Submission Supporting Public Hearing Testimony by A/Prof H Zoellner on the 23rd October 2012 To the Senate Standing Committee on Community Affairs Inquiry into the Dental Benefits Amendment Bill 2012

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One Page Executive Summary

General

- **The Medicare Chronic Disease Dental Scheme (MCDDS) was successful,** delivering comprehensive care to over 1.5 million mostly aged people with low incomes suffering significant untreated dental disease, although the MCDDS has now been closed
- **Planned child dental Medicare is inconsistent with Medicare principles and establishes dangerous precedent for Medicare to be limited:** to basic care only; defined age groups; and low income only. The competitive effect of bulk-billing could thus be lost, and National health costs would consequently rise
- **MCDDS patients will not be adequately supported by Government plans,** because Medicare will be directed to a different target population, and public dental services will not be able to adequately expand treatment capacity with planned increased funding
- **Evidence that planed increased funding of public dental services will not adequately care for past MCDDS patients is that: 1)** It was tried and failed under Keating; and **2**: Planned increased funding for public dental services is for only 30%, but varies already by up to 100% across state jurisdictions without any clear difference in public dental outcomes. *The most populous states NSW & VIC will spend less per patient after planed increased Federal funding, than other states already spend.*
- **Human rights implications cited in the Bill are internally inconsistent with closure of the MCDDS**, swapping the human rights of one group for another
- **Closure of the MCDDS leaves Australia without a structural basis for management of growing treatment needs in the ageing population**, while the ageing demographic becomes increasingly dependent on an inevitably overwhelmed public dental service

Suggestions for Improvement in the Government's Proposed Children's Scheme

- **Establishment of guidelines for diagnosis and treatment planning** to ensure funds are expended in a clinically justifiable and nationally uniform way
- **Removal of the limitation to 'Basic Dentistry' and $1,000 funding cap**, as most children have only simple service needs, and the few children requiring advanced treatment will not draw significantly on public funds but would be greatly disadvantaged by current Government plans relative to service under the MCDDS

Analysis of Government Reasons for Closing the MCDDS

- **Government claims the MCDDS is not targeted are incorrect**, because it is highly targeted to people with medical need of dental service
- **Government claims the MCDDS is used by millionaires are inconsistent with** the fact that 80% of MCDDS patients are health care card holders, while millionaires do not have the extensive dental disease evident in MCDDS patients
- **Government claims the MCDDS is extensively rorted are not supported by statistical evidence of: 1)** A complaint rate of only 1 complaint per 1,500 patients; **2)** Maximal spending per patient in the first year of about half the potential maximum; **3)** Reducing spending per patient to current levels of about half of average spending in the first year; **4)** Government information that only 65 dentists have been investigated and none prosecuted
- **Government claims the MCDDS has blown out expenditure, fail to recognize that** initial expenditure was experimental and required adjustment according to uptake
- **Government claims that MCDDS expenditure was wasteful, are inconsistent with** Government refusal to improve administration for cost containment and reducing cost per patient
- **Government claims that MCDDS expenditure was unsustainable ($900M yearly), fail to recognize** reducing cost as patients move from initial high treatment costs to low maintenance therapy costs, and also fail to include health savings across the health system. The projected maintenance cost for all MCDDS patients is $340M per year

1. Background on The Medicare Chronic Disease Dental Scheme (MCDDS) and Dental Benefits Amendment Bill 2012

1.a. MCDDS Eligibility was Based on Medical Need for Dental Treatment:

Between November 2007 and October 2012, people with life threatening chronic conditions including diabetes, congenital and valvular heart defects, head and neck cancer, bleeding disorders, immune compromise, organ transplants, dementia and mental health problems, were entitled to $2,125 of Medicare for private dentistry per year. Treatment was accessed through the Enhanced Primary Care scheme, in which a doctor wrote a referral to a dentist for service.

For further information, please find attached three brief papers outlining the known effects of dental disease on diabetes, as well as heart and vascular disease. Separate literature regarding other medical conditions can be made available to the Committee if required.

1.b. One and One Half Million People Were Treated Under the MCDDS

The MCDDS was successful, and delivered comprehensive dental care to over 1.5 million people during its 4 and a half year tenure, at a total cost of $2.7B.

1.c. Treatment was Typical of That Required by People Long Denied Access to Dental Care

Care delivered under the MCDDS was typical of that required by a population that had been denied access to dental services for a prolonged period of time, with most patients requiring an extraction, several fillings, a denture and in many instances root canal therapy and crown and bridgework.

Please see an appended publication from the Australian and New Zealand Journal of Public Health for detailed analysis of the type of treatment that was delivered under the scheme.

1.d. Most People Using the MCDDS Were Aged and had Low Incomes

The great majority of patients using the MCDDS are over the age of 55, while 80% of users were health care card holders, eligible for public dental treatment but unable to access care in the public system because of long waiting lists and waiting times. Further, the pattern of care received by these patients *(please see 1.c above)* was typical of that of people with long dental neglect, and did not reflect the needs of a wealthy population, who are known to have low dental needs.

