BJMP

British Journal of Medical Practitioners

British Journal Of Medical Practitioners

http://www.bjmp.org

Editorial Board

Managing Editors
- Dr J A Latoo
- Dr N Mazi- Kotwal

Medical Editor
- Dr Yaqub Latoo

Editorial Advisory Board
- Dr Leena Ali, Consultant Anaesthetist, UK
- Prof Kenneth Brummel-Smith, Professor of Geriatric & Clinical Medicine, USA
- Dr Saad Ghalib, Consultant Psychiatrist , UK
- Dr Inderjit Gupta, Consultant Physician, UK
- Dr Roop Kaw, Assistant Professor of Internal Medicine, USA
- Dr Manzoor Malik, Family Physician, UK
- Dr Naseer Masoodi, Assistant Professor of Clinical Medicine, USA
- Dr Chris McEvedy, Consultant Psychiatrist, UK
- Dr Ramesh Mehta, Consultant Paediatrician, UK
- Mr Patrick Omotoso, Consultant Surgeon, UK
- Mr J Omoshoro-Jones, Consultant Surgeon, South Africa
- Mr Dilip Patil, Consultant Obstetrician & Gynaecologist, UK
- Dr Abid Rajah, Consultant in Anaesthesia & Critical Care Medicine, UK
- Dr Faisal Salim, Consultant Anaesthetist, UK
- Dr M I Shaikh, Consultant Radiologist, UK
- Mr Harbinder Sharma, Consultant Urologist, UK
- Mr Manoj Sood, Consultant Orthopaedics, UK
- Dr Swati Thakur, Consultant/Attending Physician, USA

Research Advisors
- Dr Sam Tothill, PhD, UK
- Dr M Wasil, PhD, UK

Trainee Editor
- Dr M U Shah

Instructions to authors
Please visit: http://bjmp.org/content/guidance-authors

Submit an article
Please visit: http://bjmp.org/content/submit-articles

Contact us
Please visit: http://www.bjmp.org/contact

Publishers

The British Journal of Medical Practitioners (BJMP) is a quarterly peer-reviewed online international medical journal published by JMN Medical Education Ltd UK. The information, opinions and views presented in the British Journal of Medical Practitioners reflect the views of the authors and contributors of the articles and not of the British Journal of Medical Practitioners or the Editorial Board or its publishers. The British Journal of Medical Practitioners and/or its publisher cannot be held responsible for any errors or for any consequences arising from the use of the information contained in this journal.

British Journal of Medical Practitioners

Editorial

Review Articles

Viewpoint

Case Reports/Case Series

Audit Reports

Miscellaneous

Editorial

LANGUAGE AND PSYCHIATRY: "An argument for indeterminism"

Saad F. Ghalib

"Schizophrenia' is written and spoken about as if the language used simply reflected a reality already discovered or about to be discovered. Such a representational view of language has been strongly questioned in a range of theoretical ideas whose common assumption is that what we think of as reality or truth is not discovered or reported but is constructed, primarily through the strategic use of language" (Boyle 2002).

INTRODUCTION

It is not a revelation, that practising psychiatry or psychotherapy and pursuing psychological research, rely heavily on phenomenology, which may be descriptive (Jasper, Husserl), or dynamic (Freudian), and influences that which may have on several diagnostic categories. The question of how our use of terms correlates with whatever it is trying to describe, requires serious consideration. The aim of this editorial is to sketch some significant developments in psychological, biological, physical and philosophical studies, with specific reference to the role of language in these evolving scientific endeavours.

EARLIER PERSPECTIVES

As early as 1921, Wittgenstein (1889 – 1951) proposed two major ideas that revolutionised philosophical thinking. The first was the principle of verification (that statements are meaningful, only when they can be verified by experiment), which has since been adopted as the manifesto of logical positivism, and the basis of new scientific thinking. His second central thesis was to deny that logical or linguistic concepts represent reality. Furthermore, he suggested that the apparent harmony between language and reality is merely the shadow cast upon the world by grammar. In his major work "The Concept of Mind" (1949), Gilbert Ryle (1900-1976), suggested that the Cartesians (followers of Descartes) have been misled, in picturing the mind as a "ghostly" counterpart of the brain; simply due to our way of expression, when handling "one category as if it belonged to another" (Category Error).

It is undeniable that logical positivism (sentences are meaningful if they can be assessed either by an appeal to sense data or by an appeal to the meaning of the wards and the grammatical structure that constitute them) has lived up to expectations in ridding scientific methodology of metaphysical arbitrance. It also brought a range of new issues under the spotlight, which were previously unrecognised. First, the verification condition for a given Empirical statement presupposes a massive background of default auxiliary assumptions (Duhem, 1954), i.e. all experiments will presume the truth of some theories to help judge that the set-up is adequate and the instruments are reading what they are meant to read. But these presupposed theories need not be identical to the theory under test. Second, the long held dichotomy between Priori statements (true by virtue of meaning), and

Contingent statements (true by empirical evidence) is no longer tenable, and that neither is shown to be immune to revision at some point in time (Quine, 1961). Furthermore, single terms in scientific theories are meaningful only on their place in the theory.

DOES REDUCTIONISM HELP?

Although a reductionist approach (describing a phenomenon in relation to its constituent parts) has been traditional in biology, there has been some reluctance to apply reductionism to the study of human behaviour. However, it was precisely the assumption that elementary forms of learning are common to humans and simple animals, that consequently led to the discovery of the cellular and molecular basis of memory and learning (Kandel, 2000).

On the other hand, the common misconception, even in textbooks of genetics is to speak of genes determining traits of the whole organism, as if identifying a gene will mean the trait of the organism is known. If one examines the more general relation between gene, environment and organism, it is apparent that the situation is more complex. First, there is no unique phenotype corresponding to a genotype; the phenotype depends on both genotype and environment. Second, the form and direction of the environment's effect upon development differs from genotype to genotype. Third, and reciprocally, there is no unique ordering of genotype such that one can always be characterized as "superior" or "inferior" to another. (Levins and Lewontin, 1985).

Even with reductionist sciences like physics, the view held is "that physics is not about how nature is. Physics concerns what we can say about nature" (Bohr, in Peterson, 1963). This view recently echoed by Hawking (New Scientist, 2003), where he suggested that our deepest theories rely on our language of logics, which is self-referential, and cannot be complete and consistent at the same time. In simple terms, there is an eternally unbridgeable gap between what is true within a given logical framework or system and what we can actually prove by logical means using that same system. Obviously, this may open the way to confusion and paradoxes, as causality within the system cannot be determined.

PSYCHOANALYSIS BEING ANALYSED

Wittgenstein makes serious criticism of determinism in psychodynamic theories. When Freud says, "he could not believe that an idea produced by the patient could be an arbitrary one and unrelated to the idea we were in search of". He is apparently making a category error by mixing two different statements: "Everything has a meaning" (can be interpreted) is not "Everything has a cause" (Bouveresse, J 1995). The person who agrees with us about the way things had to happen "suddenly sees the cause". This, however, neither constitutes causality, nor can be empirically tested.

There is also the unjustifiable assumption that if meaning can be given to some mental events, it must be possible to assign meaning to all such events, even if it hasn't yet been found. The latter might explain the common characterization of Freudian theories as pseudoscientific.

Curiously, one may well reasonably argue (due to lack of clear causality) that both patient and therapist, having reached different explanation of the same behaviour, are entirely justified, within the context of their own paradigm of thinking.

LANGUAGE ACQUISITION AND PERCEPTION

An influential framework of language acquisition, where knowledge of language is mentally represented as "grammar" (a finite system of rules) and the fundamental properties of these grammars are part of innate endowment was first proposed by Chomsky (1965). Chomsky's ideas provide some explanation for our tendency to formulate premature theories (including scientific ones) on weak and limited evidence. Furthermore, when one considers both the evidence of how young children use language, and of how they understand it, there is often a lack of accord between the two (Huttenlocker, 1974). Interestingly, the interpretation of terms (language) is significantly dependent on the context of occurrence (the situations that it is used in) (Macnamara, 1972).

Studies of models of colour vision, support a wealth of evidence that what people treat as the same or as different depends on what language they speak. Furthermore, in the perception of space, language categories significantly mould thought and behaviour in a striking way (Scientist American, April 2004).

One may conclude from the above that our emotions, perceptions and theorizing are constrained by the limits of our own language. It is worth noting that the Epistemological limitations imposed by our language is not fixed in time but rather continuously change as we endlessly renegotiate our notion of reality as our language and our life develops (Putnam, 1994). Others went further to suggest that assigning diagnostic labels to human behaviour may even be more dramatic because people classified in a certain way, change in response to being classified (Hacking, 1999). Hence, it may be fair to say that diagnostic labels do not register the value of some passive attribute but of an attribute that is determined in part by our own actions in the process of ascribing labels to these attributes.

IN CONCLUSION

Francis Crick's (1979) remark that "we are deceived at every level by our introspection" may well be more appropriately applied to our capacity to use language. This is especially relevant when language is employed in describing human behaviour, psychiatric diagnostic categories and neurophysiological studies. By virtue of its logical incompleteness, self referentiality and the context in which it is employed, language may lead to erroneous interpretations, would that be a therapeutic session, a psychiatric diagnosis, or even describing the microscopical functions of a nerve cell.

One is not suggesting how these issues could be remedied (that would require another editorial). However, it cannot even be overestimated that, although diagnostic categories assist our every day clinical work, a detailed analysis of the limitations and complexity of our language would facilitate understanding of our patients, and lead to fruitful scientific research.

AUTHOR DETAILS
SAAD F. GHALIB, MRCPsych, Consultant Old Age Psychiatrist, South Essex Partnership Trust, United Kingdom
CORRESSPONDENCE: Dr S F Ghalib, Consultant Old Age Psychiatrist, Mental Health Unit, Basildon University Hospitals, Basildon SS16 5NL.
Email: Saad.Ghalib@southessex-trust.nhs.uk

REFERENCES

1. Boyle, M (2002): Schizophrenia. A scientific delusion? 2nd Edition, Routledge
2. Wittgenstein, L.J: Tractatus logico-philosophicus, trans. D.F. Years and B. McGuiness (Routledge and Kegan Paul, 1961)
3. Ryle, G. (1949). The Concept of Mind. London
4. Duhem, P. (1954). The Aim and Structure of Physical Theory, New York, Doubleday.
5. Quine, W.V.D. (1961). Word and Object, Cambridge, Mass., MIT press
6. Kandel, E. (2000) – Nobel Lecture www.nobel.se/medicine/laureates/2000/kandel-autobio.html
7. Levins, R. Lewontin, R. (1985). The Dialectical Biologist. Harvard University press p.90-93.
8. Neils Bohr, quoted in A. Peterson, "The Philosophy of Neils Bohr" Bulletin of the atomic Scientist. Sept 1963, p.12
9. Stephen Hawkins, quoted in the "New Scientist" 5 April 2003, p.34 – 35.
10. Bouveresse, J. (1995) "Wittgenstein Reads Freud, The myth of the Unconsious" Trans. Carol Cosman. (Princeton) p.88 – 96.
11. Chomsky, N. (1965). "Knowledge of Language: Its Nature, Origin and Use". New York.
12. Huttenlocker, J. (1974). "The Origin of Language Comprehension". In Solso, R.C. (Ed), Theories in Cognitive Psychology. Hillsdale, New Jersey.
13. Macnamara, J. (1972). Cognitive Basis of Language Learning in Infants. Psychological Review, 79, 1 – 13.
14. "Scientific American" April 2004, p.24 – 25.
15. Putnam, H. (1994). "Sense, Nonsense and the Senses: An Inquiry into the Powers of the Human Mind". The Journal of Philosophy 91: 445 – 517.
16. Hacking, I. (1999). "The Social Construction of What?" Harvard University Press. P.100 – 124.
17. Crick, F.H.C. (1979). Thinking about the Brain. Scientific American 241: 219 – 232.

Review

POLYPHARMACY: TO ERR IS HUMAN, TO CORRECT DIVINE

Nasseer A. Masoodi

ABSTRACT

Objectives
Optimizing drug therapy for elderly is often challenging. Sometimes treatment causes more harm than the disease. The aim of this article is to review the body of literature addressing polypharmacy to determine its definition, explore how polypharmacy was assessed in primary care, and seek evidence based interventions that address polypharmacy.

Data Sources
An electronic search of the PUBMED database utilizing the search terms "polypharmacy," "polypharmacy and elderly," "adverse drug reactions," "multiple medications", "inappropriate prescribing", and "Beers criteria" was performed and the search was supplemented with online site searches of relevant journals and review of reference lists of each article.

Results and discussion
Prescription of potentially inappropriate medications to older people is highly prevalent in the United States and Europe. Polypharmacy continues to be a significant issue. There is a gap in the literature regarding the interventions implemented by physicians to address polypharmacy. There are no robust prospective studies that test the clinical benefit to patients of using drug utilization review tools.

Conclusion
There is no specific definition for polypharmacy. It has been defined in many different ways depending upon patient population and study settings. Prospective randomized controlled trials are needed to identify useful interventions. Drug utilization review tools should be designed on the basis of a country's national drug formulary and should be evidence based as most existing drug utilization review tools have been designed on the basis of North American system.

INTRODUCTION:

Population demographics are changing worldwide, with life expectancy and the proportions of older persons increasing. Older people are the greatest consumers of medications and healthcare resources in developed countries. It is assumed that as more drugs become available and life expectancy continues to increase, the consumption of prescription drugs by older people will increase further and the incidence of potentially inappropriate prescribing will grow. A survey of non-institutionalized older adults in the United States showed an increased usage of all medications with advancing age, the highest prevalence of drug use being in women 65 years of age and older with 12% taking 10 or more medications and 23% taking at least five prescribed drug therapies [1]. In most industrialized nations older people consume three times as many prescription medications as younger people and purchase 70% of non-prescription medications [2]. In the United States, 12·5% of the population is over 65 years of age but consume 32% of all prescription medications and account for 25% of drug expenditure and 30% of total national healthcare expenditure [3-5]. In Ireland, 11·13% of population is over the age of 65 years but consume 47% of all prescription medications [6]. In Europe, people over 65 years of age consume

on average 2·3 times the amount of health care than do those <65 years of age [7].

POLYPHARMACY:

Polypharmacy has been defined in many different ways and the appropriate definition may differ according to patient population and study setting [9]. Fulton and Allen [10] define polypharmacy as: 'the use of medications that are not clinically indicated'. In practice, polypharmacy is defined as using more than a certain number of drugs, irrespective of the appropriateness of drug use [8, 11, 12]. Inappropriate prescribing includes the use of medicines that introduce a significant risk of an adverse drug-related event where there is evidence for an equally or more effective but lower-risk alternative therapy available for treating the same condition. Inappropriate prescribing also includes the use of medicines at a higher frequency and for longer than clinically indicated, use of multiple medicines that have recognized drug–drug interactions and drug–disease interactions, and importantly, the under-use of beneficial medicines that are clinically indicated but not prescribed for some reasons. As older patients seek treatment for various ailments from a variety of physicians, they are at increasing risk of accumulating layers of drug therapy. Individuals aged 65 and older use a disproportionate number of

prescriptions and over-the-counter medications; 31% use more than one pharmacy and 50% receive prescriptions from more than one prescriber [13]. A higher number of primary care physicians and multiple dispensing pharmacies increase the risk of drug–drug interactions [14]. The number of medications prescribed to elderly patients, and the complexity of their drug regimens increase over time [15].

The potential for an increased risk of drug–drug interactions and adverse drug reactions, and factors such as age-related changes in pharmacodynamics (PD) and pharmacokinetics (PK) must be considered. Diabetes and chronic lung disease predict a greater complexity and cost of drugs regimen in elderly patients with heart failure [16]. Besides the increase in diseases and worsening of diseases, the literature also mentions other factors as being responsible for the increase in polypharmacy, i.e. ageing, moving to a residential or nursing home and hospitalization [17, 18]. The patient's expectations, the General Practitioner's attitude and consultations with several doctors have been associated with an increase in multiple drug use [19, 20].

EFFECTS OF AGING ON DRUG METABOLISM:

Drug absorption, distribution, metabolism and elimination change as a natural consequence of the ageing process. Changes in drug absorption in older patients may result from decreases in splanchnic blood flow and gastric motility, and increases in gastric pH, and other physiological changes that are associated with ageing. Blood flow and gastric motility may be further diminished by cardiovascular and gastrointestinal drugs used to treat co-morbid conditions. Ageing influences drug excretion. Age-related decreases in glomerular filtration rate are well known. These physiological declines coupled with co-morbid conditions and the use of multiple drugs means that medications eliminated by the renal route requires dose adjustment. Drugs that influence renal function and thus elimination/excretion have the potential to pose serious clinical problems if used concomitantly. With ageing, there is a decrease in lean body mass and total body water with a relative increase in total body fat [21]. These changes lead to a decreased volume of distribution for hydrophilic drugs such as lithium, and digoxin where unadjusted dosing can result in higher plasma concentrations, thus increasing the potential for adverse effects. Conversely, lipid soluble drugs such as long-acting benzodiazepines have an increased volume of distribution, thereby delaying their maximal effects and resulting in accumulation with continued use. There is a reduction in hepatic mass and blood flow with ageing [22].

Drugs such as beta-blockers, nitrates and tricyclic anti-depressants that have a first pass effect in the liver may have a higher bioavailability in older people and thus be effective at lower doses. Cytochrome P450 oxidation declines with ageing [24] and drug–drug interactions involving these enzymes are important to recognize. Larger drug storage reservoirs and decreased clearance prolong drug half-lives and lead to increased plasma drug concentrations in older people. If serum albumin is decreased there will be an increase in the active unbound drug concentration for highly protein-bound drugs such as phenytoin, theophylline, warfarin and digoxin. Ageing is also associated with changes in the end-organ responsiveness to drugs at receptor or post-receptor level [25]. There is decreased sensitivity to beta-receptors along with a possible decreased clinical response to beta-blockers and beta-agonists [26]. Increased sensitivity to drugs such as opiates and warfarin is common [27, 28].

ADVERSE DRUG REACTIONS (ADRs):

The number of elderly is increasing dramatically. In United States, in the next 25 years, as the baby boomer generation begins to turn 65 years old, the number of elderly is expected to double to approximately 70 million. Those older than 85, is now the fastest growing segment of our population. Thus, we can expect the number of adverse drug reactions to increase proportionately. Polypathology, the age-related increase of concurrent diseases, is likely to be the main determinant of drug consumption. However, both over-prescribing and improper prescribing has been reported and seems to contribute to the age-related increase in the prevalence of adverse drug reactions (ADRs) [29, 30]. A hospital-based study from Norway showed that the risk of experiencing a drug-related problem increased linearly with the number of drugs on admission [31]. A study carried out in the USA found that nursing home patients receiving nine or more drugs were more than twice as likely as patients receiving a lower number of drugs, of experiencing an adverse effect [32]. On average, ADRs account for 3%–13% of all the admissions [33-35] and complicate 5%–20% of the stays of patients over 65 years [36-38]. More than 40% of persons aged 65 and older use five or more different medications per week, and 12% use 10 or more different medications [39]. If an elderly patient takes five or more drugs, he or she has a 35% chance of experiencing an adverse drug event [40].

