechanisms and treatment of the periodic upper airway closure which occurs during sleep in patients with OSA, there have been no studies which have attempted to answer one of the most fundamental questions related to this condition, *i.e.* what is the nature of the association between OSA and obesity? The answer to this question has been subject to much speculation. It has been suggested that deposits of fat in the tongue, or in the soft tissues surrounding the upper airspace, may narrow this airspace during wakefulness. This would predispose the airway to collapse when upper airway muscle tone is reduced during sleep [8]. There is no experimental evidence to support this view. Studies with computed tomography [9, 10] have failed to identify any abnormal deposits of fat immediately around the upper airway in a group of

obese patients with OSA. However, these authors only looked at regions immediately around the airway and at sites of airway narrowing [9, 10].

The purpose of the present study was to re-evaluate this question using magnetic resonance imaging. The technique is particularly suitable because it is possible to image the pharynx with different imaging sequences that include and exclude fat; fat deposits can then be observed by subtraction of the images. The objective of this paper is to describe and compare the site(s) and size(s) of fat deposits around the upper airway in a group of obese patients with OSA and a group of weight matched control subjects without OSA.

Methods

Overnight sleep studies

All subjects (except numbers 1 and 5) were studied overnight during sleep on at least one occasion. The subjects took no sedative drug or alcohol prior to the sleep studies. Breathing pattern was monitored by

respiratory inductance plethysmography. End-tidal P_{CO_2} , measured from expired air sampled at the nose (Beckman LB2), or oro-nasal thermocouples, were used to indicate the existence of airflow. Arterial oxygen saturation (Sa_{O_2}) was measured continuously with a pulse ear oximeter (Ohmeda 3700). The international 10–20 system of electrode placement [11] was used in order to record two electroencephalograms (C_3/A_2 and C_4/A_1) and two electrooculograms (F_7/A_1 and F_8/A_1). Overnight sleep patterns were determined from these recordings according to standard criteria [12]. All signals were recorded on analogue instrumentation tape and on chart recorders. In order to record body position and breath sounds, a video record was made during each study using a time-lapse recorder (Panasonic 8050).

Obstructive apnoeas were classified as the absence of airflow for at least 10 seconds, in the presence of attempted respiratory efforts, leading to a decrease in Sa_{O_2} of greater than or equal to 4%. Hypopnoeas were detected as a decrease in Sa_{O_2} (greater than or equal to 4%) in the presence of respiratory efforts and airflow but with reduced tidal volume. These episodes were usually terminated by a brief arousal from sleep.

Subject details

The subjects were divided into two groups based on the results of the overnight sleep studies and subject interview.

Anthropometric data for each subject are included in table 1. The first group consisted of 5 male control subjects, three of whom were monitored overnight during sleep and showed no evidence of OSA in any sleeping position or sleep stage. The other two (subjects 1 and 5) had no sleep study but they denied snoring (from subjective and familial observations) and also other symp-

toms such as abnormal daytime sleepiness, poor quality of sleep or morning headaches. These two subjects were therefore included in the group of control subjects.

The second group consisted of 6 male patients with moderate/severe OSA (with an apnoea + hypopnoea index within the range of 25–69 per sleep hour). These patients were referred to the sleep laboratory with a history of occasional or habitual loud snoring and excessive daytime sleepiness. Subjects were considered obese if their computed percentage of ideal body weight [13] was greater than 120% [14]. Subjects 7 and 11 had previously undergone uvulopalatopharyngoplasty but OSA still remained. The only criteria by which the patients with OSA were asked to participate in the magnetic resonance study was that they had unequivocal OSA, *i.e.* were not "borderline" cases.

Pharyngeal magnetic resonance imaging

This study has utilised proton magnetic resonance imaging to produce tomographic images of the pharynx. This technique is without known biological hazard [15]. We have utilised two of the pulse sequences commonly used in magnetic resonance imaging; the "spin echo" and the "inversion recovery" sequences [16, 17]. All images were obtained using a Picker International Vista MR2055 machine operating at a magnetic field strength of 0.5 Tesla. Images were first obtained using the spin echo sequence (time of "echo" signal 40 msec, repeat time 1200 msec, images were acquired with 2 repetitions of 256 phase encoding steps). Images were then acquired, in the same anatomical regions, with an inversion recovery sequence (time of inversion 110 msec repeat time 3200 msec).

The spin echo derived image revealed anatomy with contrasting signals from different tissues. The inversion

Table 1. – Subject details

Subject number	Age yrs	Height m	Body weight kg	% of ideal body weight %	AHI	Cross-sectional area of fat deposits cm^2			
						1	2	3	mean
1	26	1.68	102	152	–	4.40	2.79	2.37	3.19
2	16	1.63	91	144	<5	1.54	0.91	0.70	1.05
3	36	1.81	102	132	<5	3.05	1.61	1.81	2.16
4	57	1.73	83	126	<5	3.99	2.37	1.99	2.78
5	33	1.71	68	107	–	2.97	1.90	1.39	2.09
mean	34	1.71	89	132		3.19	1.92	1.65	2.25
6	51	1.60	88	153	25	7.11	4.01	3.39	4.84
7	45	1.63	88	143	25	5.30	3.25	1.08	3.21
8	53	1.89	113	134	43	6.88	4.50	3.98	5.12
9	57	1.75	92	126	69	3.80	5.58	4.36	4.58
10	64	1.74	78	118	64	4.02	2.44	1.88	2.78
11	56	1.54	65	116	34	3.87	1.88	0.94	2.23
mean	54	1.69	88	132	43	5.16	3.61	2.61	3.79
p	0.04	ns	ns	ns	0.01	0.04	0.03	ns	0.03