1.e. Government Proposes Two Separate Alternative Plans to the MCDDS Comprising: (1) Means Tested Basic Only Dental Care for Children; and (2) Increased Public Dental Funding for Adult Treatments, related to a Dental Benefits Amendment Bill 2012

A Disallowance motion in the Lower House proposed by the Coalition to preserve the MCDDS was unsuccessful, so that the MCDDS is being discontinued.

In replacement for the MCDDS, Government proposes through the Dental Benefits Amendment Act, to expand Teen Dental to deliver basic dentistry only, to children on a means-tested basis, via Medicare. Rebates would be limited to $1,000 per year, and would be for basic dentistry only. Expenditure of $2.7B is planned over 6 years to commence in 2014.

Because this does not address adult chronic disease needs, and also represents reduced support for children with chronic disease, Government additionally proposes to increase funding to state run public dental services. This represents $1.3B over 6 years to commence in 2014. Patients with chronic disease would only access this funding if eligible for public dental service, and would also only receive treatment via the public dental services.

1.f. The Purpose of this Document

APOH's Chairman (A/Prof H Zoellner), has been asked to present as a witness to a Public Hearing of the Senate Standing Committee on Community Affairs for discussion of the Dental Benefits Amendment Bill 2012.

Some difficulties with Government's proposed plan for children are identified by APOH, and discussed in this document.

Also, some suggestions for improvement in the Government's planned children's program are made.

Additionally, while the Government cites numerous reasons why the MCDDS was closed, the view of APOH is that these reasons were ill considered, and not consistent with the available evidence. For the purposes of clarification, this document also summarizes ways in which the Government seems misled in it's understanding of the MCDDS.

A particular concern, is that the greatest burden of dental disease is in the ageing population. The MCDDS did provide a clear trial for funding comprehensive dental care in the aging population. Discontinuation of the MCDDS undermines the opportunity to prepare Australia's health funding system for the oncoming bulge in demand for dental services by the aging population.

APOH must advise that closure of the MCDDS leaves Australia vulnerable to a medium and long-term dental crisis, where a very large proportion of the population will make increasingly impossible demands on public dental services.

2. Difficulties with the Government's Proposed Plan for Expanded Teen Dental Services

Although APOH strongly supports the principle of dental services being included in Medicare, and is broadly supportive of this Bill's intention to include child dentistry in Medicare, there are a number of points which Senate may wish to consider.

2.a. The Proposed Scheme Breaks Three Fundamental Principles of Medicare, Establishing Dangerous Precedent That Undermines the Future of Medicare

As detailed below, three fundamental principles for Medicare are broken in the Government's proposed policy.

In this way, the currently proposed legislation establishes dangerous precedent from which future governments may derive inspiration to undermine the value and effectiveness of` Medicare as a core component of the Australian Health system.

2.a.i) - Means Testing of Dental Medicare for Children Undermines Funding of Medicare and Reduces Competitive Market Forces

As a universal health insurance scheme, Medicare provides equivalent support to all citizens regardless of income.

In this way, wealthy individuals provide heavy subsidy for the health services of less well off citizens, because wealthy people pay larger sums into Medicare through their Medicare levies and taxation. Since the oral health of wealthy people is generally good, no only do the wealthy 'bank-roll' the system, but they are reasonably expected to make little claim on dental Medicare services.

In addition, Medicare bulk-billing practices generate intense downward pressure on professional fees, against which gap-charging private medical and dental practices must compete. Universality of Medicare cover ensures that the entire available market can access bulk-billing practices.

Were Medicare limited on a means-tested basis, then an insufficient proportion of the medical and dental markets would have access to the scheme, for enough bulk-billing practices to exert downward force on medical and dental private fees.

The Government's plan, however, is for dental Medicare for children to be limited on a means-tested basis, so that no downward pressure on the price of private dental service can be expected. Also, were this approach expanded to the wider Medicare system by future governments, the competitive pressure exerted by Medicare in Medicine would be lost, and the overall National cost of medical services would rise substantially.

2.a.ii) - Limitation of Dental Medicare to Only Young People Ignores the Bulk of Community Dental Need, and Undermines Long Term Dental Health of Children Treated Under the Scheme

Universality of Medicare by definition means that service is available to all individuals, regardless of age. Limiting the proposed dental Medicare program to children only, clearly breaks this fundamental quality of the wider Medicare system.

Because the bulk of community dental disease burden resides in the older population and children have generally low dental service needs, it is difficult to understand why limited Government resources are directed away from people with greatest need and towards the population with the lowest need for dental treatment.

Of particular concern in dentistry, is that young adults, becoming independent of their parents and commencing adult independent life, have essentially equivalent dental needs to older teenagers. One aspect of the teen-age population, is an increase in the rate at which decay develops, so that sudden withdrawal of dental services from young people once they reach the age of 18, will result in a corresponding deterioration in dental health in young adults.

There seems no clear reason why the dental care of any individual should be determined on the basis of age, and even less reason why access to dental services should be withdrawn from young people emerging into adulthood.