Drug interactions are significant contributors to morbidity [35]. Office visits for an adverse drug event increase from 9% of the population per year at age 25–44 years to as high as 56·8% between age 65 and 74 years [41]. Inappropriate drug use is one of the risk factors for adverse drug reactions in the elderly. The risk for an adverse drug event is 13% with the use of two medications, but the risk increases to 58% for five medications [42]. If seven or more medications are used, the incidence of adverse drug events increases to 82% [42].

INTERVENTIONS:

Older people are a heterogenous group, often with multiple concomitant illnesses and multiple prescriptions. There is a thin line between a healthy old person and an ill old person. Prescribing for older people is challenging as any new medication must be considered in the context of altered pharmacokinetics, altered pharmacodynamics and age-related changes in body composition and physiology. Both over prescription and undue prescription seem to characterize the overall pharmacological therapy of the elderly.

Polypharmacy is the main risk factors for ADRs [43]. Thus, attempts should be made to curtail inappropriate drug prescription by utilizing different available tools [44]. An interdisciplinary medication review of older individuals in the community helps to reduce the cost and number of medications. Polypathology seems the most obvious

explanation of the high number of drugs taken by older people, but additional factors deserve consideration. Changes in patient's medical status over time can cause medications that have been used chronically to become unsafe or ineffective. Particular care must be taken in determining drug dosages and treatment options when prescribing for older adults. "Pill for an ill" approach should be discouraged as many a time pharmacological treatment may carry more adverse effects then the illness itself. Use of electronic medical records and other hand held devices to prescribe appropriate medication doses and check drug to drug interactions has been found useful in reducing the medication related errors and hence adopted by various medical groups and hospital practices.

Reviewing medications at every visit is a simple and very helpful tool too especially if patients are encouraged to bring with them a printed list of their current medications (including over the counter drugs). Printing an updated list of the medication changes in bold and large font after a visit with their physician helps patients to follow the recommendations especially in case of geriatric patients who may not remember all the new changes made at an office visit.

CONCLUSIONS:

Polypharmacy is an important issue in the elderly. The problem involves many issues, a number of which have been explored in this article. One of the most important issues involves adverse drug reactions. All pharmaceutical agents have the potential for side effects; therefore, it is obvious that the more drugs one takes the more side effects one will experience. The aging process results in altered metabolism and excretion of medications, and deficits in cognition and senses. Incidence of adverse drug reaction and interactions is increased with polypharmacy. Since adverse drug reactions are a significant cause of morbidity and mortality, as well as an important cause for hospital admissions, minimizing polypharmacy is an important consideration. The general principle of "Start Low and Go Slow" holds true in most scenarios but should be modified to "Start Low, Go Slow but Use Enough" to achieve desired therapeutic effect.

AUTHOR DETAILS
NASSEER A. MASOODI, MD, FACP, CMD. Assistant Professor Clinical Sciences, Florida State University College of Medicine, Tallahassee, FL-USA; Courtesy Assistant Professor Geriatrics, University of Florida College of Medicine, Gainesville, FL-USA; Medical Director Health Services, ACV Inc, Dowling Park, FL-USA

CORRESSPONDENCE: PO BOX: 4346, Dowling Park, FL-32064, USA.
Email: haadin@yahoo.com

REFERENCES

1. Kaufman DW, Kelly JP, Rosenberg L et al. (2002) Recent patterns of medication use in the ambulatory adult population of the United States: The Slone Survey. JAMA, 287, 337.
2. Christchilles EA, Foley DJ, Wallace RB et al. (1992) Use of medications by persons 65 and over: data from the established populations for epidemiologic studies of the elderly. Journal of Gerontology, 47, 137–144.
3. Arnett RH III, Blank LA, Brown AP et al. (1990) National Health Expenditures 1988. Office of National Cost Estimates. Health Care Financing Review, 11, 1–41.
4. Gupta S, Rappaport HM, Bennett LT (1996) Inappropriate drug prescribing and related outcomes for elderly Medicaid beneficiaries residing in nursing homes. Clinical Therapeutics, 18, 183–196.
5. Golden AG, Preston RA, Barnett SD et al. (1999) Inappropriate prescribing in homebound older adults. Journal of the American Geriatrics Society, 47, 948–953.
6. Barry M (2002) Drug Expenditure in Ireland 1991–2001. IMJ, 95, 45.
7. O'Connor K, O'Mahony D (2003) Drugs and ageing. In: Liston R, Mulkerrin EC, eds. Medicine for older patients: cases and practice. Dublin, Ireland: Eireann Healthcare Publications, 205.)
8. Nguyen JK, Fouts MM, Kotabe SE, Lo E (2006) Polypharmacy as a risk factor for adverse drug reactions in geriatric nursing home residents. The American Journal of Geriatric Pharmacotherapy, 4, 36–41.
9. 9. Rollason V, Vogt N (2003) Reduction of polypharmacy in the elderly: a systematic review of the role of the pharmacist. Drugs and Aging, 20, 817–832.
10. Fulton MM, Allen ER (2005) Polypharmacy in the elderly: a literature review. Journal of the American Academy of Nurse Practitioners, 17, 123–132.
11. Koh Y, Fatimah BM, Li SC (2003) Therapy related hospital admission in patients on polypharmacy in Singapore: a pilot study. Pharmacy World and Science, 25, 135–137.
12. Mamun K, Lien CT, Goh-Tan CY, Ang WS (2004) Polypharmacy and inappropriate medication use in Singapore nursing homes. Annals of the Academy of Medicine, Singapore, 33, 49–52.
13. Safran D, Neuman P, Schoen C et al. (2005) Prescription drug coverage and seniors: findings from a 2003 national survey. Health Affairs (Millwood), Jan–Jun, Supplemental Web Exclusive: W5-152–W5-166.
14. Becker ML, Kallewaard M, Caspers PW, Schalekamp P, Stricker BH (2005) Potential determinants of drug–drug interactions associated with dispensing in community pharmacies. Drug Safety: An International Journal of Medical Toxicology and Drug Experience, 28, 371–378.
15. Cherry DK, Woodwell DA (2002) National Ambulatory Medical Care Survey: 2000 summary. Advance Data, 328, 1–32.)
16. Masoudi FA, Baillie CA, Wang Y, et al. The complexity and cost of drug regimens of older patients hospitalized with heart failure in the United States, 1998–2001. Arch Intern Med. 2005; 165:2069–76.
17. Hale WE, May FE, Marks RG, Stewart RB. Drug use in an ambulatory elderly population: a five-year update. Drug Intell Clin Pharm 1987; 21: 530–535.
18. Simons LA, Tett S, Simons J et al. Multiple medication use in the elderly. Use of prescription and non-prescription drugs in an Australian community setting. Med J Aust 1992; 157: 242–246
19. Sharpe TR, Smith MC, Barbre AR. Medicine use among the rural elderly. J Health Soc Behav 1985; 26: 113–127.
20. Williams P, Rush DR. Geriatric polypharmacy. Hosp Pract 1986; 21: 109–120
21. Meyer BR (2003) Clinical pharmacology and ageing. In: Grimley Evans J, Franklin Williams T, Lynn Beattie B, Michael J-P, Wilcock G, eds. Oxford textbook of geriatric medicine, 2nd edn. Oxford: Oxford University Press, 127–136.
22. Woodhouse KW, Ewynne HA (1988) Age related changes in liver size and hepatic blood flow: the influence of drug metabolism in the elderly. Clinical Pharmacokinetics, 15, 328–344.

23. Hunt CM, Westerkam WR, Stave GM et al. (1992) Hepatic cytochrome P-4503A (CYP3A) activity in the elderly. Mechanisms of Ageing and Development, 64, 189.

24. Hunt CM, Westerkam WR, Stave GM (1992) Effect of age and gender on the activity of human hepatic CYP3A. Biochemical Pharmacology, 44, 275.

25. Vestal RE, Wood AJJ, Shand DG (1988) Adrenoreceptor status and cardiovascular function in ageing. Journal of Hypertension, 6(Suppl. 1), S59–S62

26. Vestal RE, Wood AJJ, Shand DG (1979) Reduced beta adrenoceptor sensitivity in the elderly. Clinical Pharmacology and therapeutics, 26, 181–186.

27. Gage BF, Fihn SD, White RH (2000) Management and dosing of warfarin therapy. American Journal of Medicine, 109, 481.

28. Beers MH, Berkow R eds (2000) Clinical Pharmacology. The Merck manual of geriatrics, 3rd edn. White house Station, NJ: Merck Research Laboratories, 54–74.)

29. Onder G, Pedone C, Landi F, et al. Adverse drug reactions as cause of hospital admissions: results from the Italian Group of Pharmacoepidemiology in the Elderly (GIFA). J Am Geriatr Soc. 2002; 50:1962–8.

30. Chang CM, Liu PY, Yang YH, et al. Use of the Beers criteria to predict adverse drug reactions among first-visit elderly outpatients. Pharmacotherapy. 2005;25:831–8.

31. Viktil KK , Blix HS, Moger TA, Reikvam A (2007) Polypharmacy as commonly defined is an indicator of limited value in the assessment of drug-related problems. British Journal of Clinical Pharmacology, 63, 187–195

32. Nguyen JK, Fouts MM, Kotabe SE, Lo E (2006) Polypharmacy as a risk factor for adverse drug reactions in geriatric nursing home residents. The American Journal of Geriatric Pharmacotherapy, 4, 36–41

33. Atkin PA, Shenfield GM. Medication-related adverse reactions and the elderly: a literature review. Adv Drug React Toxicol Rev. 1995; 14:175–91.

34. Mannesse CK, Derkx FH, de Ridder MA, et al. Contribution of adverse drug reactions to hospital admission of older patients. Age Ageing. 2000; 29:35–9.

35. Pirmohamed M, James S, Meakin S, et al. Adverse drug reactions as cause of admission to hospital: prospective analysis of 18 820 patients. BMJ. 2004; 329:15–19.

36. Onder G, Gambassi G, Scales CJ, et al. 2002. Adverse drug reactions and cognitive function among hospitalized older adults. Eur J Clin Pharmacol, 58:371–7.

37. Somers A, Petrovic M, Robays H, et al. 2003. Reporting adverse drug reactions on a geriatric ward: a pilot project. Eur J Clin Pharmacol, 58:707–14.

38. Corsonello A, Pedone C, Corica F, et al. 2005. Concealed renal insufficiency and adverse drug reactions in elderly hospitalized patients. Arch Intern Med, 165:790–5.

39. Kaufman DW, Kelly JP, Rosenberg L, Anderson TE, Mitchell AA (2002) Recent patterns of medication use in the ambulatory adult population of the United States: the Slone survey. The Journal of the American Medical Association, 287, 337–344.

40. Lazarou J, Pomeranz BH, Corey PN (1998) Incidence of adverse drug reactions in hospitalized patients: a meta-analysis of prospective studies. The Journal of the American Medical Association, 279, 1200–1205.

41. Zhan C, Arispe I, Kelley E, Ding T, Burt CW, Shinogle J, Stryer D (2005) Ambulatory care visits for treating adverse drug effects in the United Sates, 1995–2001. Joint Commission Journal on Quality of Patient Safety, 31, 372–378

42. Prybys, K., Melville, K., Hanna, J., Gee, A., & Chyka, P. (2002). Polypharmacy in the elderly: Clinical challenges in emergency practice: Part 1: Overview, etiology, and drug interactions. Emergency Medicine Reports, 23(11), 145–153.

43. Hajjar ER, Hanlon JT, Artz MB, et al. Adverse drug reaction risk factors in older outpatients. Am J Geriatr Pharmacother. 2003; 1:82–9.

44. Beers, M. (1997). Explicit criteria for determining potentially inappropriate medication use by the elderly: An update. Archives of Internal   Medicine,157(14),1531–1537.

Review

Dementia with Lewy Bodies: Clinical Review

Javed Latoo and Farida Jan

Summary

The aim of this article is to review the diagnosis and management of Dementia with Lewy Bodies. Dementia with Lewy bodies (DLB) is considered the second most common cause of dementia in the elderly after Alzheimer's disease. Diagnostic criteria for DLB is categorised into central feature (progressive dementia), core features(fluctuating cognition, recurrent visual hallucinations and parkinsonism), suggestive features(rapid eye movement sleep behaviour disorder, increased sensitivity to neuroleptics and low dopamine transporter uptake in the brain's basal ganglia) and supportive features(repeated falls , transient loss of consciousness, hallucinations in other modalities, visuospacial abnormalities and autonomic dysfunction). DLB patients have the diffuse presence of Lewy Bodies in both sub cortical and cortical areas of the brain. Patients with DLB also have more severe dopamine and acetylcholine loss as compared to Alzheimer's disease. Cholinesterase inhibitors can be used for the treatment of neuropsychiatric symptoms. Treatment with levodopa-carbidopa combinations should be considered when parkinsonian symptoms cause functional impairment. Antipsychotics should be used with great caution due to increased extra pyramidal adverse reactions. Clonazepam can be helpful to manage REM sleep behaviour disorder.

Clinicians need to be aware of the diagnosis of DLB in order to provide appropriate pharmacological and nonpharmacological treatment for its cognitive, neuropsychiatric, motor and sleep disturbances without causing distressing side effects due to inappropriate drug prescription.

Abbreviations

DLB=Dementia with Lewy Bodies, AD=Alzheimer's disease, PD=Parkinson's disease, REM=Rapid eye movement, SPECT=Single-photon emission computed tomography, PET=positron emission tomography.

INTRODUCTION

Dementia with Lewy bodies (DLB) is considered the second most common cause of dementia in the elderly after Alzheimer's disease. DLB is a progressive neurological disorder characterized by core features of cognitive impairment, psychosis and Parkinsonism. The disease is commonly referred to by a number of names, such as Lewy Body Disease, Lewy Body dementia, dementia with Lewy Bodies, or diffuse Lewy Body Disease. Prevalence estimates of DLB, depending on case criteria, range from 0 to 5% with regard to the general population, and from 0 to 30.5% of all dementia cases [1]. It is claimed that DLB accounts for 20% of late onset dementia [2, 3] .Most studies suggest that DLB is slightly more common in men than in women. DLB is a disease of late middle age and old age. DLB has been described in Asian, African, and European races.

Friederich Lewy discovered abnormal proteins called Lewy Bodies in early1900's These Lewy Body proteins are spherical intraneuronal cytoplasmic inclusions 15-30um in diameter and are found in the brainstem of patients with Parkinson's disease. In DLB, these abnormal proteins are found diffusely throughout other areas of the brain including midbrain and the cerebral cortex. The brain chemical acetylcholine is depleted, causing disruption of perception, thinking, and behaviour. Lewy body dementia shares characteristics with both

Alzheimer's disease and Parkinson's disease. This can lead to difficulty or delay in reaching the right diagnosis of DLB.

CLINICAL FEATURES

First consensus guidelines for diagnosis of DLB were published in 1996 [4] and reviewed in 1999 [5].The latest consensus diagnostic criteria for DLB was agreed in the third report of the DLB consortium in 2005 [6].

COGNITIVE IMPAIRMENT

Prominent memory impairment may not be evident in the early stages. Cognitive features distinguishing DLB from AD are more prominent impairment of attention, executive functioning (e.g., planning, prioritizing, sequencing), and visuospatial problems (such as problems in following an unfamiliar route) [8, 9]. Mental inflexibility, perseveration, and intrusion are more likely with DLB than with AD [10]. Patients with DLB have more difficulties in clock drawing or figure copying as compared to patients with Alzheimer's disease who have more prominent memory changes on mini mental state examination [8, 11-13].

A core feature of DLB is the fluctuation in cognitive performance, which can occur early in the illness. By way of example, one day a patient may be able to hold a sustained conversation, the next they may be drowsy, inattentive and almost mute.

Diagnostic criteria for Dementia with lewy bodies [4-7]

Central feature
- Progressive dementia - deficits in attention and executive function are typical. Prominent memory impairment may not be evident in the early stages.

Core features:
- Fluctuating cognition with pronounced variations in attention and alertness.
- Recurrent complex visual hallucinations
- Spontaneous features of Parkinsonism.

Suggestive features:
- REM sleep behaviour disorder (RBD), which can appear years before the onset of dementia and Parkinsonism.
- Severe sensitivity to neuroleptics occurs in up to 50% of LBD patients who take them.
- Low dopamine transporter uptake in the brain's basal ganglia as seen on SPECT and PET imaging scans.

Supportive features:
- Repeated falls and syncope (fainting).
- Transient, unexplained loss of consciousness.
- Autonomic dysfunction.
- Hallucinations of other modalities.
- Visuospatial abnormalities like depth perception, object orientation, directional sense and illusions
- Other psychiatric disturbances like systematized delusions, aggression and depression.

A probable LBD diagnosis requires either:
- Dementia plus two or more core features, or
- Dementia plus one core feature and one or more suggestive features.

A possible LBD diagnosis requires:
- Dementia plus one core feature, or
- Dementia plus one or more suggestive features.

Data from [4-7]

VISUAL HALLUCINATIONS

Visual Hallucinations are another core feature distinguishing DLB from AD. In DLB, hallucinations are typically recurrent, well formed, and complex and are usually detailed. Patients may see images of people or animals that they recognise. Some patients see coloured patterns or shapes. Presence of hallucinations with substantial fluctuation in attention can lead clinicians to diagnose delirium. Hallucinations are not always distressing to patients and many learn to distinguish between real and unreal images: some people actually come to enjoy them. In many patients visual hallucinations are accompanied by delusions which tend to be persecutory in nature.

PARKINSONISM

Spontaneous features of Parkinsonism are another core feature of DLB. Patients usually present with rigidity, bradykinesia, gait changes, masklike faces [14], reduced arm swing and a tendency to falls. Resting tremor is less common in DLB than in PD. Development of dementia within 12 months of extrapyramidal signs suggests DLB, whereas late development of dementia makes PD with dementia more likely [4]. Patients who have dementia with Lewy bodies tend to respond less favourably to levodopa with carbidopa as compared to patients who have Parkinson's disease with dementia [11, 15].

OTHERS CLINICAL FEATURES

Severe sensitivity to antipsychotics occurs in up to 50% of DLB patients who take them, developing Parkinsonism even if they have not shown such signs before drug administration. The associated Parkinsonism is often prolonged, profound and may even be fatal. REM sleep behaviour disorder occurs in about one half of these patients. REM sleep behaviour disorder usually presents with vivid dreams associated with simple or complex motor behaviour during REM sleep [11]. Diagnosis of DLB is also supported by repeated falls and syncope, transient loss of consciousness hallucinations in other modalities, visuospacial abnormalities and autonomic dysfunction.