Anatomical levels of the palatopharynx are denoted by 1, 2 and 3 in the table, representing three contiguous transverse sections taken from the level of the inferior surface of the hard palate (level 1) and from more caudal regions (levels 2 and 3 respectively). Abbreviations: AHI: number of obstructive apnoeas per hour of sleep; p: level of significant difference between control subjects (numbers 1–5) and patients with OSA (numbers 6–11) using the Mann-Whitney U test; ns: not significant.

recovery derived images revealed the anatomy but with fat specifically removed from the image. When pairs of images, obtained by the two imaging sequences, were compared then the location of fat within such images could be determined (see Results). The efficacy of the inversion recovery sequence in the suppression of fat from the images was determined for each subject before each study. The sequence used in the present study was found to be ideally suited for suppression of the signal due to fat from sites of known fat deposition *i.e.* subcutaneous sites. All images were obtained in the naso-, oro-, and laryngo-pharynx. Contiguous transverse 7 mm sections (obtained approximately perpendicular to the posterior pharyngeal wall), and a midline sagittal section, were obtained with the subjects supine. The subjects breathed normally and were asked not to swallow or cough and to remain very still for the duration of the study. To help the subjects stay awake (important in reducing motion artifact, especially in the patients with OSA should they fall asleep) the subjects listened to the radio. The total time of data acquisition was about 20 minutes for each of the two sequences. All subjects gave written consent to participate in the study after full explanation of its nature.

Anatomical definitions and terminology

For the purpose of analyses we have defined 4 pharyngeal regions of interest (figure 1) based on the anatomical definitions of Gray's anatomy [18]. The inferior

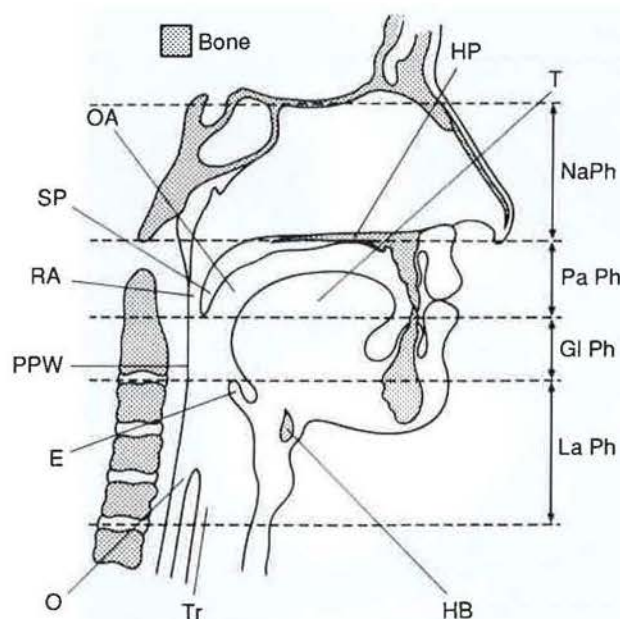


Fig. 1. - Schematic representation of sagittal section through the head and neck showing anatomical landmarks and anatomical reference levels used in analyses of magnetic resonance images. Abbreviations: Na Ph: nasopharynx; Pa Ph: palatopharynx; Gl Ph: glossopharynx; La Ph: laryngopharynx; OA: oral airspace; RA: retropalatal airspace; SP: soft palate; HP: hard palate; PPW: posterior pharyngeal wall; T: tongue; E: epiglottis; HB: hyoid bone; O: oesophagus; Tr: trachea.

surface of the hard palate has been taken to represent the naso/oropharyngeal junction. The oropharynx has been divided into two regions, the palatopharynx and the glossopharynx. The palatopharynx is that region in the oropharynx that includes the entire length of the soft palate. The glossopharynx is below the level of the soft palate and extends to the cephalad tip of the epiglottis. The laryngopharynx is below the tip of the epiglottis and extends to the cricoid cartilage. The pharyngeal regions that were imaged in this study extended from just below the level of the orbits to the superior margin of the thyroid cartilage in 5 of the 11 subjects and further, to the laryngeal vestibule, in the other 6. The anatomical reference levels were determined from anatomical landmarks on the transverse spin echo images.

Analyses of magnetic resonance images

For each subject the images obtained with both sequences were matched together by comparing anatomical landmarks.

Sites of fat deposition. Sites of fat deposition were determined from all scans in all subjects by comparing images obtained in the same anatomical regions with the two separate imaging sequences. These observations were performed for all the separate regions of the pharynx described above and were carried out by two independent observers.

Other analyses. To further illustrate and compare sites of fat deposition between the two groups, the airspace and fat deposits identified from images obtained at three separate levels of the palatopharynx were traced. Since the inferior surface of the hard palate (figure 1) was an identifiable and comparable anatomical landmark between subjects, then analyses were performed on the images obtained at that level and on those images obtained at the two scan levels immediately below, *i.e.* analyses of fat deposits at the level of the palatopharynx were performed along a 21 mm segment of the palatopharynx. The location of fat was determined by overlaying the tracing from the spin echo derived image with that of the inversion recovery derived image. The sizes of these fat deposits were then determined for each subject from these traces by planimetry. These analyses were performed with the experimenter unaware of whether the subject was a patient with OSA or a control.

The design of this study did not allow meaningful measurements to be made with respect to the size of the pharyngeal airspace. With magnetic resonance imaging, the composite image for each transverse section is made up over the total imaging time (approximately 20 minutes in this study). The occurrence of coughs, swallows or sighs during this time all render the image susceptible to motion artifact which can "blur" slightly the definition of the walls of the pharyngeal airspace. This was apparent on only a few of the images obtained (usually at the level of the tongue) but clearly render measurements relating to the size of the oropharyngeal airspace (mean or minimal cross-sectional areas) subject to error.

However, at the levels of the palatopharynx at which fat deposits were quantified, the boundaries of fat were clearly defined and readily identifiable.

Statistical analyses

Mann-Whitney U tests were used to test for differences between control subjects and the patients with OSA. A one tailed test was used since the hypothesis was that the patient group were different from the control group in a single direction. Differences between groups were considered significant if $p < 0.05$.

Results

The group of control subjects were significantly younger than the patients with OSA (mean age of control subjects 34 years, median 33 years, range 16–57 years; mean age of the patients with OSA 54 years, median age 55 years, range 45–64 years, $p = 0.04$) although the two groups were well weight matched (table 1).

Sites of fat deposition

Identifiable fat deposits were located in distinct regions of the pharynx:

Nasopharynx. Fat deposits were observed lateral to the maxillary sinuses and medial to the zygomatic arches in all subjects, including the three subjects who were not obese. The typical appearance of such deposits is shown in figure 2.

Oropharynx. No identifiable fat deposits were located immediately adjacent to the oropharyngeal airspace in any of the subjects studied. However, significant deposits of fat were observed further removed from the edge of the airspace. At the level of the palatopharynx, fat deposits were located postero-lateral to the retropalatal airspace in the vicinity of the carotid vessels. The typical appearance and location of these deposits is shown in figure 3 and were observed in all subjects, including the three subjects who were not obese. Deposits of this type were present at all levels of the palatopharynx although at the level of the glossopharynx they were not present in any subject (figure 4).

Fatty streaks in the tongue, of the type shown in figure 4, were observed in 3 of the 6 patients with OSA (numbers 8, 9 and 11) and 2 of the 5 control subjects (numbers 3 and 5). Evidence of fat in the tongue was observed usually in those scans taken at the level of the glossopharynx. Fat in the palate, of the type shown in figure 5, was observed in 4 of the 6 patients with OSA (numbers 6, 8, 9 and 11) and none of the control subjects.

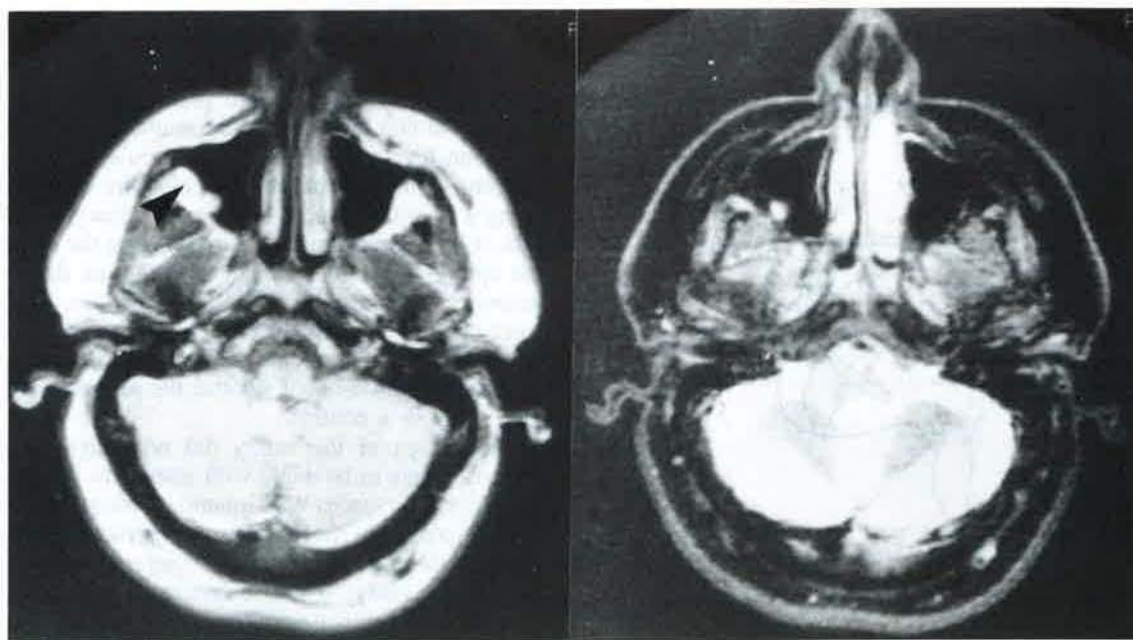


Fig. 2. – The pair of transverse sections shown in this figure are taken from the level of the nasopharynx in subject 1 and show the maxillary sinuses separated by the nasal septum. The spin echo derived image is shown on the left, fat deposits appear white. To differentiate fat deposits from other tissues which also appear white, the inversion recovery derived image of the same region is shown on the right. Fat has been removed from this image (see Methods). Fat deposits can be localised by comparing pairs of such images. Subcutaneous fat deposits are clearly seen. Fat deposits lateral to the maxillary sinuses are indicated by the arrowhead on the spin echo derived image.