PATHOGENESIS

The pathology of DLB closely resembles that of Parkinsonism disease. Patients with DLB are characterised by the diffuse presence of Lewy Bodies in both subcortical and cortical areas of the brain whereas Parkinson's disease patients have lewy bodies in the subcortical areas of the brain mainly substantia nigra and locus cerules [11, 16]. Both DLB and Parkinson's disease are associated with abnormal aggregation of alpha-synuclein which is a nerve terminal protein that is a better marker of lewy bodies than ubiquitin. Biochemically, numerous neurotransmitters, including acetylcholine and dopamine are diminished in DLB. The decrease in acetylcholine may be more severe than in Alzheimer's disease.

Pathological features in DLB [17, 18]
- Diffuse Lewy bodies - Essential for diagnosis of DLB
- Lewy neuritis
- Senile Plaques (all morphological types)
- Neurofibrillary tangles
- Neuronal loss in substantia nigra
- Neuronal loss in locus coeruleus
- Meynert nucleus neuronal loss
- Microvacuolation and synapse loss
- Neurochemical abnormalities and neurotransmitter deficits e.g. Ach, Dopamine

Data from [17, 18]

DIFFERENTIAL DIAGNOSIS

DLB can be easily confused with Alzheimer's disease (AD) and Parkinson's disease (PD).It is important to differentiate between DLB, AD and PD due to differences in treatment approaches. As compared to AD, patients suffering from DLB more frequently show signs of frontal lobe dysfunction, more prominent visual and auditory hallucinations, fluctuating cognitive performance, greater sensitivity to neuroleptics [19] and parkinsonian symptoms. Patients with DLB also have more severe dopamine and acetylcholine loss as compared to AD. DaT FP-CIT scan can be useful to differentiate between DLB and AD. Other diagnoses which can be confused with DLB include delirium and psychiatric illnesses.

Differential Diagnosis of DLB [20]
- Alzheimer's Disease
- Parkinson's Disease
- Dementia in Parkinson's Disease
- Psychiatric illnesses like mania and psychotic depression
- Vascular Dementia
- Delirium

INVESTIGATIONS

It is important to do dementia screen to rule out any reversible causes of cognitive impairment.

Blood tests
Laboratory studies should include those usually ordered in a dementia evaluation [21], including the following:
- FBC, ESR, CRP, biochemical screen
- Urea and creatinine
- T4 and TSH
- Glucose
- B12 and folate
- Clotting & albumin
- Syphilis serology
- HIV - if in young person
- Caeruloplasmin

Urine tests
Perform a midstream urine test if delirium is a possibility.

Imaging studies
- Structural imaging can be used to exclude other cerebral pathologies and help establish the subtype of dementia. Imaging studies may help to identify treatable causes such as subdural haematoma, normal pressure hydrocephalus, and cerebral tumours.
- Brain MRI is indicated to distinguish DLB from vascular dementia. Patients with vascular dementia often have white matter lesions on MRIs, whereas patients with DLB do not.
- Regionally distinct patterns of hypoperfusion on single-photon emission computed tomography (SPECT) or hypometabolism on positron emission tomography (PET) can help differentiate Frontotemporal Dementia, AD and Vascular Dementia, and dopaminergic loss in the basal ganglia can differentiate DLB from AD [22.]

- Reduced dopamine transporter activity in the basal ganglia is seen with positron emission tomography (PET) scanning or single-photon emission CT (SPECT) scanning.
- DaTSCAN (Ioflupane, 123-I FP-CIT) SPECT imaging.DaTSCAN contains Ioflupane labelled with radioactive iodide in an ethanolic solution. DaTSCAN is a drug used as part of a diagnostic procedure called SPECT imaging. DaTSCAN SPECT is indicated for detecting loss of functional dopaminergic neuron terminals in the striatum. The sensitivity of the FP-CIT scan for the diagnosis of DLB is 88% and specificity is 100 % [23].It helps to differentiate probable dementia with Lewy bodies from Alzheimer's disease.

MANAGEMENT

There is limited evidence about specific interventions but available data suggests a role for cholinesterase inhibitors, atypical antipsychotics, levodopa and clonazepam. For the treatment of agitation and hallucinations associated with DLB, acetyl cholinesterase inhibitors are the drugs of choice. In a small minority of patients, motor features are worsened with cholinesterase inhibitors. Most experts recommend atypical neuroleptics when cholinesterase inhibitors are ineffective. Levodopa/carbidopa may improve motor function in some patients with DLB; however, in many patients this combination has no effect and may exacerbate psychiatric symptoms or confusion. Depression is frequent in DLB patients and may result from damage in the dorsal raphe and locus ceruleus and/or as a psychological response to impaired function. Selective serotonin reuptake inhibitors are the drugs of choice.

PHARMACOLOGICAL TREATMENT

Acetyl cholinesterase inhibitors
Cholinergic deficits in DLB are even more severe than in AD [24]. Patients with DLB are more likely to improve with cholinesterase inhibitor therapy. Encouraging results have been obtained with Rivastigmine, Donezepil and galantamine. Double-blinded, placebo-controlled studies [25-27] have demonstrated that rivastigmine may decrease neuropsychiatric symptoms associated with DLB, particularly apathy, anxiety, hallucinations, and delusions. There is also some evidence from several case reports, open label trials and case series about the use of acetyl cholinesterase inhibiters including Rivastigmine and Donepezil in DLB [28-32].

Atypical neuroleptics
Due to increased sensitivity to antipsychotics, clinicians are generally cautious about the use of these drugs in patients with DLB. There have been multiple studies about the use of atypical antipsychotics like risperidone, olanzapine and quetiapine in DLB patients for the management of neuropsychiatric symptoms [33-37]. Patients with DLB frequently have distressing neuropsychiatric symptoms. When these symptoms are mild, no medical treatment may be necessary. Acetyl cholinesterase inhibitors should usually be tried first to treat neuropsychiatric symptoms [38]. Atypical antipsychotics appear to be better tolerated by DLB patients [39]. Most experts recommend atypical neuroleptics when cholinesterase inhibitors are ineffective. Neuroleptics should be reserved for situations where the psychosis is causing serious distress or putting the patient or others at risk. Very slow titration of the neuroleptic medication is indicated.

Anti-Parkinson's Medications

Patients with DLB can have troublesome parkinsonian symptoms which might need treatment. Treatment with levodopa-carbidopa combinations should be considered when symptoms cause functional impairment. Most of the evidence for benefit comes from case series [40, 41].

Benzodiazepines

Clonazepam can be helpful in treating REM sleep behaviour disturbances in DLB patients [42, 43].

Antidepressants

Patients with DLB have increased frequency of depression and anxiety. Selective serotonin reuptake inhibitors (SSRI's) are the drugs of choice.

NONPHARMACOLOGICAL TREATMENT

Nonpharmacological management mainly involves education of the patient and carers to deal with specific symptoms of the illness as well as general issues of caring for a patient with dementia [20].Various interventions including education of patient and family, structuring of environment, teaching behavioral skills and improving sensory impairment have been found useful in other types of dementias and might also be useful in patients suffering from dementia with lewy bodies [44-48].

AUTHOR DETAILS
JAVED LATOO, DPM, MRCPsych. Specialty Registrar (ST5) in Psychiatry with special interest in Neuropsychiatry, Royal Free and University College, London, United Kingdom
FARIDA JAN, MRCPsych. Specialty Registrar (ST4) in Old Age Psychiatry, Eastern Deanery, United Kingdom
CORRESSPONDENCE: North East London NHS Foundation Trust, Mascalls Park Hospital, Mascalls Lane Brentwood, Essex, CM145HQ, United Kingdom
Email: javedlatoo@googlemail.com

REFERENCES

1. Julia Zaccai, Cherie McCracken and Carol Brayne. A systematic review of prevalence and incidence studies of dementia with Lewy bodies Age and Ageing 2005 34(6):561-566.
2. Campbell S, Stephens S, Ballard C, Dementia with Lewy bodies: Clinical features and treatment, Drugs Aging, 2001; 18:397–407.
3. McKeith IG, Spectrum of Parkinson's disease, Parkinson's dementia, and Lewy body dementia, Neurol Clin, 2000; 18: 865–902.
4. McKeith IG, Galasko D, Kosaka K, et al. Consensus guidelines for the clinical and pathologic diagnosis of dementia with Lewy bodies (DLB): report of the consortium on DLB international workshop. Neurology. Nov 1996; 47(5):1113-24.
5. McKeith IG, Perry EK, Perry RH. Report of the second dementia with Lewy body international workshop: diagnosis and treatment. Consortium on Dementia with Lewy Bodies. Neurology. Sep 22 1999; 53(5):902-5.
6. McKeith IG, Dickson DW, Lowe J, et al. Diagnosis and management of dementia with Lewy bodies: third report of the DLB Consortium. Neurology. Dec 27 2005; 65(12):1863-72.
7. Lewy Body Dementia Association, Inc. Information page accessed online on 23 Aug. 08 at http://www.lbda.org/category/3438/symptoms.htm
8. Ballard C, Ayre G, O'Brien J, Sahgal A, McKeith IG, Ince PG, et al. Simple standardised neuropsychological assessments aid in the differential diagnosis of dementia with Lewy bodies from Alzheimer's disease and vascular dementia. Dement Geriatr Cogn Disord 1999; 10:104-8.
9. Mori E, Shimomura T, Fujimori M, Hirono N, Imamura T, Hashimoto M, et al. Visuoperceptual impairment in dementia with Lewy bodies. Arch Neurol 2000; 57(4):489-93.
10. Doubleday EK, Snowden JS, Varma AR, Neary D. Qualitative performance characteristics differentiate dementia with Lewy bodies and Alzheimer's disease. J Neurol Neurosurg Psychiatry 2002; 72:602-7.
11. McKeith I, Mintzer J, Aarsland D, Burn D, Chiu H, Cohen-Mansfield J, et al. Dementia with Lewy bodies. Lancet Neurol 2004; 3:19-28.
12. Knopman DS, Boeve BF, Petersen RC. Essentials of the proper diagnoses of mild cognitive impairment, dementia, and major subtypes of dementia. Mayo Clin Proc 2003; 78:1290-308.
13. Mosimann UP, McKeith IG. Dementia with Lewy bodies-diagnosis and treatment. Swiss Med Wkly 2003; 133:131-42.
14. Aarsland D, Ballard C, McKeith IG, Perry RH, Larsen JP. Comparison of extrapyramidal signs in dementia with Lewy bodies and Parkinson's disease. J Neuropsychiatry Clin Neurosci 2001; 13(3):374-9.
15. Stewart JT. Defining diffuse Lewy body disease. Tetrad of symptoms distinguishes illness from other dementias. Postgrad Med 2003; 113:71-5.
16. NussbaumRL, Ellis CE.Alzheimer's disease and Parkinson's disease {Published correction appears in N Eng J Med 2003; 348:2588}.N Engl J Med 2003; 348: 1356-64.
17. Alex Mitchell, Textbook of Neuropsychiatry and Behavioural Neurology Saunders 2004.
18. Paul G. Ince, Elaine K. Perry, Chris. M. Morris. Dementia with Lewy Bodies. A Distinct Non-Alzheimer Dementia Syndrome? Brain Pathology. April 1998.
19. Bolla LR, Filley CM, Palmer RM, Dementia DDx: Office diagnosis of the four major types of dementia, Geriatrics, 2000; 55:34–46.
20. Frank Christopher, Dementia with Lewy bodies, Can Fam Physician 2003; 49:1304-1311.
21. NICE (November 2006). Dementia guidelines
22. O'Brien JT. Role of imaging techniques in the diagnosis of dementia.Br J Radiol. 2007 Dec;80 Spec No 2:S71-7
23. Walker Z et al. Dementia with Lewy bodies: a comparison of clinical diagnosis, FP-CIT SPECT imaging and autopsy.J Neurology Neurosurgery Psychiatry.2007 Nov;78(11):1176-81
24. Gomez-Tortosa E, Ingraham AO, Irizarry MC, Hyman BT. Dementia with Lewy bodies. J Am Geriatr Soc 1998; 46:1449-58.
25. Wesnes K, McKeith IG, Ferrara R, Emre M, Del Ser T, Spano PF, et al. Effects of rivastigmine on cognitive function in dementia with Lewy bodies: a randomized placebo-controlled international study using the cognitive drug research computerised assessment system. Dement Geriatr Cogn Disord 2002; 13:183-92.
26. McKeith IG, Grace J, Walker Z, Byrne EJ, Wilkinson D, Stevens T, et al. Rivastigmine in the treatment of dementia with Lewy bodies: preliminary findings from an open trial. Int J Geriatr Psychiatry 2000; 15:387-92.
27. McKeith IG, Del Ser T, Spano PF, Emre M, Wesnes K, Anand R, et al. Efficacy of rivastigmine in dementia with Lewy bodies: a randomised, double-blind, placebo-controlled international study. Lancet 2000; 356(9247):2031-6.
28. Maclean LE, Collins CC, Byrne EJ. Dementia with Lewy bodies treated with rivastigmine: effects on cognition, neuropsychiatric symptoms, and sleep. Int Psychogeriatr 2001; 13(3):277-88.
29. Grace J, Daniel S, Stevens T, Shankar KK, Walker Z, Byrne EJ, et al. Long-term use of rivastigmine in patients with dementia with Lewy bodies: an open-label trial. Int Psychogeriatr 2001; 13(2):199-205.
30. Samuel W, Caligiuri M, Galasko DR, Lacro J, Marini M, McClure FS, et al. Better cognitive and psychopathologic response to donepezil in patients prospectively diagnosed as dementia with

Lewy bodies: a preliminary study. Int J Geriatr Psychiatry 2000; 15:794-802.

31. Querfurth HW, Allam GJ, Geffroy MA, Schiff HB, Kaplan RF. Acetylcholinesterase inhibition in dementia with Lewy bodies: results of a prospective pilot trial. Dement Geriatr Cogn Disord 2000;11:314-21.

32. Shea C, MacKnight C, Rockwood K. Donepezil for treatment of dementia with Lewy bodies: a case series of nine patients. Int Psychogeriatr 1998; 10(3):229-38.

33. Sechi G, Agnetti V, Masuri R, Deiana GA, Pugliatti M, Paulus KS, et al. Risperidone, neuroleptic malignant syndrome and probable dementia with Lewy bodies. Prog Neuropsychopharmacol Biol Psychiatry 2000; 24(6):1043-51.

34. Kato K, Wada T, Kawakatsu S, Otani K. Improvement of both psychotic symptoms and Parkinsonism in a case of dementia with Lewy bodies by the combination therapy of risperidone and L-DOPA. Prog Neuropsychopharmacol Biol Psychiatry 2002; 26(1):201-3.

35. Fernandez HH, Trieschmann ME, Burke MA, Friedman JH. Quetiapine for psychosis in Parkinson's disease versus dementia with Lewy bodies. J Clin Psychiatry 2002; 63(6):513-5.

36. Walker Z, Grace J, Oveshot R, Satarasinghe S, Swann A, Katona CL, et al. Olanzapine in dementia with Lewy bodies: a clinical study. Int J Geriatr Psychiatry 1999;14:459-66.

37. Cummings JL, Street J, Masterman D, Clark WS. Efficacy of olanzapine in the treatment of psychosis in dementia with Lewy bodies. Dement Geriatr Cogn Disord 2002; 13:67-73.

38. Cummings JL. Cholinesterase inhibitors: expanding applications. Lancet 2000;356(9247):2024-5

39. Andreasen NC, Black DW, Delirium, dementia, and amnestic disorder. In: Introductory Textbook of Psychiatry, 4th ed., Arlington, Virginia: American Psychiatric Publishing, Inc., 2006.

40. Byrne EJ, Lennox G, Lowe J. Diffuse Lewy body disease, clinical features in 15 cases. J Neurol Neurosurg Psychiatry 1989; 47:709-17.

41. Geroldi C, Frisoni GB, Bianchetti A, Trabucci M. Drug treatment in Lewy body dementia. Dement Geriatr Cogn Disord 1997; 8:188-97.

42. Stewart JT. Defining diffuse Lewy body disease. Tetrad of symptoms distinguishes illness from other dementias. Postgrad Med 2003; 113:71-5.

43. Boeve BF, Silber MH, Ferman TJ. REM sleep behavior disorder in Parkinson's disease and dementia with Lewy bodies. J Geriatr Psychiatry Neurol 2004; 17:146-57.

44. Mosimann UP, McKeith IG. Dementia with Lewy bodies-diagnosis and treatment. Swiss Med Wkly 2003; 133:131-42.

45. Burn DJ, McKeith IG. Current treatment of dementia with Lewy bodies and dementia associated with Parkinson's disease. Mov Disord 2003; 18(suppl 6):S72-9.

46. Kalra S, Bergeron C, Lang AE. Lewy body disease and dementia. A review. Arch Intern Med 1996; 156:487-93.

47. Doody RS, Stevens JC, Beck C, Dubinsky RM, Kaye JA, Gwyther L, et al. Practice parameter: management of dementia (an evidence-based review). Report of Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2001; 56:1154-66.

48. Neef and Walling. Dementia with Lewy Bodies; An Emerging Disease.Am Fam Physician 2006; 73:1223-9.

Review

Anaesthetic management of obese parturient

Nimit Shah and Yaqub Latoo

SUMMARY

Obesity has become a ticking time bomb. The population of obese people and so also obese pregnant patients is increasing worldwide and it won't be long before when anaesthetists will be more commonly faced with managing obese parturients with a large spectrum of comorbidities. The last confidential enquiry into maternal and child health (CEMACH) 2002 - 2005 report stressed obesity as a major risk factor associated with maternal mortality and following suit of its recommendation, we write this review article on management of obese parturients, highlighting the problems in obese parturients and recommending guidelines for management of such patients. As the use of regional anaesthesia in obstetrics anaesthesia has increased, the trainee anaesthetists are relatively less skilled to provide general anaesthesia. General anaesthesia with all the airway management problems has been the major reason of maternal mortality in the previous CEMACH reports. An epidural block though technically difficult, provides optimal analgesia and can be extended for caesarean section if required. Hence obese parturient should be assessed and consulted by a senior anaesthetist as early as 28 weeks of gestation in the pregnancy for formulating a plan for labour analgesia and anaesthesia for caesarean section if required. Epidural analgesia should be provided in early labour prophylactically to avoid general anaesthesia. Early anaesthetic assessment, prophylactic epidural block, ensuring its effectiveness, alternative plan for failed regional block along with preparation for general anaesthetic and difficult intubation, involving senior help in the management and multidisciplinary approach are advocated to mitigate potential anaesthetic risks.

Abbreviations: BMI - Body mass index

Obesity has become a major health problem of modern society and increasing globally at nearly epidemic proportions especially in western and European countries [1,2,3,4].