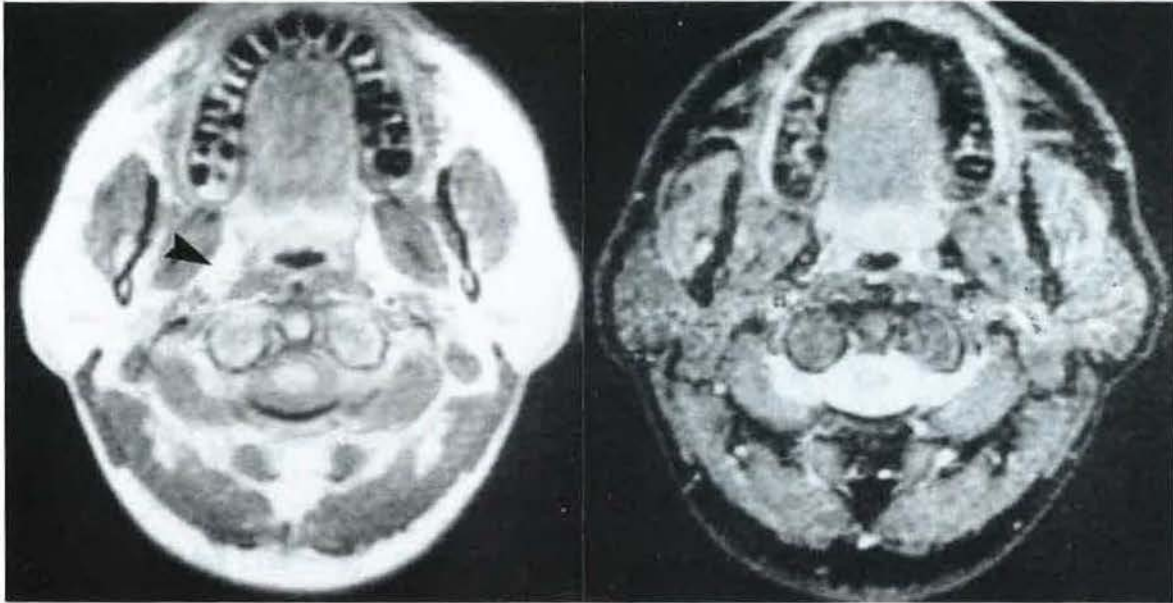


Fig. 3. – The pair of transverse sections shown in this figure are taken from the level of the palatopharynx in subject 3 and are presented in the same way as in figure 2. Large amounts of subcutaneous fat are clearly observed. There are no identifiable fat deposits immediately around the airspace. Fat deposits located postero-lateral to the airspace are indicated by the arrowhead on the spin echo derived image.