DEFINING OBESITY

Obesity can be simply defined as a condition in which body fat is in excess beyond a point incompatible with physical and mental health and normal life expectancy [5] or as a metabolic disorder that is primarily induced and sustained by an over consumption or underutilization of caloric substrate. There are 2 types of obesity; Android obesity which is truncal distribution of fat associated with high incidence of cardiovascular disorders and Gynecoid obesity where fat is distributed to thighs and buttocks associated with pregnancy and not tightly linked to cardiovascular problems [6,7].

Indices used to for obesity are Ideal body weight in kilograms (Broca's Index), and more commonly the BMI or body mass index (also called Quetelet's index).

1) Ideal body weight = height in centimeters - 100 for men (105 for women). Overweightness is 20% more than ideal body weight and morbid obesity is twice the Ideal Body weight.
2) BMI = weight in kgs/ square of height in meters

PREVALENCE

In the US more than 60 million adults can be classified as either overweight or obese with morbid obesity affecting more than 9 million adults. Approximately 30 – 40 % of females are obese and it is estimated that 50 per cent of women will be obese by 2050. A study looking at trends in pre-partum obesity in nine states of the United States found an increase in pre-partum obesity from 13% in 1993–1994 to 22% in 2002–2003 [8].

WHO CLASSIFICATION OF OBESITY [9]

Classification	Body mass index (kg/m2)	Associated health risks
Underweight	<18.5	Low
Normal range	18.5–24.9	Average
Overweight	>25.0	
Preobese	25.0–29.9	Increased
Obese class I	30.0–34.9	Moderately increased
Obese class II	35.0–39.9	Severely increased
Obese class III	>40	Very severely increased

In the UK, 56% of all women are over the recommended BMI, with 33% of them classified as overweight (BMI > 25) and 23% obese (BMI > 30). The Health Survey of England published in 2002 gives data about the prevalence of obesity in England. Females in the reproductive age group (16 – 44 years) have shown a dramatic increase in BMI. The percentage of women with BMI above 30 increased from 12% in 1993 to 18.3% in 2002. Also alarming is that the percentage of morbidly obese women has doubled in the last decade [10]. The dramatically increasing rate of obesity in the general population also extends to women of reproductive age.

PATHOPHYSIOLOGICAL CHANGES IN OBESE PREGNANT PATIENT

Obesity compounds most of the physiological changes in pregnancy

Airway - Obesity and pregnancy each increase the chance of difficult airway. Limited mouth opening and limited neck movements are common in obesity. There is narrowing of the pharyngeal opening due to excess adipose tissue and on airway examination, the airway will have more commonly high of mallampati grades. In pregnancy, particularly in pregnancy induced hypertension, the mucous membranes in the airway are oedematous and hence more prone to bleeding. Breast enlargement in pregnancy also predisposes to difficult airway.

Respiratory system – There are significant changes in an obese parturient and most of them are additive. In early pregnancy , in a non obese parturient, even before the uterus is large enough to affect respiratory function, women begin to have a sensation of dyspnea. This sensation likely occurs from the increased alveolar ventilation, probably secondary to progesterone effects on the respiratory center in the brainstem [11,12]. By the fifth month of pregnancy, the growing uterus begin to cause a progressive decrease in expiratory reserve volume (ERV), residual volume (RV) and functional residual capacity (FRC), which at term are about 15–20% less than those of the non-pregnant state[13]. Obesity in non-pregnant subjects is associated with a decrease in expiratory reserve volume (ERV), residual volume (RV) and functional residual capacity (FRC), most likely caused by the added weight of excess fat on the chest and abdomen and decreased chest compliance[13 – 16]. Eng et al.[13] showed, however, that obese parturients did not have a significant additional reduction in functional residual capacity (FRC) compared to normal-weight parturients, which might be partially explained by the fact that the study was performed with the parturients in the sitting position. Another possible explanation is progesterone which has a relaxing effect on smooth muscle and decreases airway resistance, thus reducing some of the negative effects of obesity on the respiratory system [12, 17].

Dempsey et al.[18] have showed that excess body weight in obesity increases oxygen consumption and CO_2 production in a linear fashion. The work of breathing is increased in obese parturients due to chest wall weight and they typically show a rapid and shallow breathing pattern [18]. This leads in turn to a higher ventilatory requirements and work of breathing [12,19]. The supine, lithotomy, induction of general anaesthesia and especially the Trendelenburg position worsen lung volumes significantly. The functional residual capacity (FRC) is further reduced and the closing capacity (CC) encroaches on the funtional residual capacity (FRC) resulting in small airway collapse, ventilation perfusion mismatch, shunting and hypoxemia[20]. These physiologic changes make the obese parturient particularly prone to rapid desaturation, stressing the importance of adequate denitrogenation ('pre-oxygenation') before induction of general anaesthesia.

In non-obese parturients, physiologic changes during pregnancy are thought to protect from obstructive sleep apnea, due to high circulating levels of progesterone, which is a ventilatory stimulant [18]. However, obesity increases the risk for obstructive sleep apnea significantly and this syndrome is not uncommon in the obese parturients. Obesity hypoventilation syndrome (OHS , Pickwickian syndrome) is seen in 8% of population of obese parturients characterized by morbid obesity, alveolar hypoventilation and daytime somnolence. In response to chronic hypoventilation and hypoxemia, they develop polycythaemia, increased cardiac output, cardiomegaly, pulmonary hypertension and eventually right heart failure. There is a significant increase in morbidity and mortality. They are more to obstructive sleep apnoea. Pulmonary embolism and pneumonia are also common in these patients [25].

Cardiovascular system – Cardiac output increases in pregnancy, with a significant increase in cardiac output, becoming detectable by the third week of pregnancy and a 35 - 40% increase by the end of the first trimester. Cardiac output continues to rise throughout the second trimester until it reaches a level that is approximately 50% more than that in the non-pregnant state. For the remaining pregnancy, cardiac output remains relatively stable around that level. During labour, cardiac output increases further by approximately 10% in the early first stage, 25% in the late first stage and 40% in the second stage. Uterine contractions increase cardiac output by further 10–15% and in the immediate post-partum period the cardiac output peaks at as much as 75% above prepartum values [19]. Obesity increases cardiac output even further because of extra amount of fat. Every 100 g of fat increases the cardiac output by 30–50 ml/min[22]. Blood volume is increased in pregnancy and even more when pregnancy is complicated by obesity. In non-obese parturients, there is a significant reduction in afterload [22]. In obese pregnant parturients, however, afterload reduction may be impaired due to increased peripheral resistance and greater conduit artery stiffness [23]. Additionally, obesity is associated with a higher prevalence of hypertension, diabetes mellitus, hyperlipidemia and poor cardiac function and it is one of the leading risk factors for coronary artery disease and cerebrovascular accidents [24]. Due to hyperdynamic circulation, there ensues left ventricular hypertrophy and diastolic dysfunction. Systolic function might remain normal but progressively systolic dysfunction may ensue. Pulmonary blood volume increase due to increased cardiac output. Pulmonary hypertension can develop and is exacerbated by supine position, airway obstruction and hypoxemia can develop. In obesity hypoventilation syndrome, right ventricular failure can develop. Increased number of peripartum cardiomyopathy cases are seen in obese pregnant parturients but it is unclear if obesity is a risk factor [25].

The obese pregnant parturients are at an increased risk of supine hypotension syndrome (SHS) due to compression of major abdominal vessels. This is exacerbated by large panniculus which adds to the uterine compression. Tseuda et al. have reported two cases of sudden death on assuming the supine position in morbidly obese patients [26].

Gastrointestinal system – Obesity further decreases lower oesophageal tone which is already decreased in pregnancy and increase the risk of aspiration of gastric contents and Mendelson's syndrome [27, 28]. Hiatus hernia is increased in obese patients. Roberts and Shirley studied obese and non obese pregnant parturients in labour; the gastric volume in obese parturients is five times greater than in the controls [29,30,31]. Obese population have a higher incidence of diabetes, which can cause delayed gastric emptying, increasing the risk for aspiration. Also,

it is well known that obesity predisposes to difficult or failed intubation, both of which are associated with a higher incidence of aspiration.

Others - Gestational diabetes is common. Obesity metabolic syndrome includes dyslipidemia, impaired endothelial function, high blood pressure, increased inflammatory mediators, insulin resistance and hyperinsulinemia even in absence of diabetes [25].

PHARMACOKINETICS AND PHARMACODYNAMICS CHANGES

Obesity increases both fat and lean masses; however, the percentage of fat tissue increases more than does the lean mass, affecting the apparent volume of distribution of anaesthetic drugs according to their lipid solubility. Thiopental sodium and propofol dosages are calculated on total body weight (TBW). Benzodiazepine loading doses should be adjusted on actual weight, and maintenance doses should be adjusted on ideal body weight. The loading dose of lipophilic opioids is based on total body weight (TBW), whereas maintenance dosages should be cautiously reduced because of the higher sensitivity of the obese patient to their depressant effects. Pharmacokinetic parameters of muscle relaxants are minimally affected by obesity, and their dosage is based on ideal rather than total body weight (TBW). Minimum alveolar concentration is decreased. Inhalation anaesthetics with very low lipid solubility, such as sevoflurane and desflurane, allow for quick modification of the anaesthetic plan during surgery and rapid emergence at the end of surgery, hence representing very flexible anaesthetic drugs for use in this patient population. Drug dosing is generally based on the volume of distribution for the loading dose and on the clearance for maintenance. In the obese patient, the volume of distribution is increased if the drug is distributed both in lean and fat tissues whereas the anaesthetic drug clearance is usually normal or increased [32]. Albumin binding of drugs is unchanged in the obese, but levels of fatty acids, triglycerides, and a1-acid glycoprotein are increased and may influence plasma protein binding. In pregnancy, the volume of distribution is increased, albumin concentration decreased and the renal clearance is increased. Net effect is unpredictable. Pseudocholinesterase levels are decreased in pregnancy.

LOCAL ANAESTHETIC REQUIREMENTS

Lower dose of local anaesthetic is required (less by 25%) when injected neuraxially. Proposed mechanisms are pregnancy induced hormone related changes in the action of spinal cord neurotransmitters, potentiation of the analgesic effect of the endogeneous analgesic systems, increased permeability of the neural sheath [25] and decreased dilution by decreased volume of cerebrospinal fluid (CSF) Hodgkinson et al [33] have shown an increased cephalad spread of local anesthetics in obese patients. Hogan et al.[34] found a lower average cerebrospinal fluid (CSF) volume in obese subjectsI, which could explain the decreased local anesthetic dose requirements due to decreased anaesthetic dilution. Since similar changes were noticed with external abdominal compression and abdominal pressure increases linearly with increased body weight [35], increased abdominal pressure is probably the cause. Some [36] have also attributed the decrease in cerebrospinal fluid (CSF) volume to compression of the dural sac due to engorgement of the epidural venous plexus and increased epidural space pressure, resulting from

compression of the inferior vena cava by gravid uterus with redistribution of the venous return from the lower limbs and pelvis. Hogan et al.[34], however, suggested that the mechanism by which increased abdominal pressure decreases the CSF volume is probably inward movement of soft tissue (mostly fat) in the intervertebral foramen, which displaces CSF. This hypothesis is based on their findings that the greatest change in CSF volume during abdominal compression was found at sites in which intervertebral foramina were present. Greene suggests that larger buttocks of obese patients place the vertebral coloumn in the Trendelenburg position, exaggerating the cephalad spread of the local anaesthetic [25, 37].

CONSEQUENCES OF OBESITY IN PREGNANCY

Obesity severely complicates pregnancy. It affects both the mother and foetus

Maternal consequences

There is increased risk of gestational diabetes and type 2 Diabetes. There is 3 - 10 fold higher risk of gestational diabetes (type 1 insulin dependent diabetes mellitus)[38]. Studies have shown when pregnancy is complicated by gestational diabetes, there is a higher risk of developing type 2 diabetes mellitus in later life [39]. There is a 2 – 4 fold increased risk of preeclampsia. The risk is almost 5 times greater in the morbidly obese group; typically a BMI > 35 [38, 40]. But there is no increased risk of HELLP (haemolysis, elevated liver enzymes, low platelets) syndrome [31]. Obesity is an independent risk factor for hypertension [41].

It is reported [42] that there is a higher chance of failure to progress, prolonged second stage of labour and a failed induction of labour in obese compared to non obese parturients and this is secondary to soft tissue dystocia. There is a higher risk of instrumental delivery of up to 18% in women with a BMI between 35 and 40 and up to 34% in patients with BMI greater than 40. Also there is an increased risk of failed instrumental delivery leading to caesarean section. There is 3 times higher risk of caesarean section in a obese parturient. This is due to fetal macrosomia, higher risk of shoulder dystocia and/or failed cervical dilatation [38, 43, 44]. About two thirds present as emergency caesarean section [45]. The obese parturient is at a higher risk of having a prolonged incision to delivery time, blood loss greater than 1000 ml and prolonged operative times. There is an increased risk of wound infections and endometritis and dehiscence [46, 47]. There is an increased risk of major postpartum haemorrhage. The risk of postpartum haemorrhage rises with increasing BMI and is about 30% more frequent for a moderately raised BMI and about 70% more frequent for a highly raised BMI compared with the normal BMI group [45,48]. There is an increased risk of thromboembolism – obesity and pregnancy are each independent risk factors for deep vein thrombosis. Both pharmacological and mechanical methods should be used for thromboprophylaxis.

Obese women spend an average of 5 more days in hospital resulting in 5 fold increase in cost of care due to potential complications such as wound infections and postpartum haemorrhage [49].

Fetal consequences

Maternal obesity is associated with large for gestational age infants. There is increased risk of a macrosomic foetus, independent of maternal diabetes [43, 50]. There is an increased risk of shoulder dystocia upto three times more common in the morbidly obese parturients. The risk of foetal macrosomia and shoulder dystocia increases with increase in BMI [44].

There is an increased risk of Infant birth defects. Since 1994 a number of studies have shown an association between maternal obesity and infant birth defects. Anomalies include neural tube defects such as anencephaly, anomalies of the heart and intestinal tract, omphaloceles, orofacial clefts, and multiple congential anomalies of the central nervous system [43, 51, 52].

There is an increased risk of stillbirth, a three times increase in antepartum stillbirth was found in morbidly obese parturients compared with women of normal BMI [52, 53].

Due to the depth of maternal adipose, foetal monitoring by intermittent or continuous Electronic Foetal Monitoring using external transducers may be technically difficult. The use of fetal scalp electrodes and intrauterine pressure catheters to ensure an acceptable standard of fetal monitoring may be needed.

In a study 'Maternal obesity and pregnancy outcome: a study of 287213 pregnancies in London' (N J Sebire, et all, International journal of obesity (2001) 25, 1175 - 82) complications such as gestational diabetes mellitus proteinuric pre-eclampsia; induction of labour; delivery by emergency caesarean section; postpartum haemorrhage; genital tract infection; urinary tract infection, wound infection; birth weight above the 90th centile, and intrauterine death were significantly higher in obese pregnant parturients than non obese pregnant parturients. However, delivery before 32 weeks gestation and breastfeeding at discharge were significantly less likely in the overweight groups. In all cases, increasing maternal BMI was associated with increased magnitude of risk [38]. Weiss et al.[54] found for nulliparous patients a caesarean delivery rate of 20.7% in the control group compared with 33.8% in the obese and 47.4% in the morbidly obese group.

The Confidential Enquiry into Maternal and Child Health 2004 reported that 35% of all maternal deaths occurring in the triennium 2000–2002 were in obese women with BMI > 30. The most recent CEMACH reports in the United Kingdom reported that obesity was a cofactor in a significant number of the maternal deaths between 2003 and 2005. Twenty seven percent of the women who died had BMI > 30. Of these women, 12% had a BMI between 30 and 34, 7% had values between 35 and 39 and 8% had a BMI of 40 or more. 295 women who died 119 were overweight and 64 of those were morbidly or super-morbidly obese. In 30 per cent of women who experienced a stillbirth or perinatal death, the maternal BMI was recorded at more than 30.

Furthermore, Cedergren et al found a 3 fold increased rate of stillbirths, 5 fold increased risk of preeclampsia and a 3 fold increased risk of caesarean section. The success rate for a vaginal delivery in obese parturient with a previous caesarean section is less than 15% [38].

ANAESTHETIC MANAGEMENT

Obese parturients have severely limited physiological reserve and a higher risk of emergency surgical intervention. Hence the anaesthetic risks increase greatly. Obesity and pregnancy each has multisystem effects, many of which are additive. A thorough understanding of the physiology, associated conditions and morbidity, available options for anaesthesia and possible complications is important.

Senior anaesthetist must be involved early in multidisciplinary approach for patient care as early as 28 weeks of gestation. The preoperative assessment include evaluation of airway, respiratory and cardiovascular system and pregnancy associated problems such as pregnancy induced hypertension, gestational diabetes etc and also should include patient education. An examination of the back should be done.

Airway

The obese parturients need thorough pre-operative assessment for difficult airway as incidence of failed intubation is 8 times higher than non obese patients. In the obstetric population, between one in 280 and one in 750 attempted tracheal intubations fail [45], compared to one in 2230 in the general population [17, 55,56]. In contrast, the incidence of difficult intubation in obese population, is as high as 15.5% [57]. Dewan [58] found the incidence as high as 33% in morbidly obese parturients. A 6-year review of failed intubations in parturients in a United Kiingdom region reported 36 cases of failed intubation and it was found that the average BMI of these women was 33 [57]. So it is evident that incidence of difficult or failed tracheal intubation in obese parturients is very high and emphasizes optimal assessment and management of the airway. An airway assessment should include mallampati classification, thyromental distance, neck extension (atlanto-occipital joint extension), mouth opening (vertical dimension). The combination of two tests (mallampatti and thyromental distance), though in a small study of 80 parturients receiving general anaesthesia, has been shown to be 100% sensitive with 70% positive predictor value [59]. These tests can be done in less than 1 minute; hence they are also useful in an emergency scenario. Other features shown to be of significance are short neck, receding mandible and protruding incisors [60]. It is of interest to note that neck circumference, not BMI, is more predictive of a difficult intubation in morbidly obese patients [61]. A study has shown a gestational weight gain of more than 15 kgs is associated with three times increase in suboptimal layngoscopic view as compared to that in non obese parturients of the same age [57, 62]. This means weight gain in pregnancy should be limited in obese parturients and if an obese parturient presents who has gained more than 15 kgs of weight in pregnancy, she will be more likely to have a difficult airway [62]. A plan of airway management should be formulated in case of an emergency for all women regardless of the primary obstetric and anaesthetic plan. Although rapid sequence intubation with proper positioning and back up equipment may be adequate for most women, an alternative airway plan should be considered. A history of snoring, diagnosis of sleep apnoea, lack of teeth, and large breasts all increase risk of difficult intubation and awake fibreoptic intubation should be considered in all patients with limited range of neck, head or jaw movements, short neck, neck

circumference of 15 inches and above, and mallampati score of 3 and above [25]

Respiratory System

Usually a complete history and chest examination and routine investigations including an ECG is adequate for a preoperative anaesthetic fitness. However chest X ray, arterial blood gas, pulmonary function tests can be done to aid further evaluation of respiratory reserve. Measurement of oxygen saturation by pulse oximetry in sitting and then supine can provide evidence of airway closure during normal tidal volume ventilation, thereby identifying candidates for post operative oxygen administration [17].

Women with obesity are more likely to have obstructive sleep apnoea but the prevalence is unknown in pregnancy. Sleep disturbances and day time fatigue are normal at the end of pregnancy and so obstructive sleep apnoea may go undiagnosed. J Mhyre [25] has suggested women with a BMI > 35, neck circumference of greater than 16 inches, symptoms of suspected airway obstruction during sleep (include frequent or loud snoring, observed pauses in breathing during sleep, frequent arousals from sleep or arousal with a choking sensation) should be screened by polysomnography for obstructive sleep apnoea and advised continuous positive airway pressure (CPAP) if required.

If obesity hypoventilation syndrome is suspected arterial blood gas is useful to screen hypoxia, hypercarbia and acidosis and echocardiogram should be done to evaluate cardiac function and patient should be referred to cardiologist [25,63]

Cardiovascular system

Cardiovascular co-morbidities such as hypertension, ischaemic heart disease and heart failure can co-exist in obese parturients. Nearly 40% of the obese population experience angina without demonstrable coronary artery disease [64]. Pulmonary hypertension can be present. Hence cardiologists should be involved early in the care of symptomatic morbidly obese parturients to investigate and optimise the disease status wherever appropriate [17]. Echocardiogram may be useful.

The obese parturients cannot be accurately stratified for perioperative risk using the usual screening indices such as Goldman's index etc as obesity and pregnancy are not included as risk factors in these indices and they might be classed in the lower risk group despite having significantly increased risk.

Others

Patients should be assessed for pregnancy associated problems such as pregnancy induced hypertension and gestational diabetes mellitus etc.

Peri-operative issues such as transfers, beds, intravenous access, central venous access, difficulty in measuring non invasive blood pressure, arterial cannulation, different size regional anaesthesia kit should be anticipated, discussed and planned for.

Post operative intensive care management /high dependency care should be sought for. Deep vein thrombosis prophylaxis must be put in place. The management plan should be liaised with whole team including consultant anaesthetists, consultant obstetricians, consultant intensivists, midwives, operating department practioners (ODP's) and physiotherapists

ANALGESIA FOR LABOUR

Each of the risk factors of fetal macrosomia and shoulder dystocia which are increased in obese parturient result in more painful contractions and complicated labour [65]. Although there are various modalities of pain relief, analgesia using neuroaxial blockade has been shown to be the most effective [66]. The anticipated technical difficulties should not preclude the use of epidural analgesia in obese parturients. It is been shown effective pain relief during labour can improve maternal respiratory function and attenuate sympathetically mediated cardiovascular responses [67, 68]. Available evidence shows that the rate of caesarean delivery does not increase with epidural analgesia during labour [66], though obesity increases the need for caesarean section. Hence, placing a functional epidural catheter is advantageous should any operative intervention be required. In addition, epidural analgesia can be extended into the postoperative period where adequate pain relief can optimise care.

The challenges for the anaesthetist should not be underestimated. Technical problems include appropriate positioning of the patient, identification of the midline and epidural space, and dislodgement of catheters [45, 69, 70]. The initial failure rate for epidural catheter placement can be very high (42%) [45] and multiple attempts of catheter placement are common. Jordan et al. noted 74.4% of massively obese parturients needed more than a single attempt and 14% needed more than three attempts for successful epidural placement [71]. The knee–chest position required for doing epidural in the lateral position is difficult to obtain in the obese. One study has shown that cardiac output decreased more in the lateral decubitus position with maximal lumbar flexion compared with the sitting position [72]. Moreover, in the lateral position, gravity can drag down the pad of fat obscuring the midline. Another study found the depth of the epidural space from skin to be greater in patients where the epidural was inserted in the lateral decubitus position [73]. Overall, the sitting position is preferable and should be used.

STEPS AND CAVEATS

Early placement and confirmation of optimal epidural analgesia even before onset of labour (when a term patient presents before labour) is prudent. This allows sufficient time to manage a failed epidural block (because not only the incidence of failed initial epidural catheter placement is high in obese parturients, but the incidence of failed epidural during labour due to migration of epidural catheter in the fatty subcutaneous tissues is also high)[17, 74, 75]. Re-evaluate the airway and cardiorespiratory status.

Senior anaesthetist preferably a consultant anaesthetist should be involved. Ensure wide intravenous cannula (preferably 14 or 16 gauge) in place. In case of problems with blood pressure cuff not measuring, cuff can be placed on calf/forearm; will help in getting the trends if no accurate reading. Invasive blood pressure monitoring might be needed. Ensure pulse oximetry monitoring and supplement oxygen by mask if required.

Perform in sitting position. Ensure midline position as even if slight deviation of the midline will lead to exaggerated directional errors due to increased length of epidural space from the skin and hence failure of epidural. Midline might be not possible to palpate, in this case drop a line from C1 spinous process to lower skin crease and this may be guide as a midline. Strapping excess fat away from the midline might be necessary.

If highest points of iliac crests are palpated for the Tuffier's line then because of fat pads on the sides, higher spaces might be inadvertently selected and increased chance of spinal cord damage. In case of difficulty, lower thoracic space may be selected.

A recent study in pregnant patients has shown a positive correlation between BMI and the distance to skin to the lumbar puncture [76]. Although the epidural space may be deeper in overweight people, the majority of studies report that only a few have an epidural space deeper than 8 cms [73, 77]. Hence it seems appropriate to use a standard needle to identify the epidural space on the first attempt. In morbidly obese patient ultrasound technique has been found valuable in establishing epidural [78, 79]

In case of difficulty in insertion, a deliberate spinal with 25 guage needle might be performed (no injection of drugs) to assess the midline and depth of epidural space. There is an increased risk of dural tap [25], but decreased risk of postdural puncture headache [75]. In case of dural tap, epidural can be converted to continuous spinal catheter analgesia with extreme caution. Also there is an increased risk of Intravenous placement of epidural catheter due to engorged epidural veins and decrease in epidural space. The meniscus drop (negative pressure) test is not reliable as epidural pressure may be high [25]. Minimum 5 cms of catheter in space should be left. To minimize catheter displacement, it should be secured on assumption of upright or preferably lateral position from the initial flexed position. The epidural should be checked with a test dose and a functioning epidural should be ensured. Sometimes a longer epidural needle might be required. There is an advantage to titrate block height. Minimum local anaesthetic concentration (MLAC) is lower in obese pregnant patients compared to non pregnant patients [80]

If epidural is contraindicated or impossible to site then entonox is an useful adjunct. Intramuscular opioids are not reliable. Patient controlled analgesia can be used but cautiously as increased chance of sedation and respiratory depression. Remifentanyl, an ultra short acting opioid, has favourable pharmacokinetics to be used as an opioid for patient controlled analgesia but not enough data is available for its use in obese parturients. It is metabolized by red blood cells and tissue esterases both in mother and foetus and hence does not accumulate and is easily antagonized if required. However it is a potent respiratory depressant and hence should be used very cautiously in obese parturients who would be susceptible to its sedative side effects and hence they should be managed in high dependency unit with appropriate monitoring and one to one nursing by skilled midwife and under observation of a highly skilled anaesthetist. The dosage should be carefully titrated individually and naloxone and difficult airway trolley ready. Patients with obstructive sleep apnoea would be very susceptible to its sedative side effects and hence should be avoided. Proper training of patients is required as its peak effect is 2 -3 minutes

and if the button of patient controlled analgesia is pressed at the onset of contraction it would be less effective. The duration of its use should be minimized as much as possible.

ANALGESIA/ANAESTHESIA FOR CAESAREAN SECTION

Obesity and Caesarean section have been identified as independent risk factors for maternal morbidity and mortality [44]. Analysis of direct maternal deaths due to anaesthesia, in the confidential enquiries report on maternal mortality in the United Kingdom from 1979 to 2005, reveals that the majority of deaths occurred under general anaesthesia, compared with regional anaesthesia [17]. Most parturients who die of complications of general anaesthesia die of airway management problems, including aspiration, failed intubation, inadequate ventilation, and respiratory failure.[54, 55]

Factors that play a role in general anaesthesia being more likely to be associated with maternal mortality than regional anaesthesia are unexpected airway difficulties, pulmonary aspiration of gastric contents, emergency general anaesthesia (including conversion of a failed regional), peripartum haemorrhage, and embolism necessitating general anaesthesia, and resident lack of experience in general anaesthesia for caesarean section [28, 81].

In Why Mothers Die 2000–02, 35% of all the women who died were obese, 50% more than in the general population [82]. Hence regional anaesthesia preferably epidural should be opted unless contraindicated or difficult.

Direct maternal deaths due to anaesthesia by types of anaesthesia in United Kingdom 1979–2005. Derived from CEMD reports. Since 1979, maternal deaths are reported as direct and indirect.

Year	Total(n)	GA(n)	RA(n)	Other (n)
2002-05	6	4	1	1
2000-02	6	6	0	0
1997-99	3	2	1	0
1994-96	1	0	1	0
1991-93	8	7	1	0
1988-90	4	3	1	0
1985-87	8	7	1	0
1982-84	18	17	1	0
1979-81	22	22	0	0

GA, general anaesthesia; RA, regional anaesthesia; Other – eg Central VP line insertion

REGIONAL ANAESTHESIA FOR CAESAREAN SECTION

Different techniques can be used. Epidurals are reliable but have high failure rate, spinal is a familiar technique while combined spinal epidural has minimal side effects such as headache, high block , hypotension and can be used post operatively as well as for redo surgery.

Use 25% less local anaesthetic dose compared to non obese patient due to altered neuro-axial physiology and anatomy.

Epidural

A working epidural as above can be continued for caesarean section and it also provides post operative pain relief. However it may be inadequate in more than 25% of these patients, mainly because of difficulty in blocking the sacral roots, resulting in visceral pain upon stimulation of the bladder [83].

Spinal

An obese woman is a candidate for spinal anaesthesia if the airway examination is normal, cardiopulmonary derangements are minimal and the obstetricians aim to complete the surgery within 90 minutes [25]. In obese parturients spinals can be technically difficult requiring varied needle lengths and being unable to titrate to block for surgery and surgical duration. If the spinal wears off, general anaesthesia with all its inherent risks, will be required. Tuohy needle can be used as an introducer for the spinal needle [25]. Spinal opioids can provide post operative analgesia but respiratory monitoring becomes essential.

Combined Spinal Epidural

Success of combined spinal epidural will depend on familiarity with technique. It is more versatile to titrate the block and dose and also a faster onset compared to epidural alone. This technique can be useful for post operative analgesia and re-operative anaesthesia [84]. There is higher rate of success for surgical anaesthesia compared to spinal or epidural alone. Several studies have shown that catheters inserted as a part of combined spinal epidural technique produce anaesthesia /analgesia more reliably than those inserted via a standard epidural technique [85 – 88]. The appearance of cerebrospinal fluid indirectly confirms correct epidural needle placement and increase the chance of functional epidural catheter. There is a possible flaw when spinal injection alone produces the desired block and epidural remains untested; when epidural is required and fails, general anaesthetic might be needed [25] and hence a small dose intrathecally might be used to establish the analgesia to make mother pain free (which therefore also decreases the risk of hypotension) and then epidural should be used to make sure it is working for the complete surgical anaesthesia.

Continious Spinal anaesthesia

Operators need to be familiar with technique. Continuous spinal anaesthesia must be done always with consultant anaesthetist. It is occasionally used in patients who have accidental dural puncture. It may be used when epidural is indicated and difficult to site. It provides reliable and predictable block and allows to titrate the block to desired level and duration. It provides surgical anaesthetic level within minutes in emergency situations with incremental doses. It is important to flush the catheter before placement to avoid introducing air into the spinal space which could cause pneumoencephalus headache [25]. It is also very important to mark it as an intrathecal catheter and to be used by anaesthetist only. This can be used for analgesia as well as anaesthesia.

Incidence of headache and infection is higher with this technique compared to other regional techniques but overall incidence of post dural puncture headache in obese parturients is lower [89, 90]. Final density and level are proportional to the dose in mgs, not the volume delivered

GENERAL ANAESTHESIA FOR CAESAREAN

Consultant anaesthetist should be involved as early as possible. Strategy should be to avoid need for emergency general anaesthesia by being proactive and establishing effective regional analgesia and anaesthesia as early as possible. Airway assessment regarding difficult airway must be done. Preparation for general anaesthesia and difficult intubation (ensure lower sized endotracheal tube and a laryngeal mass airway) must be in place including awake fibre optic laryngoscope. Anti-aspiration prophylaxis must be given before conduct of anaesthesia

Collins et al. [91] investigated the effect of the position of the patient on the view obtained during laryngoscopy in 60 morbidly obese patients. They found that the 'ramped' position, accomplished by arranging blankets underneath the patient's upper body and head until horizontal alignment is achieved between the external auditory meatus and the sternal notch, clearly improves the laryngeal view when compared with the standard 'sniff' position. HELP (Head elevated laryngoscopy position) should be given to make sure that airway is in alignment.

After all monitoring including foetal monitoring is in place, patient must be prepared awake and draped. Adequate preoxygenation { 8 vital capacity breaths of 100% oxygen [92]} is ensured as otherwise they rapidly desaturate. Baraka et al. [92] showed that pre-oxygenation achieved by eight vital capacity breaths within 60 s at an oxygen flow of 10 liters/min not only results in a higher partial pressure of arterial oxygen (PaO_2), but also in a slower hemoglobin desaturation when compared with the four deep breaths technique. Use standard rapid sequence induction with cricoid pressure and left lateral tilt in patients with no anticipated difficult airway. For general anaesthesia make sure drug doses for (thiopentone, suxamethonium, atracuruim) are calculated before hand keeping in view altered distribution and elimination in obese patients. Dewan suggests that at least 4 mgs/kg of thiopentone (up to a maximum dose of 500 mgs) should be used if chosen, to avoid the risk of maternal awareness, hypertension and decreased uterine blood flow during light anaesthesia [93]. Administration of a larger dose may be associated with delayed arousal in the event of failed intubation. For suxamethonium, dose based on 1 - 2 mgs/kg of actual bodyweight up to maximum of 200 mgs [93].

Tracheal intubation should be confirmed by capnography in addition to auscultation. Endobronchial intubation should be promptly recognized and managed. In the event of failure to intubate the trachea after rapid sequence induction, it is imperative to institute a failed intubation drill without delay. Repeated attempts and a second dose of suxamethonium are seldom beneficial and often detrimental. The primary objective in the management of failed intubation is to ensure adequate maternal oxygenation despite the concerns of foetal wellbeing or risk of regurgitation [17].

Patients will need suitable ventilators for adequate ventilation. They need large tidal volumes of 10–12 mls/kg and positive end expiratory pressure (PEEP) may be avoided [25], as though it increases partial pressure of oxygen in blood (PaO2) [25], it might decrease cardiac output and oxygen delivery to foetus [25]. Extubation must be done when awake in left lateral position or semi upright position after adequate reversal of muscle relaxant

Antibiotic prophylaxis is a must as high incidence of wound infection in these patients [46, 47]. There is increased risk of post operative respiratory failure and hence morbidly obese parturients are best managed in intensive care management or high dependency care post operatively after general anaesthesia [75]. Adequate pain control (Patient controlled analgesia / patient controlled epidural analgesia (PCA/PCEA) to assure post op deep breathing. Infiltrative analgesia at the end of surgery can be carefully used to decrease requirement of post op analgesia. Post operative oxygen should be given and continuous positive airway pressure if required.

Thromboprophylaxis should be given after liasing with the obstetricians as to the dose and frequency required. Both pharmacological and mechanical methods and early mobilization should be used for thromboprophylaxis. It has been suggested that low molecular weight heparin (LMWH) dosing should be based on actual body weight [94].

The anticoagulation status of the patient becomes particularly important for the anesthesiologist when the patient has a spinal or an epidural catheter. According to European guidelines (when a single daily dosing of low molecular weight heparin (LMWH's) is used), catheters can be removed 10–12 hrs after the last dose of low molecular weight heparin (LMWH) and 4 hrs before the next dose.

Subcutaneous and Intramuscular routes of drug administration should be avoided as they are less reliable.

CONFLICT OF INTERESTS
None declared

AUTHOR DETAILS
NIMIT SHAH, FRCA, FFARCSI, DNB. Specialist registrar, Norfolk and Norwich hospital, Norwich NR4 7UY.
YAQUB LATOO FRCA. Consultant Anaesthetist, Bedford hospital, Bedford, MK42 9DJ.