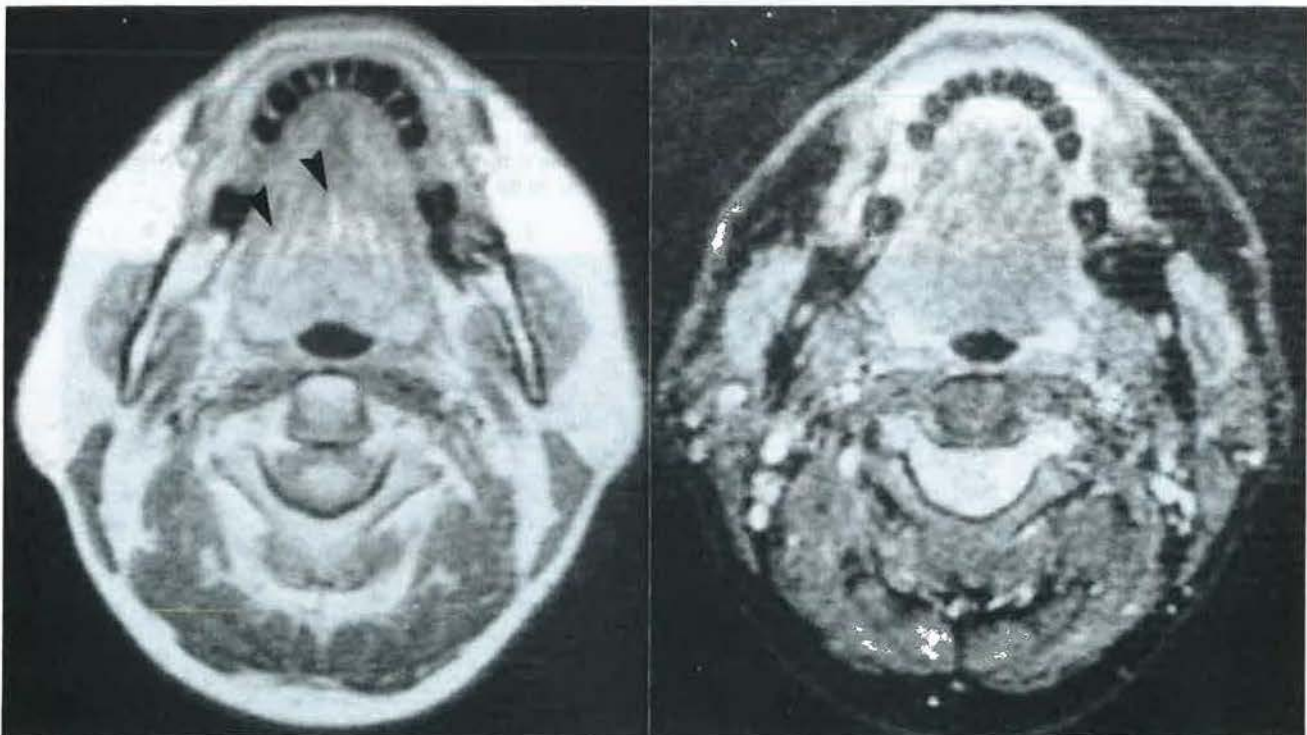


Fig. 4. – The pair of transverse sections shown in this figure are taken from the level of the glossopharynx in the same subject depicted in figure 3 and are presented in the same way as those in figure 2. The figure shows that although subcutaneous fat deposits are again readily observed, fat deposits postero-lateral to the airspace (as in figure 3) are not present at this level of the oropharynx. Fatty streaks are observed in the tongue (see arrowheads on the spin echo derived image).

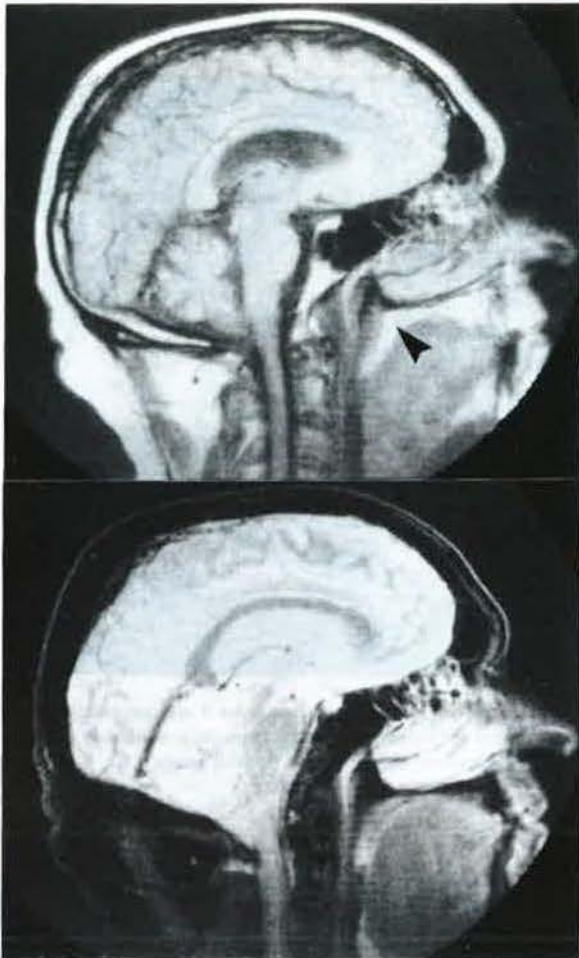


Fig. 5. - The pair of sagittal sections shown in this figure are presented with the spin echo derived image above that of the inversion recovery derived image for subject 8. Fat located in the soft palate is indicated by the arrowhead on the spin echo derived image.

% of ideal body weight	CSA of fat deposits (cm ²)	Normal subjects	Patients with OSA	% of ideal body weight	CSA of fat deposits (cm ²)
152	4.40			153	7.11
144	1.54			143	5.30
132	3.05			134	6.88
126	3.99			126	3.80
107	2.97			118	4.02
				116	3.87

Fig. 7. - This figure shows a representation of the sites and sizes of fat deposits located around the retropalatal airspace (within a 4 cm radius of its centre) from scans taken at the level of the inferior surface of the hard palate. The airspace (centre) and the fat deposits surrounding it (shaded regions) are shown. For each subject, the cross-sectional area (CSA) of the fat deposits and the percentage (%) of ideal body weight are shown beside each trace. Control subjects (numbers 1-5, as in table 1) are shown on the left of the figure and are orientated from top to bottom, whilst the patients with OSA (numbers 6-11) are shown on the right in the same orientation.