CORRESPONDENCE: Dr Y Latoo, Consultant Anaesthetist, Bedford hospital, Kempston Road, Bedford, MK42 9DJ.
Email: yaqublatoo@aol.com

REFERENCES

1. Seidell JC. Epidemiology of obesity. Semin Vasc Med 2005; (1):3–14.
2. Friedman N et al. Overweight and obesity: an overview of prevalence, clinical impact, and economic impact. Dis Manag 2004; 7 (Suppl 1):S1–S6.
3. Malecka-Tendera E et al. Childhood obesity: a pandemic of the twentyfirst century. Int J Obes 2006; 30 (Suppl 2):S1–S3.
4. Deitel M. Some consequences of the global obesity epidemic. Obes Surg 2005; 15:1–2.
5. National Institutes of Health Consensus Development Conference Statement. Health implications of obesity. Ann Int Med 1985; 103: 147–51
6. Bray GA (1985) complications of obesity. Ann Intern Med 103:1052 – 1062
7. M Tanaka et al – Anesthetic management in obese parturient, J of Anesthesia (1999), 13:217 - 229
8. Kim SY, Dietz PM, England L et al. Trends in pre-pregnancy obesity in nine states, 1993–2003. Obesity (Silver Spring) 2007; 15: 986–93.
9. World Health Organization. Obesity: Preventing and Managing the Global Epidemic. Report on a WHO consultation, WHO Technical Report Series 894. Geneva: WHO, 2000
10. Department of Health, London. Health survey for England 2002 – trends [WWW document].
11. Naimark A, Cherniak R. Compliance of the respiratory system and its components in health and obesity. J Appl Physiol 1960; 15: 377–82.
12. Unterborn J. Pulmonary function testing in obesity, pregnancy, and extremes of body habitus. Clin Chest Med 2001; 22: 759–67.
13. Eng M, Butler J, Bonica JJ. Respiratory function in pregnant obese women. Am J Obstet Gynecol 1975; 123: 241–5.
14. Cullen JH, Formel PF. The respiratory defects in extreme obesity. Am J Med 1962; 32: 525–31.
15. Lee JJ, Larsen RH, Buckley JJ et al. Pulmonary function and its correlation to the degree of obesity in 294 patients. Anesth Rev 1981; 8: 28–32.
16. Vaughan RW, Cork RC, Hollander D. The effect of massive weight loss on   arterial oxygenation and pulmonary function tests. Anesthesiology 1981; 54: 325–8.
17. Saravanakumar K etal. Obesity and obstetric anaesthesia. Anaesthesia 2006; 61:36–48.
18. Lefcourt LA, Rodis JF. Obstructive sleep apnea in pregnancy. Obst Gynecol Surv 1996; 51: 503–6
19. Chang AB. Physiologic changes of pregnancy. In: Chestnut DH, ed. Obstetric anesthesia: principles and practice, 3rd edn. Philadelphia: Elsevier Mosby, 2004: 15–36.
20. Øberg B, Poulsen TD. Obesity: an anaesthetic challenge. Acta Anaesthesiol Scand 1996; 40: 191–200.
21. Stephansson O et al. Maternal weight, pregnancy weight gain and the risk of antepartum stillbirth. American Journal of Obstetrics and Gynecology 2001; 184: 463–9.
22. Veille JC, Hanson R. Obesity, pregnancy, and left ventricular functioning during the third trimester. Am J Obstet Gynecol 1994; 171: 980–3.
23. Vasan RS. Cardiac function and obesity. Heart 2003; 89: 1127–9.
24. Tomoda S, Tamura T, Sudo Y et al. Effects of obesity on pregnant women: maternal hemodynamic change. Am J Perinat 1996; 13: 73–8.
25. J Mhyre – anaesthetic management of morbidly obese pts
26. Tsueda K et al, Obesity supine death syndrome: reports of two morbidly obese patients. Anesthesia and Analgesia 1979; 58: 345–7.
27. Mendelson CL. The aspiration of stomach contents into the lungs during obstetric anesthesia. Am J Obstet Gynaecol 1945; 52: 191–204.
28. Olsson GL, Hallen B, Hambraeus-Jonzon K. Aspiration during anaesthesia: a computer-aided study of 185358 anaesthestics. Acta Anaesthesiol Scand 1986; 30: 84–92.
29. Brock Utne JG, Dow TG et al gastric and lower oesophageal pressure in early pregnancy. Br J Anaestheisa 1981; 53: 381 -384
30. Obrien TG Jr; LOS pressure and oesophageal function in obese humans J clin gastroenterology. 1980; 2:145 -148
31. Roberts RB, Shirley MA – reducing the risk of acid aspiration during caesarean section, anaesthe analgesia 1974; 53: 859 - 868
32. A . Casati , M . Putzu, Anesthesia in the obese patient: Pharmacokinetic considerations . Journal of Clinical Anesthesia , Volume 17 , Issue 2 , Pages 134 - 145
33. Hodgkinson R, Husain FJ. Obesity and the cephalad spread of analgesia following epidural administration of bupivacaine for cesarean section. Anesth Analg 1980; 59: 89–92.
34. Hogan QH, Prost R, Kulier A et al. Magnetic resonance imaging of cerebrospinal fluid volume and the influence of body habitus and abdominal pressure. Anesthesiology 1996; 84: 1341–9.
35. Hackney JD, Crane MG, Collier CC et al. Syndrome of extreme obesity and hypoventilation: studies of etiology. Ann Int Med 1960; 51: 541–52.
36. Greene NM. Distribution of local anesthetic solutions within the subarachnoid space. Anesth Analg 1985; 64: 715–30.
37. Greene NM Distribution of local anesthetics within the subarachnoid space. Anesthesia and Analgesia 1985;64:715 -30
38. Maternal obesity and pregnancy outcome: a study of 287 213 pregnancies in London N J Sebire et al International Journal of Obesity (2001) 25, 1175 - 82
39. Gabbe SG : Gestational Diabetes mellitus NEJM 315 (1986) 1025
40. Kumar AS – Pregnancy outcome in women with morbid obesity J Gynaecology Obstetric 97 (2001) 101

41. Gross T etall - Obesity in pregnancy : risks and outcome. Obstet Gynaecol 56 (1980) 446

42. Johnson SR et al. Maternal obesity and pregnancy. Surg Gynaecol Obstet 164: 431 -7

43. Ehrenburg HM et al. The influence of obesity and diabetes on the prevalence of macrosomia. American Journal of Obstetrics and Gynecology 2004; 191: 964–8.

44. Weiss et all – Obesity, obstret complications and caesarean delivery rate American J Obstetr GYnaec 190 (2004) 1091 -7

45. Hood DD et al. Anesthetic and obstetric outcome in morbidly obese parturients. Anethesiology 1993; 79:1210–1218.

46. Usha Kiran TS et al. Outcome of pregnancy in a woman with an increased body mass index. British Journal of Obstetrics and Gynaecology 2005; 112: 768–72.

47. Myles TD et al. Obesity as an independent risk factor for infectious morbidity in patients who undergo cesarean delivery. Obstetrics and Gynecology 2002; 100: 959– 64.

48. Andreasen KR et al. Obesity and pregnancy. Acta Obstetricia et Gynecologica Scandinavica 2004; 83: 1022–9.

49. Galtier-Dereure F et al. Obesity and pregnancy: complications and cost. American Journal of Clinical Nutrition 2000; 71 (Suppl.): S1242–8.

50. Obesity in pregnancy. Yu CK, Teoh TG, Robinson S BJOG. 2006 Oct;113(10):1117-25. Epub 2006 Aug 10

51. Watkins ML et al. Maternal obesity and risk for birth defects. Pediatrics 2003; 111: 1152–8.

52. Stephansson O et al. Maternal weight, pregnancy weight gain and the risk of antepartum stillbirth. American Journal of Obstetrics and Gynecology 2001; 184: 463–9.

53. Cedergren Mi : Maternal morbid obesity and risk of adverse pregnancy outcome Obstetr Gynecol 103 (2004) 219

54. Weiss JL, Malone FD, Emig D et al. Obesity, obstetric complications and cesarean delivery rate – A population-based screening study. Am J Obstet Gynecol 2004; 190: 1091–7.

55. Munnur U et al , Airway problems in pregnancy. Crit Care Med 2005; 33 (10 Suppl):S259–S268.

56. Schneider MC. Anaesthetic management of high-risk obstetric patients. Acta Anaesthesiol Scand 1997; 41 (1 Suppl 111):163–165.

57. Juvin P, et al. Difficult tracheal intubation is more common in obese than lean patients. Anesthesia and Analgesia 2003; 97: 595 – 600.

58. D'Angelo R, Dewan DD. Obesity. In: Chestnut DH, ed. Obstetric Anesthesia: Principles and Practice, 2004: 893–903

59. Merah NA et al. Prediction of difficult laryngoscopy in a population of Nigerian obstetric patients. West African Journal of Medicine 2004; 23: 38–4.

60. Rocke DA et al,. Relative risk analysis of factors associated with difficult intubation in obstetric anesthesia. anesthesiology 1992; 77: 67–73.

61. Bell RL et al. Postoperative considerations for patients with obesity and sleep apnea. anaesthesiol clin N Am 2005; 23:493–500

62. Shankar KB et all – airway changes during pregnancy 1997 – anaesthesiology 87: A895

63. ACOG committee opinion number 315. Obesity in pregnancy – Obstetr Gynaecol 2005; 106:671 -75

64. Lean ME. Obesity and cardiovascular disease: the waisted years. British Journal of Cardiology 1999; 6: 269–73.

65. Hess PE, Pratt SD, Lucas TP et al. Predictors of breakthrough pain during labor epidural analgesia. Anesth Analg 2001; 93: 414–8.

66. Howell CJ. Epidural versus non-epidural analgesia for pain relief in labour (Cochrane Review). In: The Cochrane Library, Issue 4. Chichester: John Wiley & Sons, Ltd., 2004.

67. Von Ungern-Sternberg BS et al, The effect of epidural analgesia in labour on maternal respiratory function. Anaesthesia 2004; 59: 350–3.

68. Cascio M, et al. Labour analgesia with intrathecal fentanyl decreases maternal stress. Canadian Journal of Anaesthesia 1997; 44: 605–9.

69. Patel J. Anaesthesia for LSCS in a morbidly obese patient. Anaesthesia and Intensive Care 1999; 27: 216–9.

70. Chetty V et al - Difficult spinal anaesthesia for caesarean section in two obese pregnant patients. West African Journal of Medicine 2001; 20: 274–6.

71. Jordan H, Perlow MD, Mark A, Morgan MD. Massive maternal obesity and perioperative cesarean morbidity. American Journal of Obstetrics and Gynecology 1994; 170: 560–5.

72. Vincent RD, Chestnut DH. Which position is more comfortable for the parturient during identification of the epidural space? International Journal of Obstetric Anesthesia 1991; 1: 9–11.

73. Hamza J et al Parturient's posture during epidural puncture affects the distance from skin to epidural space. Journal of Clinical Anesthesia 1995; 7: 1–4.

74. Robinson HE, O'Connell CM etall: Maternal outcomes in pregnancies complicated by obesity. Obstet Gynecol 2005; 106: 1357 - 64

75. Manuel C Vallejo – Anaesthetic management of the morbidly obese parturient; Current opinion in Anaesthesiology 2007, 20: 175 - 180

76. Clinkscales CP et all An observational study of the relationship between lumbar epidural space depth and BMI in Michigan parturients IJOA 2007;16:323-7

77. Watts RW. The influence of obesity on the relationship between body mass index and the distance to the epidural space from the skin. Anaesthesia and Intensive Care 1993; 21: 309–10.

78. Wallace DH, Currie JM. Indirect sonographic guidance for epidural anesthesia in obese pregnant patients. Regional Anesthesia 1992; 17: 233–6.

79. Grau T, et Ultrasound imaging improves learning curves in obstetric epidural anesthesia: a preliminary study. Canadian Journal of Anesthesia 2003; 50: 1047–50.

80. Melzack R- Severity of labour pain : influence of physical as w ell psychological variables Can Med Asso J 1984; 130:579 -584

81. Hawkins JL. Maternal mortality: anesthetic implications. Int Anesthesiol Clin 2002; 40:1–11.

82. Lewis G (ed). Confidential Enquiries into Maternal and Child Health. Why Mothers Die. The Sixth Report of the United Kingdom Confidential Enquiries into Maternal Deaths in the United Kingdom. London. RCOG Press. 2004. www.cemach.org.uk

83. Rawal N, Holmstrom B, Crowhurst JA et al. The combined spinal–epidural technique. Anesth Clin North Am 2000; 18: 267–95.

84. Kuczkowski KM, Benumof JL. Repeat Caesarean section in a morbidly obese parturient: a new anaesthetic option. Acta Anaesthesiologica Scandinavia 2002: 46: 753 – 4

85. van de Velde M, Teunkens A, Hanssens M et al. Post dural puncture headache following combined spinal epidural or epidural anesthesia in obstetric patients. Anaesth Intensive Care 2001; 29: 595–9.

86. Pan PH, Bogard TD, Owen MD. Incidence and characteristics of failures in obstetric neuraxial analgesia and anesthesia: a retrospective analysis of 19,259 deliveries. Int J Obstet Anesth 2004; 13: 227–33.

87. Norris MC. Are combined spinal–epidural catheters reliable? Int J Obstet Anesth 2000; 9: 3–6.

88. Eappen S, Blinn A, Segal S. Incidence of epidural catheter replacement in parturients: a retrospective chart review. Int J Obstet Anesth 1998;7: 220–5.

89. Coker LL Continuous spinal anaesthesia for Caesarean section for a morbidly obese patient: a case report. AANA J 2002;70: 189 -192

90. Faure E et al Incidence of postdural puncture headache in morbidly obese parturients. Reg Anesth 1994; 19: 361-363

91. Collins JS, Lemmens HJ, Brodsky JB et al. Laryngoscopy and morbid obesity: a comparison of the 'sniff' and 'ramped' positions. Obes Surg 2004; 14: 1171–5.

92. Baraka AS, Taha SK, Aouad MT et al. Preoxygenation – comparison of maximal breathing and tidal volume breathing techniques. Anesthesiology 1999; 91: 612–6.

93. Bray GA (1985) complications of obesity. Ann Intern Med 103:1052 – 1062

94. Michota F, Merli G. Anticoagulation in special patient populations: are special dosing considerations required? Clev Clin J Med 2005; 72 (Suppl. 1): S37–42.

Viewpoint

Depression and Iatrogenic Hopelessness

Jaleel Khaja

"That element of tragedy which lies in the very fact of frequency, has not yet wrought itself into the coarse emotion of mankind; and perhaps our frames could hardly bear much of it. If we had a keen vision and feeling of all ordinary human life, it would be like hearing the grass grow and the squirrel's heart beat, and we should die of that roar which lies on the other side of silence". (George Elliot)

I wonder what would be the magnitude of the roar on the other side of silence when Depression, as WHO has it, becomes the second most common disabling condition worldwide by 2020[1]. I don't know about others but I myself would be relying on my chronic ailment of selective deafness to remain blissfully unaware of the enormity of emotional pain and ceaseless suffering, if these were to penetrate into the much narrowed range of my sensitivity. I am sure my wise colleagues won't die of the roar either and would have plenty of effective safeguards to choose from. You might, however, argue that these measures may be gratuitous, as the emotional coldness, which is endemic in our circles, may suffice. You may have a point.

Okay let me cut out the rhetoric and tell you what it is basically about. Depression is a major health problem[2] and 6% of the population meet the criteria for the disorder or Dysthymia at any one time[3]. In individuals between 15-44 years Depression accounts for 10% of all DALYS (disability-adjusted life-year), which is projected to rise to 15% by 2020, making Depression second only to Ischemic Heart Disease in terms of worldwide disease burden[4]. The irony is, while the time-bomb is ticking away and the race against time is underway, my learned colleagues seem to be engrossed in an endless duel over Antidepressants. "The term 'antidepressants' is a misnomer" and 'they are not effective at all'[5] sums up the stand 'the Critical Psychiatry Network' takes on this group of medications in particular while others dig their heels and maintain that 'Antidepressants do work and there is lack of evidence for Cognitive Behavioural Therapy in mild Depression'[6]. The debate has raged on for decades now and this year it really came to a head when a major publication[7] made a news headline writing off Antidepressants nearly completely and then only three months later we had fresh guidelines by another high impact association[8] recommending their use.

It might sound alarmist but these days patients would be running a huge risk of getting bogged down by diametrically opposite views if they spend more than a few minutes on the internet only trying to find help. People with Depression may very likely find the College recommendations[9] on when and why to use Antidepressants as straightforward and easy to follow. But if they push a search engine to full throttle they will soon find themselves on a long and bumpy road that basically leads to nowhere. They may suddenly stumble on a view that 'meta-analyses show selective serotonin reuptake inhibitors have no clinically meaningful advantage over placebo'[10] after negotiating a number of trials suggesting otherwise. With a couple of further clicks on the keyboard they, like myself, will find themselves struggling to see any concordance between the argument that the effect of Antidepressants could be exaggerated by their sedative property[10] and the conclusion that 'Insomnia and REM sleep suppression were reported with all SSRIs'[11]. Surfing on they may even start to wonder whether the beneficial effects of a whole range of medications like Antihypertensives, Antiepileptics, Antibiotics, Hypoglycaemics etc. could be exaggerated, if they come across a view that 'unblinding effects' (for example those due to side effects) 'may inflate the efficacy of antidepressants in trials using inert placebos'[12]. The doubt would be perfectly legitimate because the principle of testing medications against 'active placebos' appears to be so neatly applicable right across the board and naturally begs the question whether it is applied generally in actual practice. Unfortunately, after a while the answer to the question whether the Antidepressants are beneficial or not will start to appear ever so elusive and it won't be long afterwards that a deep sense of disappointment will set in, making these desperate searchers to log off from the Net as well as hope.

Dividing the whole of Psychiatry into two warring parties is not what I have intended to do in the above lines. That would be too simplistic. My attempt is only about trying to get a couple of messages across to all of us who claim to have patients´ best interest at heart. I cannot think of any other way to convey the crux of my first message than to take a quote from 'War is a Racket'[13]. The author says in the book, 'For a great many years, as a soldier, I had a suspicion that war was a racket; not until I retired to civil life did I fully realize it'. So while we maintain a high quality of our research studies about treatments in Psychiatry by employing rigorous methods, observing scientific detachment and keeping every enquiry dispassionate, it is imperative that we remain on guard against the possibility of us turning into inadvertent racketeers for those who are only driven by their monstrous appetite for profiteering.

My second message is specifically for 'the Critical Psychiatry Network' and to those of similar persuasion. I have had an honour to work with one of your main proponents and I can safely claim that you are a group of exceptionally talented Psychiatrists. You are doing a great job of saving Psychiatry against a potential risk of it simply degrading into some kind of quackery. However, I am not entirely comfortable with the unidirectional nature of your works, which appear to be mainly focussing on disproving positive claims made about the efficacy of treatments. It is about time that an equal amount of your energy and time is devoted towards establishing what can effectively treat Depression and help rekindle hope.

AUTHOR DETAILS
JALEEL KHAJA, MRCPsych, Staff Grade Psychiatrist,
Northamptonshire Healthcare NHS Trust
CORRESPONDENCE: 90 Heathfield Park Drive, Essex RM6 4FJ
Email: jkhaja@yahoo.com

REFERENCES:

1. Murray, C. & Lopez, A. (1996) *The Global Burden of Disease.* Cambridge, MA: Harvard University Press.

2. Greenberg, P., Stiglin, L., Finkelstein, S., *et al* (1993) The economic burden of depression in 1990. *Journal of Clinical Psychiatry*, 54, 405–418.

3. Keller, M., Lavori, P., Mueller, T., Keller, M., Lavori, P., Mueller, T., *et al* (1992) Time to recovery, chronicity and levels of psychopathology in major depression: a 5 year prospective follow-up of 431 subjects. *Archives of General Psychiatry*, 49, 809–816.

4. Murray, C. & Lopez, A. (1996) *The Global Burden of Disease.* Cambridge, MA: Harvard University Press.

5. Joanna Moncrieff Canadian Journal of Psychiatry Feb. 2007;52,2; Pro Quest Medical Library pg 96.

6. SAGE Press Release in connection with BAP guidelines published in May 2008.

7. Irving Kirsch *et al, Initial Severity and Antidepressant Benefits: A Meta-Analysis of Data Submitted to the Food and Drug Administration;* PLoS Medicine, Feb. 2008, Vol. 5, Issue 2.

8. Anderson *et al,* Journal of Psychopharmacology May 2008 http://jop.sagepub.com/cgi/rapidpdf/0269881107088441v2

9. www.rcpsych.ac.uk

10. Joanna Moncrieff *Canadian Journal of Psychiatry* Feb. 2007;52,2; Pro Quest Medical Library pg 96.

11. Mayers A G, Baldwin D S, *Antidepressants and their effect on sleep;* Human Psychopharmacology: Clinical and Experimental 2005 Vol. 20(8), (Database of Abstracts of Reviews of Effects (DARE)).

12. Moncrieff J, Wessely S, Hardy R, *Active placebo versus antidepressants for depression;* January 2004 Systematic Review (Cochrane).

13. Major General Smedley D Butler (1898-1931), United States Marine Corps, author of *War is a racket.* http://www.lexrex.com/enlightened/articles/warisaracket.htm

Case Series

Pathological Fractures as the Presenting Symptom of Parathyroid Adenoma: A Report of Three Cases.

Rajesh Rachha

ABSTRACT

Primary Hyperparathyroidism is usually diagnosed as an incidental finding of hypercalcemia in blood tests or due to symptoms secondary to the high calcium. Overt bone disease is an extremely rare presentation. Fractures in hyperparathyroidism are unusual and usually affect the vertebrae. Pathological fractures have been described in patients with parathyroid carcinoma. We report on three patients with benign parathyroid adenoma who primarily presented with pathological fractures of long bones. Excision of the parathyroid adenoma and immobilization of the fractures resulted in the return of the serum chemistry to normal, improvement of bone density and fracture union.

INTRODUCTION

Patients who have untreated primary hyperparathyroidism with Ostitis Fibrosa Cystica have become a rarity. Primary hyperparathyroidism is usually diagnosed as a result of chance finding of raised serum calcium or complications associated with hypercalcemia such as polyuria, polydipsia, muscle weakness, gastrointestinal upsets and renal stone formation. Bone disease is rarely overt. Radiographic manifestations are seen in less than 2% of patients and include subperiosteal erosions, diffuse osteoporosis, cystic lesions (brown tumours), pathological fractures, 'salt and pepper' mottling of skull and loss of lamina dura in the mandible[1].

The reported incidence of fractures in hyperparathyroidism is quite low, about 10% in two large series[2] and apart from vertebral compression fractures, no characteristic fracture pattern have been described.

Extensive bony involvement with pathological fractures as a presenting feature due to parathyroid carcinoma has been documented[3], but multiple pathological fractures, as a presenting feature of primary hyperparathyroidism due to parathyroid adenoma is extremely rare. Here we describe three patients seen at our institution in the period from 2001 to 2004 who presented with pathological fractures due to parathyroid adenoma.

CASE 1

A 50-year-old housewife was admitted with painful right thigh and inability to weight bear following a twisting injury of her right leg. She also complained of generalized weakness, lethargy and muscle pain over the past few months. Radiographs revealed segmental fracture of right femur (figure 1a) and gross osteopenia with a cortical index of 0.2 (normal: > 0.45). She also sustained a fracture of left femur following a very trivial injury while transferring her from trolley to bed.

Investigations (table–1) revealed hypercalcemia (12Mg/dl), hypophosphatemia (2.6Mg/dl) and elevated parathyroid hormone level (70 Pmmol/l). 25-hydroxyvitamin D value was in the lower limit of normal (22ng/ml). Renal parameters were normal.

High-resolution ultrasound (HRUS) of neck revealed a hypoechoic mass measuring 4.1 x 1.7 cm in the posterior aspect of right thyroid lobe, suggestive of parathyroid adenoma. Bone scan showed patchy tracer uptake in almost entire skeleton with generalised osteoporosis and microfractures, suggestive of metabolic bone disease.

Figure 1a. Gross osteopenia with segmental pathological fractures of right femur.

Patient underwent parathyroid adenectomy under the general surgeons. Histopathology confirmed the diagnosis of parathyroid adenoma.

Figure1b. 12 Months post Parathyroid adenectomy showing improved bone density and Mallunion of fractures.

Following surgery patient developed hypocalcemic tetany and seizures, treated with intravenous calcium gluconate and followed by oral calcium supplements. Fractures were treated conservatively by splinting in groin to toe casts, as the bone quality was very poor. The biochemical tests came back to normal three months after surgery and radiographs revealed fracture healing and improvement in bone density. Casts were removed and mobilization commenced. Over next six months fractures healed but were mall-united (figure 1b). At eighteen months the patient was fully weight bearing with minimal functional disability.

CASE 2

A 32-year-old female patient presented with pain in the right thigh and inability to weight bear after a trivial fall at home. Radiographs revealed fracture middle one-third of right femur, osteopenia and with subperiosteal resorption (figure 2a)

Figure 2a. severe osteopenia with pathological fracture of right femur.

Biochemical tests and parathyroid hormone assay (see table-1) was suggestive of primary hyperparathyroidism. 25-hydroxyvitamin D was within normal limits (35ng/ml). High-resolution ultrasound of neck revealed hypoechoic lobulated lesion measuring 8.4x1.1x1.3 cm, on the inferior and posterolateral aspect of the left lobe of thyroid suggestive of parathyroid adenoma. Bones scan showed tracer uptake at the

site of fracture and patchy sclerosis of femur, and increased uptake in left sacroiliac joint.

Figure 2b. Fracture union with intramedullary nail insitu.

The patient underwent parathyroid adenectomy under the care of general surgeons. Histopathology confirmed the diagnosis of parathyroid adenoma. Patient received parentaral calcium supplements in the immediate post operative period and later by oral route. Femur fracture was initially treated in groin to toe cast and the calcium levels were controlled, after 2 months bone quality improved, despite callus formation there was mobility at the fracture site, and hence the fracture was managed with intramedullary nail and bone grafting (figure 2b).

It took ten months for the fracture to unite.

CASE 3

A 20-year-old female patient presented with diffuse pain in the left elbow of two months duration following a trivial injury to her elbow. Plain radiograph of the elbow showed a well-defined lytic lesion with sub cortical erosions and with break in the cortex suggestive of Brown tumour with pathological fracture (figure 3a).

Figure 3a. Brown tumour lower end of humerus with pathological fracture

Biochemical analysis revealed hypercalcemia and hypophosphatemia and parathyroid hormone assay was suggestive of primary hyperparathyroidism (table-1). 25-hydroxyvitamin D levels were within normal limit (40ng/ml). High-resolution ultrasound neck showed 2.6 X 1.1 X 1.6 cm hypo echoic mass lesion inferior to lower pole of thyroid suggestive of left parathyroid adenoma.

Bone scan showed patchy increased tracer concentration in entire skeleton with increased tracer concentration in the distal end of humerus.

She underwent parathyroid adenectomy and histopathological examination confirmed the diagnosis of parathyroid adenoma. The fracture was managed in a cast.

Figure 3b. One year post parathyroid adenectomy shows healed fracture with increased bone density and sclerosis.

Patient was followed up with regular check on her calcium levels and serial radiographs of elbow. By 12 months the fracture was completely healed and brown tumour resolved with increased bone density and sclerosis (figure 3b).

	Ca++ Mg/dl	Phos Mg/dl	Alk. Phos. IU/L	Intact PTH Pmmol/l	25-hydroxy Vit D ng/ml
Normal	8 -10	3 - 4.5	34-135	1.1 – 6.5	20 - 56
Case 1	12	2.6	3000	70	22
Case 2	11.5	2.0	745	56	35
Case 3	10.5	2.5	1184	48	40
TABLE –1 Serum biochemical parameters in the patients at presentation					

DISCUSSION:

Primary hyperparathyroidism is a well-recognised entity identified almost more than a century ago by Von Recklinghausen. He and his co-workers coined the term Osteitis fibrosa cystica[1].

This condition is more common in females. Peak age incidence is between 30 to 50 years and incidence increases with age, though patient aged as young as 14 years was documented. In U.S.A annual incidence is around 0.2% in patients > 60 years[1]. All the patients in our series are females with florid changes observed in-patient aged 52 years other 2 patients are aged 20 years and 32 years.

Disease results from excessive secretion of parathyroid hormone either due to solitary (50-85%) or multiple (10%) adenomas, hyperplasia (10-40%), or rarely due to a carcinoma of a single parathyroid gland. Extensive bony involvement with pathological fractures as a presenting feature due to parathyroid carcinoma has been documented[3]. In our series of 3 patients all of them were diagnosed to have solitary parathyroid adenoma. Our first patient aged 52 years had coexisting vitamin D deficiency, which explains severe osteomalacia and multiple fractures. Coexistence of vitamin D deficiency in patients with Primary Hyperparathyroidism may put the patient at a significant higher risk of loosing bone mineral density and development of osteoporosis.

Two distinct types of bone lesions are described in primary hyperparathyroidism[4]. The slowly progressive type- leads to cortical thinning and osteoporosis & the rapidly progressive type. Pathological fractures may occur through a cyst or in a weakened long bone. A principal test at present is the 'Immunoassay' for PTH 1-84 as it distinguishes the hypercalcaemia of malignancy from that of hyperparathyroidism[5].

Once the diagnosis of primary hyperparathyroidism has been made by biochemical analysis, the site or sites of adenomatous or hyperplastic parathyroid tissue must be identified[6]. Some authors advocated 'Exploratory neck operation' as most adenomas are localized in the neck[7]. CT scan and Thallium subtraction scans are useful for detecting parathyroid pathology in normal as well as ectopic locations.

USG of neck can be helpful in picking abnormal parathyroid tissue but CT scan and MRI are more sensitive to assess ectopic sites[8]. In our series of 3 cases, High Resolution Ultrasound Scan (HRUS) of neck helped in localizing the parathyroid adenoma (90% of adenomas are in the neck). Excised parathyroid gland has to be subjected to histopathological examination to confirm and differentiate adenoma, hyperplasia and malignancy. Histopathological examination in all our 3 cases confirmed the diagnosis of parathyroid adenoma.

Our experience with fractures in primary hyperparathyroidism revealed that these take longer to heal and are prone to malunion unless splinted internally or externally. Average time taken for fracture union in our series was 12 months. Non-union of fractures is rare and healing proceeds uneventfully after excision of an adenoma.

Bone histology returns to normal within 5-6 weeks[6]. Brown tumours usually resolve with increase in bone density and sclerosis after parathyroid adenectomy.

The extensive skeletal involvement due to hyperparathyroidism has rarely been reported. The substantial improvement in bone density, in promotion of fracture healing and in preventing pathological fractures after successful parathyroid adenectomy has been demonstrated in our series of 3 cases.

CONCLUSION:

In conclusion, a high index of suspicion is necessary to diagnose this unusual presentation of primary hyperparathyroidism. A pathological fracture in young lady with marked osteopenia is highly suggestive. A combination of biochemical tests, including serum levels of calcium, phosphorus, alkaline phosphatase and parathormone assay will help in diagnosing primary hyperparathyroidism in 90% of the cases. All patients with Primary Hyperparathyroidism should have Vitamin D level assessment in order to exclude the coexistance of Vitamin D deficiency with Primary Hyperparathyroidism. High Resolution Ultrasound Scan of neck provides valuable preoperative information in selected cases especially in those undergoing minimally invasive parathyroid surgery. Surgical excision and calcium supplementation along with external or internal splinting of fractures allowed the fractures to heal.

AUTHOR DETAILS:
RAJESH RACHHA, Diploma (Orthopaedics), MRCS. Senior Clinical Fellow, Bedford hospital NHS trust, United Kingdom
CORESSPONDENCE: Dr R Rachha, Bedford Hospital, Kempston Road, Bedford, MK42 9DJ.
Email: drrajeshracha@yahoo.com

REFERENCES:

1. Henry,J.Mankin.: An instruction course lecture- Metabolic bone disease. The American Academy of orthopaedic surgeons. Journal of Bone & Joint Surgery; 1994;Vol- 76A, No.5; 760-788.

2. Chalmers J, Irvine GB: Fractures of the femoral neck in elderly patients with hyperparathyroidism. Clin Orthop Related Res. 1988 Apr;(229): 125-130

3. R.G.Deshmukh., S.A.L.Alsagoff., S.Krishnan et al. Primary hyperparathyroidism presenting with pathological fracture. The Royal College of Surgeons of Edinburgh, December 1998; Vol-43, 424-427.

4. Lancourt JE, Hochberg F. Delayed fracture healing in primary hyperparathyroidism Clin OrthoP 1977; 124: 214-218

5. Nussbaum,S.R., & Polt,J.T., Jr.: Immunoassays for parathyroid hormone 1-84 in the diagnosis of hyperparathyroidism. J Bone and Min. Res., 6 (supplement 2); s43- s50, 1991.

6. George,D.C., Incaro,S.J., Devlin,J.T et al .: Histology of bone after parathyroid adenectomy – A case report. J Bone & Joint Surgery .1990;Vol 72A, No: 10 ; pp 1558- 1561.

7. Satava,R.M., Jr., Beahrs, O.H., and Scholz, D.A.: Success rate of cervical exploration for hyper parathyroidism. Arch.Surg., 1975;Vol-110. 625-628.

8. Winzelberg,G.G.: Parathyroid imaging. Ann.Intern. Med.,1987;Vol-107: 64-70.

Audit Report

Quality of Electronic Discharge Summaries at Newham University Hospital: An Audit

Syeda M. B. Kazmi

INTRODUCTION

The effectiveness and quality of care for patients is largely dependent on communication between physicians both in secondary care and in primary care. Written communication between secondary and primary care in the form of a discharge summary is vitally important for informing General Practitioners (GPs) and other healthcare professionals of the details regarding patients' admission into hospital, as well as ensuring there is continuity of care in the community. Discharge summaries are often the only form of communication that occurs at the transition between secondary and primary care.

Prior to discharging patients from hospital, a discharge summary (whether this be hand written or typed) is required to be completed. Ideally copies are kept in patient files and given to the patient, as well as forwarding a copy to the GP. Correctly completing all relevant sections on a discharge summary is part of good medical practice, Clinical Governance and hospital/patient documentation.

Historically, discharge summaries have been found to be poorly written, contain inaccurate and ineffective information. Several studies have identified areas which are lacking in discharge summaries when looking at the quality of the discharge summary. These include: inadequacies of medical evaluation, level of experience of the discharge author and accuracy. Subsequently, there has been a general move towards electronic discharge summaries with a standard format.

Advantages of EPR over a paper-bases system
- More information included
- Do not need to later type or dictate a formal letter
- Permanent electronic record
- Available immediately
- Always legible
- Full details with GP at time of discharge
- Allows more accurate clinical coding

Disadvantages of EPR
- Takes longer to complete than a paper summary

In April 2005, Newham University Hospital Trust (NUHT) began using the Cerner Millennium Electronic Patient Record (EPR) system. This electronically stores information about a patient, for example discharge summaries, previous blood results, imaging results etc. It is intended that all discharge summaries are typed directly onto this computerized system. The summaries are then available to view by all authorized parties anywhere in the hospital. This system has the advantage of enabling access to portions of a patient's medical record whilst waiting for patients' old notes. Furthermore, more information can be added to the discharge summaries contemporaneously or even after the point of discharge. Table 1. Shows some advantages and disadvantages of EPR vs paper discharge summaries.

AIMS AND OBJECTIVES

This study assessed the quality of discharge summaries completed by medical specialties at Newham University Hospital Trust using the EPR system. The aim was to identify any problems and where they are occurring. The information gained would help in addressing any problems identified, to improve the quality of discharge summaries.

METHODS

I had an in-depth discussion with my supervisor about the feasibility, methodology, data collection, patient confidentiality, ethics and relevance of this audit to the Hospital.

I undertook a thorough literature search in Medline and other internet searches, reviewed the Journals in NUHT Library for similar audits which looked at the quality of discharge summaries.

A sample of 100 Medical (respiratory, endocrine and gastroenterology) and Care of the Elderly (CoE) discharge summaries of patients from NUHT were retrospectively audited. The hospital Audit Department provided me with the names of the last 100 patients who had been discharged from Medicine and CoE during October 2007. These were sequential discharges, not selected at random. We did not analyze any patients from the specialties of Surgery, Gynaecology, Paediatrics, Cardiology or Emergency Medicine.

After obtaining the names, the discharge summaries were analysed and information was collected on the following fields:

- Name of Consultant on the discharge summary sheet
- Which team the consultant belonged to
- If the discharge consultant was correct
- If date of admission had been completed
- If date of discharge had been completed
- If the patient was given a diagnosis
- If a follow up appointment was suggested and what type of follow up this was (GP or NUHT or another hospital)
- If a follow up appointment had been made
- If the discharge summaries had been signed and bleep number provided

This data was then tabulated using simple statistical analysis (mainly descriptive) and the results calculated into a percentage.

The names of the consultants on the discharge summaries were divided into Medical, Care of the Elderly and Accident & Emergency. The medical team was further divided into team A, B and C, according to the specialty they worked under, for example Team A: endocrine, team B: gastroenterology, and team C: respiratory medicine. By dividing the consultants under different teams allowed me to confirm if they were the right consultant. A&E was included into this field as many discharges still have A&E consultants on them despite the patients being admitted to hospital.

This audit did not address the following issues:
1. If the GP is correct
2. If the GP received the summary
3. If the GP made follow up arrangements post discharge
4. The accuracy of the diagnosis
5. If the patient has more than one electronic medical records

RESULTS

The examination of 100 medical records yielded 94 discharge summaries available for audit, leaving 6 medical records with no evidence of a discharge summary.

As mentioned above, the consultants on the discharge summary were divided into the following categories. The number beside them represents how many discharge summaries belonged to each respective team.

> Care of the elderly team: 46
> Adult medicine: 36
> A&E: 9
> Other: 3

It was found that 57(60.6%) of the discharge summaries contained the correct consultant name. However, on 22(23.4%) of the discharge summaries it was unclear if the discharge consultant was correct.

**Correct Discharge Consultant**
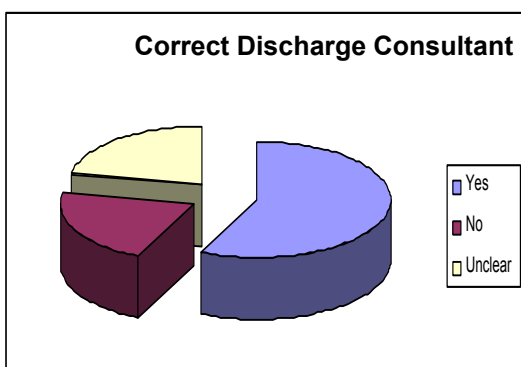
From the 96 discharge summaries completed, every single summary had an admission date on it, however, only 75(79.8%) of discharge summaries had a discharge date.

Seventy five (79.8%) of discharge summaries had been signed by the author (with their name), but only 71(75.5%) had wrote their bleep number.

Sixty five (69.1%) summaries were identified to have a diagnoses under the heading of acute problems, whereas only

22(23.4%) had only symptoms. The remaining 13 (13.8%) summaries had no diagnosis or symptoms completed.

Finally, when analyzing the discharge summaries regarding follow up arrangements, 91(96.8%) discharge summaries had a follow up suggested, of which 27(29.7%) were to be followed up by GP, 59(64.8%) were to be followed in NUHT and 5 patients were to be followed up at another hospital.