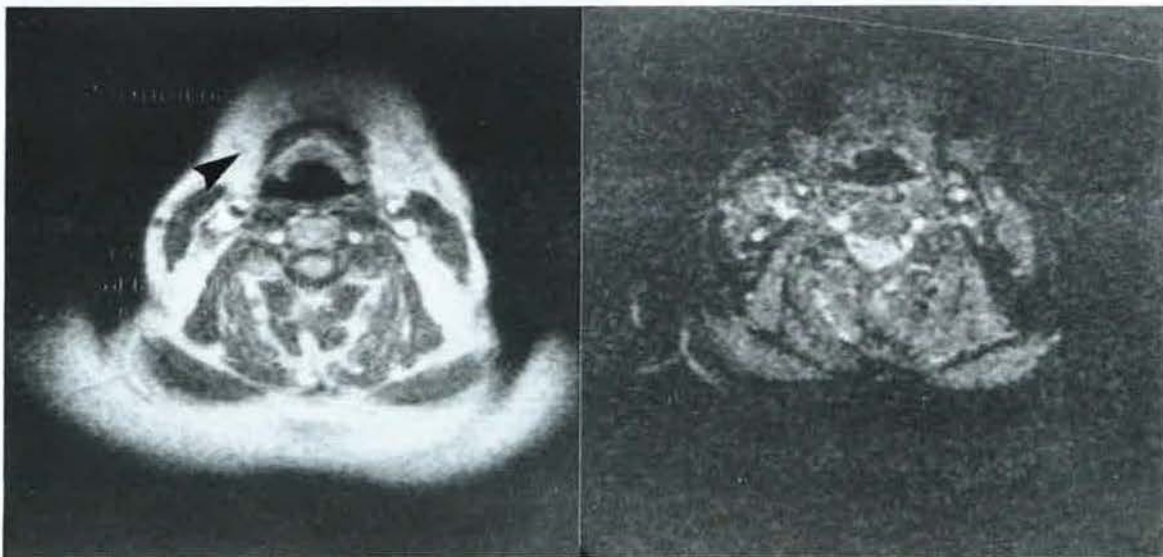


Fig. 6. - The pair of transverse sections in this figure are taken from the level of the submental laryngopharynx in subject 6. The images are presented in the same way as those in figure 2. Large amounts of fat are located lateral and anterior to the laryngopharyngeal airspace (see arrowhead on the spin echo derived image).

Laryngopharynx. In all obese subjects, fat was observed anterior to the laryngopharynx airspace in the submental regions (see figure 6).

Sizes of fat deposits and distribution about the palatopharyngeal airspace

The distribution of fat deposits around the palatopharyngeal airspace at the level of the inferior margin of the hard palate (see figure 1 for reference) is shown in figure 7. In the regions depicted in this figure, significantly more fat was located around the retropalatal airspace in the patients with OSA compared to the control group ($p=0.04$, see table 1). The sizes of the fat deposits located around the retropalatal airspace at the other levels of the palatopharynx are also shown in table 1. The data show that at two of the three anatomical levels of the palatopharynx that were analysed, more fat was present postero-lateral to the retropalatal airspace in the patients with OSA compared to the group of control subjects. Furthermore, the mean amount of fat located around this airspace in the palatopharyngeal region was also significantly increased in the patients with OSA ($p=0.03$, see table 1). There was a tendency for more fat to be located around the retropalatal airspace in the more obese subjects, compared to the less-obese subjects (see figure 7), although an association between the degree of obesity and size of the fat deposits, at any level of the palatopharynx, did not reach statistical significance (all Spearman's rank correlation coefficients, $[r_s]<0.47$, all $p>0.14$, $n=11$).

Discussion

The significance of the present study with magnetic resonance (MR) imaging is that it has been possible to show the anatomical sites of fat deposition around the pharynx in a group of obese subjects, some of whom have OSA. In addition, we have been able to make an attempt at quantifying the size of the fat deposits, at least at the level of the pharynx that tends to close in OSA.

The significance of this study, in relation to the understanding of the pathogenesis of OSA, is that there appears to be more fat in those areas surrounding the collapsible segment of the pharynx in the patients with OSA. Such fat deposits may predispose the upper airspace to collapse during sleep by compromising upper airway muscle function directly, or, indirectly, by making a narrowed airspace more dependent on their activity for patency. This study fails to prove that any of the fat deposits demonstrated are responsible for the pathogenesis of OSA; it is not yet known if one or more of the deposits described in this paper shrinks or disappears at the time when OSA improves by dieting or weight loss.

The validity of this study depends on the ability to reliably identify fat deposits from MR images. To this end, two imaging sequences have been used; the "inversion recovery" sequence excludes fat from the derived

image and these images are compared to the "spin echo" derived images in which the signal due to fat is not excluded. For each subject, the efficacy of the inversion recovery technique in the exclusion of the signal due to fat was tested by looking for its exclusion from sites of known fat deposition, *i.e.* subcutaneous sites. The imaging protocol used in the present study produced reliable and consistent exclusion of this signal from such sites. Since the relaxation properties of hydrogen nuclei in fat are not influenced by its anatomical location [19] then the imaging sequence used in the present study is assumed to be equally effective for the exclusion of the signal due to fat in other regions, *i.e.* closer to the pharyngeal airspace.

Selection of control subjects

The aim of this study was to determine sites/sizes of fat deposits around the pharynx in a group of patients with OSA and to compare such sites/sizes with those from an appropriately matched control group. The control subjects were well matched to the patients with OSA with respect to the degree of obesity, but they were significantly younger (table 1).

This discrepancy arose because of the difficulty in finding more elderly obese subjects who do not snore or have evidence of OSA. Clearly such a group would act as more appropriate controls, particularly because it seems possible that more fat could be deposited around the pharynx as a subject gets older. However, the relevance of this apparent limitation to our study is unknown at present because we are unaware of any data relating ageing to fat deposition around the head and neck.

Quantification of data

The aim of this study was not only to describe sites of fat deposition but also to quantify their size. Since MR imaging is insensitive to increases (or decreases) in the amount of triglycerides within regions of known fat deposition [20] then the intensity of the signal due to fat cannot be used as a measure of the "amount" of fat within that region. However, boundaries of fat deposits can be clearly identified with MR imaging (figures 2 and 3) and the quantification of such deposits has therefore been made with respect to the cross-sectional areas measured from the transverse sections. These measurements have been made only at the level of the palatopharynx where the boundaries of the fat deposits were readily identifiable and were clearly defined (unlike those in submental regions). The cross-sectional areas of the palatopharyngeal fat deposits were calculated within an arbitrarily defined region of interest; the size of the region was the same for all subjects and was chosen to include that region extending from the centre of the airspace to the bony projections of the maxillae. This region was thought to include those fat deposits which may possibly influence the adequacy of the pharyngeal airway as a conduit for airflow.

The relationship between OSA and obesity

OSA is often accompanied by obesity and weight loss is of benefit in treatment [6, 7]. Furthermore, it has been suggested that obesity is a primary aetiologic factor in the development of OSA [6]. However, in patients with OSA who have no obvious cranio-facial abnormalities that may predispose the upper airspace to collapse during sleep [1–4], the mechanisms underlying such an association are unclear. The following account considers how the sites of fat deposition observed in the present study may predispose the upper airspace to collapse during sleep in obese subjects.

Sites of fat deposition and their possible functional role in OSA

Deposits of fat located lateral to the maxillary sinuses (figure 2) are separated from the nasal airspace by the nasal bone. Hence, these deposits will not compromise nasal airway patency nor would they be expected to play a role in the development of upper airway obstruction during sleep.

Fat deposits located at the level of the oropharynx may have implications for the development of OSA since upper airway obstruction during sleep is almost always located at this level (10, 21–24). Previous studies with CT have failed to identify abnormal fat deposits immediately around the oropharyngeal airspace in a group of obese patients with OSA [9, 10]. However, these authors only looked at the tissue immediately surrounding the airspace and at sites of airway narrowing. The present study with MR confirms but extends these findings. This study shows that deposits of fat are present at sites spatially removed from the edge of the oropharyngeal airspace, but it is still conceivable that these deposits could play a role in the genesis of OSA in obese subjects.

Palatopharyngeal fat deposits. Fat deposits located postero-lateral to the airspace were present at the level of the palatopharynx although they were not detectable at the level of the glossopharynx (figures 3 and 4). Fat deposits located at this level are of interest regarding the pathogenesis of OSA since upper airway obstruction is almost always observed at this level of the oropharynx during obstructive apnoeas [10, 21–23] and because at two of the three levels of the palatopharynx at which the sizes of these deposits were measured, significantly more fat was present in the patients with OSA.

Excess fatty accumulations at the level of the palatopharynx may cause displacement of soft tissues, by simple mechanical effects, causing narrowing of the retropalatal airspace during wakefulness and so predisposing this airspace to collapse during sleep. Such a mechanism could explain previous observations that the retropalatal airspace is narrower in awake, obese patients with OSA compared to less obese control subjects [9, 10]. However, it is apparent from figure 7 that, at the level of the inferior surface of the hard palate, the airspace tended to be larger than that of the controls in 3 of the patients

with OSA (numbers 6, 8 and 9). However, this was not apparent from those images taken from lower levels of the palatopharynx. This anomaly may be due to the fact that at that one level of the palatopharynx the size of the airspace is very variable because the soft palate curves and descends toward the oropharyngeal cavity (figure 1) and the soft palate may “hang down” more into the oropharyngeal cavity in those 3 patients with OSA. This speculation is supported by the observation that patients 6, 8 and 9 were also 3 of the 4 patients in whom fat was observed in the soft palate.

Another possible mechanism by which fat deposits located at the level of the palatopharynx may predispose the upper airspace to collapse during sleep is by interference with upper airway muscle function directly, for mechanical reasons, or due to changes in the intrinsic elastic properties of the airway wall and its surrounding structures [25].

Many patients with OSA have a long, bulky and floppy soft palate which may be the result of mechanical injury produced by years of heavy snoring, leading to airway narrowing by loss of tissue tone, oedema and interference with the effectiveness of the soft palate elevator muscles by disruption of the muscle-tissue linkage [26]. However, the soft palate may also be bulky because of the presence of fat. In the present study there was evidence of deposits of fat in the soft palate in the majority of the patients with OSA but none in those of control subjects. Deposits of fat in the soft palate may predispose the retropalatal airspace to collapse during sleep by reducing the size of this airspace (below the level of the inferior surface of the hard palate) and/or by making the soft palate more dependent on palatal muscle activity for maintaining upper airway patency [27].