Follow up Arrangements
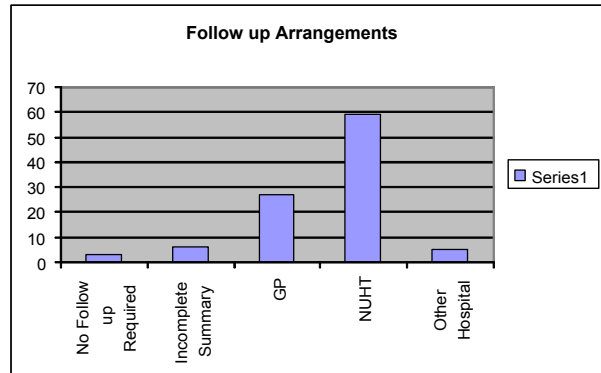
Of the discharge summaries which had follow up arranged in NUHT, only 40(67.8%) patients had a follow up appointment made.

Appointment made at NUHT
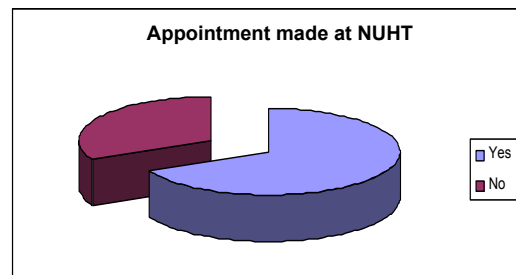
DISCUSSION

This study supports previous studies, confirming that a new approach to discharge summary completion is required. One of the main problems identified in this summary, was the use of incorrect consultants on the discharge summary. The current method used is clearly not effective; therefore it is important that the author completing the discharge summary ensures that the correct consultant is on the summary.

Changing the Consultant name on the discharge summaries is of great importance because this means that GPs are able to refer patients back to the correct consultants when seeking advice or trying to arrange further follow up with that consultant. Furthermore, it allows the appropriate National Health Service (NHS) funding to be given to the relevant department.

It was unclear in 22 discharge summaries if the discharge Consultant was correct. This was partly due to the fact that several discharge summaries had no author name or bleep number. By documenting your name and bleep number on a discharge summary, is not only accessible to physicians in primary and secondary care, but also to hospital pharmacist, in case they need to contact you when medication needs to be amended.

The second important problem identified in this study, was the lack of follow up appointments made, despite having it requested on the discharge summary. Of the patients that had follow up appointments suggested, only 40(67.8%) patients had appointments made, which meant that 19(32.2%) patients had no appointment made. The possible explanations for this may be that I started analyzing the patient summaries and follow up appointments too early post discharged, therefore not allowing enough time for the appointments to be made, or perhaps the appointments are simply not being made.

As this study did not look to see whether follow appointments with GPs had been made, we are unable to comment on this. However, previous studies have shown that follow up appointments are not always made with the GPs post discharge. As a result, the percentage of patients actually receiving a follow up post discharge from hospital may be lower than anticipated.

The third problem this study identified was the lack of discharge dates on the summaries. Having the discharge date on discharge summaries is not only important for hospital doctors but its of vital importance for GPs, as it provides them with information about how long a patient remained in hospital, and the severity of their illness. For example, if a patient was discharged from hospital after 2 days with an Asthma Exacerbation, we can assume that the severity of their exacerbation was not too severe. However, if the same pt remains in hospital for 15 days, this gives us more information about the severity of their exacerbation.

The fourth problem demonstrated in this study, was the infrequent number of diagnoses entered under the acute problems section in the discharge summaries. Only 65 (69.1%) summaries were identified to have a diagnoses, 22(23.4%) summaries had symptoms only. The remaining 13 (13.8%) summaries had no diagnosis or symptoms completed. It is important to document diagnosis or symptoms as it allows accurate medical coding. It is also often difficult to fathom why a patient was admitted to hospital even after reading the entire discharge summary. Furthermore, the benefit of accurate clinical coding is accurate payment for the services provided by the hospital.

The final problem which needs to be addressed is the completion of a discharge summary for all patients that have been admitted to hospital. This study found that 6 medical records showed no evidence of a discharge summary. One possible explanation for this would be if a patient had self discharged from the hospital, and not been formally discharged by a team or if a patient had died. However, in this case it is still important that a discharge summary is completed. A GP will still need to know why a patient was admitted and why they self discharged

CONCLUSION

It is clear from this study that there needs to be more robust processes put in place to ensure accurate recording of data on the information sent out to General Practitioners. Medical Practitioners completing the summaries should be encouraged to ensure that all fields on the discharge summary are adequately completed in order for us to reap the benefits.

Poor communication in the discharge summaries impacts poorly on patient care and increases the costs to the NHS due to increased rates of readmission into hospital. We can recognize this as a major problem confronting the NHS and so completing discharge summaries in full can help reduce his burden.

RECOMMENDATIONS

A number of recommendations have been identified and include:

- The need to raise the awareness of this problem amongst hospital colleagues including Clinical Governance and Audit department with the objective to improve the quality of the summary. The preferred format may be a presentation or advisory email.
- For consultants to communicate with junior doctors on a regular basis and go through their discharge summaries, highlighting areas of improvement. This is currently done by some medical teams at NUHT.
- For the author of the discharge summary to ensure the correct consultant name is on the discharge summary.
- All medical teams should complete a discharge summary regardless if a patient has self discharged or died.
- To relay this information back to the ward clerks and ensure that they understand the importance of making follow up appointments as soon as they have been given a discharge summary.
- A repeat audit should be performed in 12 months to look for improvements in the data completion.

AUTHOR DETAILS
SYEDA M. B. KAZMI, Foundation Year 2 Trainee, Barts and The London School of Medicine and Dentistry, Queen Mary University of London, London, United Kingdom
CORRESPONDENCE: DR S M B Kazmi, 15 Kevelioc Road, Tottenham, London, N17 7PR
Email: mar1amkazmi@hotmail.com

REFERENCES
1. Mann R and Williams J. Standards in medical record keeping. Clinical medicine. 2003 jul-Aug; 3 (4): 329-32
2. Paterson JM, Allega RL. Improving communication between hospital and community physicians. Feasibility study of a handwritten, faxed hospital discharge summary. Discharge Summary Study Group. Can Fam Physicians. 1999 Dec; 45: 2893-9
3. Wilson S, Ruscoe W, Chapman M, Miller R. General practitioner – hospital communications: a review of discharge summaries. Journal of Quality in Clinical Practice. 2001 Dec; 21(4): 104-8
4. Foster DS, Paterson C, Fairfield G. Evaluation of immediate discharge documents–room for improvement? Scott Med J. 2002 Aug; 47(4): 77-9
5. Garasen H and Johnsen R. The quality of communication about older patients between hospital physicians and general practitioners: a panel study assessment. BMC Health Services Research. 2007 Aug24; 7: 133

UPCOMING MEDICAL MEETINGS/CONFERENCES

Miscellaneous

2008 BRITISH FERTILITY SOCIETY (BFS) SUMMER COLLEGE
September 02-05, 2008   Obstetrics/Gynecology / Urology
Contact: BFS Secretariat Tel: 011-44-145-464-2217 Fax: 011-44-145-406-42222 Email: bfs@bioscientifica.com
 Website: www.britishfertilitysociety.org.uk
United Kingdom / Liverpool

2008 EUROPEAN HEADACHE & MIGRAINE TRUST INTERNATIONAL CONGRESS
September 04-07, 2008   - Neurology
Contact: Hampton Medical Conferences Ltd. Tel: 011-44-20-8979-8300 Fax: 011-44-20-8979-6700
Email: enquiries@ehmticongress2008.com
 Website: www.hamptonmedical.com
United Kingdom / London

2008 ANNUAL AND ACADEMIC MEETING OF BRITISH ASSOCIATION FOR PAEDIATRIC OTORHINOLARYNGOLOGY (BAPO)
September 12, 2008   - Otolaryngology / Pediatrics
Contact: BAPO Tel: 011-44-118-322-7137
Email: bapo@mac.com Website: www.bapo.org.uk
United Kingdom / Epsom

15TH PAEDIATRIC RHEUMATOLOGY EUROPEAN SOCIETY CONGRESS.
September 14-17, 2008- Pediatrics / Rheumatology
Contact: Hampton Medical Conferences Tel: 011-44-020-8979-8300 Fax: 011-44-020-8979-6700
Email: pres2008@hamptonmedical.com
 Website: www.pres2008.ukevents.org
United Kingdom / London

2008 SCIENTIFIC CONFERENCE OF THE BRITISH HYPERTENSION SOCIETY
September 15-17, 2008   - Cardiology
Contact: Gerry McCarthy, Hampton Medical Conferences Ltd
Tel: 011-44-208-979-8300 Fax: 011-44-208-979-6700
Email: gmccarthy@hamptonmedical.com
 Website: www.bhsoc.org/courses_conferences.htm
United Kingdom / Cambridge

2008 ANNUAL CONGRESS OF THE BRITISH ORTHOPAEDIC ASSOCIATION (BOA)
September 16-19, 2008   Orthopedics
Contact: BOA Tel: 011-44-207-405-6507 Fax: 011-44-207-831-2676 Email: N/A Website: www.boa.ac.uk
United Kingdom / Liverpool

JOINT CONFERENCE OF THE BRITISH THORACIC SOCIETY AND THE BRITISH INFECTION SOCIETY: INFECTIONS IN ACUTE MEDICINE.
September 16, 2008 General Medicine / Infectious Disease / Respirology
Contact: Royal College of Physicians of London Tel: 011-44-207-224-1539 Fax: 011-207-487-5218
Email: conferences@rcplondon.ac.uk
 Website: www.rcplondon.ac.uk
United Kingdom / London

2ND INTERNATIONAL SYMPOSIUM ON PHEOCHROMOCYTOMA.
September 17-20, 2008 Endocrinology / Neurology / Other

Specialties
Contact: Hampton Medical Conferences Ltd Tel: 011-44-208-979-8300 Fax: 011-44-208-979-6700
Email: isp2008@hamptonmedical.com
 Website: www.isp2008.ukevents.org
United Kingdom / Cambridge

ROYAL FREE HOSPITAL HANDS ON GYNAECOLOGICAL ENDOSCOPY SKILLS WORKSHOP
September 17-19, 2008 Obstetrics/Gynecology / Surgery
Contact: Therese Eleftheriou, Course Administrator Tel: 011-44-20-7794-0500 ext. 33863 Fax: 011-44-20-7431-1321
Email: courses@gynendo.com Website: www.gynendo.com
United Kingdom / London

2008 BRITISH ASSOCIATION OF PERINATAL MEDICINE (BAPM) ANNUAL GENERAL MEETING & FORUM ON CLINICAL GOVERNANCE IN PERINATAL CARE
September 17, 2008 Pediatrics
Contact: BAPM Office Tel: 011-44-20-7092-6085 Fax: 011-44-20-7092-6001 Website: www.bapm.org
United Kingdom / London

CHRONIC FATIGUE SYNDROME BRISTOL
September 18, 2008 Family Medicine / General Medicine / Psychiatry / Rheumatology
Contact: Joyce Achampong, Regional Events Co-ordinator
Tel: 011-44-20-7290-2980 Fax: 011-44-20-7290-2989
Email: joyce.achampong@rsm.ac.uk Website: www.rsm.ac.uk
United Kingdom / Bristol

MANAGEMENT OF CHRONIC KIDNEY DISEASE
September 22-25, 2008 Family Medicine / General Medicine / Nephrology
Contact: Dr Charlotte Moonan, University of Warwick Tel: 011-44-24-7652-3540 Fax: 011-44-24-7652-3701
Email: Charlotte.Moonan@warwick.ac.uk
 Website: www.britishrenal.org
United Kingdom / Coventry

50TH ANNIVERSARY ANNUAL SCIENTIFIC MEETING OF SCOTTISH SOCIETY OF PHYSICIANS
September 26-27, 2008 General Medicine
Contact: Hampton Medical Conferences Ltd. Tel: 011-44-20-8979-8300 Fax: 011-44-20-8979-6700
Email: ssp@hamptonmedical.com
 Website: www.hamptonmedical.com
United Kingdom / Glasgow

2ND INTERNATIONAL CONFERENCE OF THE SOCIETY FOR ACUTE MEDICINE
September 29-30, 2008 Emergency Medicine / General Medicine / Internal Medicine
Contact: Christina Lawson, Eventage Tel: 011-44-41-639-8123
Fax: 011-44-41-639-8123 Email: christine.lawson@eventage.co.uk
 Website: www.acutemedicine.org.uk
United Kingdom / London

CHILDBIRTH AND PELVIC FLOOR TRAUMA
October 02-03, 2008   Obstetrics/Gynecology
Contact: Conference Office, Royal College of Obstetricians & Gynaecologists Tel: 011-44-20-7772-6200 Fax: 011-44-20-7723-0575 Email: through website Website: www.rcog.org.uk

United Kingdom / London

REFRESHER DAY ON OBSTETRIC ANAESTHESIA AND ANALGESIA

October 08, 2008   Anesthesiology
Contact: Obstetric Anaesthetists' Association Secretariat Tel: 011-44-20-8741-1311 Fax: 011-44-20-8741-0611 Email: available through webpage Website: www.oaa-anaes.ac.uk
United Kingdom / London

A NEW ERA FOR STROKE PATIENTS.

October 14, 2008 Cardiology
Contact: Royal College of Physicians of London Tel: 011-44-207-224-1539 Fax: 011-207-487-5218
Email: conferences@rcplondon.ac.uk
 Website: www.rcplondon.ac.uk
United Kingdom / London

UPDATES IN INTERNAL MEDICINE

October 17, 2008 Internal Medicine
Contact: Mrs. Anne Fairbairn, Royal College of Physicians of Edinburgh Tel: 011-44-131-247-3649 Fax: 011-44-131-220-4393 Email: a.fairbairn@rcpe.ac.uk
 Website: www.rcpe.ac.uk/education/events
United Kingdom / Edinburgh

ACUTE AND GENERAL MEDICINE FOR THE PHYSICIAN.

October 27-29, 2008 General Medicine
Contact: Royal College of Physicians of London Tel: 011-44-207-224-1539 Fax: 011-207-487-5218
Email: conferences@rcplondon.ac.uk
 Website: www.rcplondon.ac.uk
United Kingdom / London

DIABETES & ENDOCRINOLOGY: SOMETHING FOR EVERYONE

October 29, 2008   Endocrinology / General Medicine / Geriatrics
Contact: Christine Berwick, Royal College of Physicians of Edinburgh Tel: 011-44-131-247-3634 Fax: 011-44-131-220-4393 Email: c.berwick@rcpe.ac.uk
 Website: www.rcpe.ac.uk/education/events
United Kingdom / Edinburgh

CARDIOVASCULAR MEDICINE

October 31, 2008 Cardiology
Contact: Ms. Eileen Strawn, Symposium Co-ordinator Tel: 011-44-131-225-7324 Fax: 011-44-131-220-4393
Email: e.strawn@rcpe.ac.uk
 Website: www.rcpe.ac.uk/education/events
United Kingdom / Edinburgh

36TH MEETING OF THE BRITISH SOCIETY FOR PAEDIATRIC ENDOCRINOLOGY & DIABETES

November 05-07, 2008 Endocrinology / Pediatrics
Contact: Shirine Borbor Tel: 011-44-1454-642-210 Fax: 011-44-1454-642-222 Email: conferences@endocrinology.org
 Website: www.bsped.org.uk
United Kingdom / Swansea

2ND EUROPEAN NEW YORK SCHOOL OF REGIONAL ANESTHESIA (NYSORA) SYMPOSIUM ON REGIONAL ANAESTHESIA & PAIN MEDICINE

November 07-09, 2008   Anesthesiology / Pain Management
Contact: Pat Pokorny, Course Secretariat, ChoiceLive Tel: 011-44-870-013-2930 Fax: 011-44-870-013-2940
Email: pat.pokorny@choicelive.com

 Website: events.choicegroup.co.uk
United Kingdom / London

THREE-DAY COURSE ON OBSTETRIC ANAESTHESIA AND ANALGESIA

November 10-12, 2008   Anesthesiology / Obstetrics/Gynecology
Contact: Meeting Secretariat Tel: 011-44-2-087-411-311
Fax: 011-44-2-087-410-611   Website: www.oaa-anaes.ac.uk
United Kingdom / London

MEDICAL COMPLICATIONS IN PREGNANCY

November 10-12, 2008 Anesthesiology / General Medicine / Obstetrics/Gynecology
Contact: Symposium Office, Imperial College London Tel: 011-44-20-7594-2150 Fax: 011-44-20-7594-2155
Email: sympreg@imperial.ac.uk
 Website: www.prossl.com/symposiassl/events.asp
United Kingdom / London

JOINT UK CONSENSUS CONFERENCE ON ACUTE MEDICINE

November 13-14, 2008   Emergency Medicine / General Medicine / Internal Medicine
Contact: Mrs. Margaret Farquhar, Consensus Conference Co-ordinator Tel: 011-44-131-247-3636 Fax: 011-44-131-220-4393
Email: m.farquhar@rcpe.ac.uk
 Website: www.rcpe.ac.uk/education/events
United Kingdom / Edinburgh

55TH ROYAL COLLEGE OF OBSTETRICIANS & GYNAECOLOGISTS STUDY GROUP FOLLOW UP: CANCER & REPRODUCTIVE HEALTH

November 19, 2008   Obstetrics/Gynecology / Oncology
Contact: Conference Office, Royal College of Obstetricians & Gynaecologists Tel: 011-44-20-7772-6200 Fax: 011-44-20-7772-6388 Website: www.rcog.org.uk
United Kingdom / London

2008 ANNUAL SCIENTIFIC MEETING OF ASSOCIATION OF LAPAROSCOPIC SURGEONS OF GREAT BRITAIN & IRELAND

November 20-21, 2008   Surgery
Contact: ASGBI Tel: 011-44-20-7973-0305
Email: jtreglohan@asgbi.org.uk
 Website: domain1686280.sites.fasthosts.com/uploads/Final%20Flyer.pdf
United Kingdom / Colchester

2008 NEONATAL UPDATE

November 24-28, 2008 Obstetrics/Gynecology
Contact: Symposium Office, Imperial College London Tel: 011-44-20-7594-2150 Fax: 011-44-20-7594-2155
Email: sympreg@imperial.ac.uk
 Website: www.prossl.com/symposiassl/events.asp
United Kingdom / London

NEUROLOGY: ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH SYMPOSIUM

November 27, 2008   Neurology
Contact: Mrs. Margaret Farquhar, Symposium Co-ordinator
Tel: 011-44-131-247-3636 Fax: 011-44-131-220-4393
Email: m.farquhar@rcpe.ac.uk
 Website: www.rcpe.ac.uk/education/events
United Kingdom / Edinburgh