Glossopharyngeal fat deposits. In patients with OSA, the glossopharyngeal airspace has not consistently been observed to be a site of upper airway narrowing during wakefulness [9, 10, 28]. The lack of fat deposits located postero-lateral to this airspace may reflect these observations.

Deposits of fat in the tongue were observed in normal subjects and the patients with OSA, these deposits were usually located at the level of the glossopharynx. The role of these fat deposits were usually located at the level of the glossopharynx. The role of these fat deposits in predisposing this airspace to collapse during sleep is unclear, although they may increase the dependence of the tongue on pharyngeal dilator muscle activity (particularly genioglossus) for the maintenance of upper airway patency.

Laryngopharyngeal fat deposits. The severity of obstructive apnoeas during sleep shows sleep position dependence, the apnoeas being more frequent with the patient in the supine position ([29], personal observation). REMMERS *et al.* [30] proposed that in the supine subject the weight of the tongue superimposed on a decreased motor drive during sleep was responsible for upper airway closure during obstructive apnoeas. More recently, KOENIG and THACH [31] have emphasised that the force of gravity

could act by means other than directly on the tongue itself. In an animal model these authors showed that simulation of submental fat collections, with lard-filled bags placed on the anterior surface of the neck, narrowed the internal diameter of the oropharynx, increased upper airway resistance to airflow and made the critical pressure needed to induce upper airway collapse less negative. Furthermore, anecdotal clinical evidence suggests that submental fat collections may indeed contribute to the pathogenesis of OSA; KOOPMAN *et al.* [32] describe a subject who was cured of OSA by removal of a lipoma from the anterior aspect of the neck. In the present study, submental fat collections were observed in all obese subjects. Fat deposits of this type may be another important factor predisposing the upper airspace to collapse during sleep, particularly in the supine sleeping subject.

This study demonstrates that more fat is present in the regions surrounding the collapsible segment of the pharynx in patients with OSA, compared to equally obese control subjects without OSA. These results have implications for the pathogenesis of OSA in obese subjects. However, the definitive experiments of studying the sites and sizes of these fat deposits in a group of non-obese normal subjects and secondly in a group of patients with OSA, before and after improvement by weight loss have yet to be performed.

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Situation et taille des dépôts graisseux péri-pharyngés chez des patients obèses atteints du syndrome d'apnée obstructive et chez des contrôles pairés pour le poids. R. Horner, R. Mohiaddin, D. Lowell, S. Shea, E. Burman, D. Longmore, A. Guz.

RÉSUMÉ: Beaucoup de patients atteints de syndrome d'apnée obstructive du sommeil sont obèses. Il a été suggéré que les dépôts de graisse dans les tissus mous entourant les voies aériennes supérieures pouvaient être un facteur important dans la pathogénie du syndrome d'apnée du sommeil chez les sujets obèses. Aucune donnée expérimentale ne confirme cette vue. Dans cette étude, nous avons utilisé la résonance magnétique (MR) pour déterminer la situation et la dimension des dépôts graisseux autour des voies aériennes supérieures, dans un groupe de patients obèses avec syndrome d'apnée du sommeil occlusive ($n=6$, \bar{x} % du corps idéal 132%, limites 116–153%), et un groupe de sujets-contrôle, pairés pour leur poids, sans syndrome d'apnée du sommeil ($n=5$, \bar{x} % du poids idéal du corps 132%, limites 107–152%). Des coupes transverses contiguës de 7 mm et une coupé sagittale médiane ont été obtenus au niveau du nez, de l'oro- et du laryngo-pharynx. Deux séquences séparées d'imagerie ont été utilisées. La première a révélé les

aspects anatomiques typiques obtenues par résonance magnétique; l'image dérivée de la seconde était similaire, mais le signal "graisse" a été spécifiquement prélevé. Chez chaque sujet, les dépôts graisseux ont pu être observés en comparant des paires d'images obtenus dans les mêmes régions anatomiques. Chez tous les sujets, d'importants dépôts de graisse sont présents dans des régions distantes dans l'espace de la crête de l'espace aérien oro-pharyngé. Ces dépôts étaient localisés postéro-latéralement par rapport à l'espace aérien à hauteur du voile du palais. Des quantités de graisse significativement plus importantes sont observées dans ces régions chez les patients atteints de syndrome d'apnée du sommeil ($p=0.03$). De plus, des dépôts graisseux dans le voile du palais ont été observés chez 4 des 6 patients avec syndrome d'apnée du sommeil, mais chez aucun des sujets-contrôle. L'on a noté des traînées graisseuses dans la langue chez 2 des 5 sujets-contrôle et chez 3 des 6 syndromes d'apnées du sommeil. Des dépôts graisseux ont également été observés en avant de l'espace aérien laryngo-pharyngé, dans la région sous-mentonnière, chez tous les sujets obèse. Cette étude a montré que chez les sujets atteints de syndrome d'apnée du sommeil, on trouve plus de graisse dans les régions entourant la partie collabable du pharynx, par comparaison à des sujets-contrôle également obèses mais sans syndrome d'apnée du sommeil. Ces données ont des implications évidentes pour la pathogénie du syndrome d'apnée occlusive du sommeil chez les sujets obèses.

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