There is the further practical impact of sending a signal to young people, that once the 'teenage years' are passed, that oral health is assured, whereas infact life-long care is needed, especially as people age and accumulate chronic disease, and medication use that causes dry mouth, immune compromise and worse dental infection.

2.a.iii) - Limitation of Dental Medicare for Children to Basic Service Only Undermines Oral Health and is Inconsistent with the Wider Health Service

The high quality of health care in Australia, is in part because Medicare provides comprehensive medical care, as opposed to only basic medical care. There seems no clear reason why when including management of oral disease under Medicare, service should be restricted to basic service only.

2.b. Opening of a Limited Child Medicare Program in Replacement for Closure of the Medicare Chronic Disease Dental Scheme Does Not Seem Logical

Government appears to argue, that the proposed Medicare program for children is in substitution for closure of the Medicare Chronic Disease Dental Scheme. This does not seem logical for the following reasons.

2.b.i) - The Two Schemes Service Two Separate Populations of People

The great majority of people who have been eligible for the Medicare Chronic Disease Dental Scheme are aged and with chronic disease, while the children's program is by definition for younger people, most of whom are otherwise healthy.

2.b.ii) Children with Chronic Disease will have Reduced Support

Children who do suffer with chronic disease, and who have enjoyed the benefit of the Medicare Chronic Disease Dental Scheme, will have reduced levels of care under the proposed new scheme. This is because the proposed new scheme is limited to basic dentistry only and only, providing $1,000 maximum rebate over two years, compared with comprehensive care and maximum possible expenditure of $4,250 per two year period under the Medicare Chronic Disease Dental Scheme.

2.c. There is Internal Inconsistency with Human Rights Implications in the Proposed Bill and Closure of the Medicare Chronic Disease Dental Scheme

Page 2 of the bill makes note that the bill engages the right to health and right to social security, Article 12(1) of the International Covenant on Economic, Social and Cultural Rights, and Article 9 of the ICESCR on the right to social security including social insurance.

Exclusion of people on the basis of age and income, as well as closure of the Medicare Chronic Disease Dental Scheme, seem in opposition to the spirit of the above cited Articles.

3. Suggestions for Improvement of the Government's Proposed Program for Children's Dental Medicare

3.1. A Need to Establish Clear Clinical Guidelines for Dental Diagnosis and Treatment Planning

A frequent and reasonable criticism of the MCDDS, has been that there may have been over-servicing for expensive crown and bridgework. Although there is no clear publicly available evidence confirming this, other than general statistical arguments (detailed in the attached manuscript published in the Australian and New Zealand Journal of Public Health), one lesson of the MCDDS is that a consistent standard for diagnosis and treatment planning is desirable for any Nationally funded dental scheme.

To that end, APOH proposes that clear National standards for diagnosis and treatment planning for dental disease must be established and administered by Medicare in the Government's newly proposed scheme.

This would ensure that all Medicare supported dental services are in accordance with current best practice, and also that any funds expended are appropriately clinically justified.

3.2. Limiting Child Dental Medicare Funding to Basic Dentistry Only and $1,000 Per Two year Period Will Greatly Disadvantage Some Children For Only Negligible Medicare Savings, and Further Distances the Proposed Scheme from the Rest of Medicare

As outlined above in 2.a.iii and 2.b.ii, the proposed new scheme for children is to limit available funding to $1,000 per two year period, and for basic dentistry only.

For the great majority of children, the average maximum anticipated expenditure of $500 per year will not be needed, because very few children have extensive need for dental procedures. A 'basic dentistry only' approach will also be acceptable for most children, because very few children have need for advanced dental services.

The proposed limitation on scope of practice and funding does, however, profoundly disadvantage the occasional child who has high dental service needs, especially when such children suffer chronic disease.

APOH argues that the very small number of children needing extensive dental treatment should have access to any funding required without limitation on the complexity of work or funding, as they constitute only a small proportion of the child population and will not draw significantly upon Medicare funds, but will be materially disadvantaged by the Government's policy.

As such, APOH argues that removal of the 'basic dentistry' and '$1,000 per two year cap' will have negligible effect on expenditure, especially if clinical guidelines for diagnosis and treatment planning are instituted (3.1). Removal of the funding cap would bring the proposed new scheme more closely into alignment with arrangements in the rest of Medicare, which is an uncapped scheme.

4. Numerical and Other Evidence That Reasons Cited by Government for Closing the MCDDS are Misguided

4.a. Response to the Government Argument That 'The MCDDS was Not Targeted'

The MCDDS was by definition targeted to patients with medical need of dental treatment.

A related Government argument has been that the MCDDS was not means tested. However, since the MCDDS was within Medicare, and Medicare is not means tested, any means testing of the MCDDS would be out of keeping with the broader Medicare system.

Establishing means testing in Medicare sets a dangerous precedent, because future governments may expand this principle to include all of Medicare. Were this to occur, Medicare could not be funded as is currently the case, because Medicare levy payments and taxes from relatively few wealthy people currently carry the cost of service delivery to large numbers of people with low incomes.

4b. Response to Government Argument that: 'The MCDDS was Used by Millionaires, While Children and Low Income People were Unable to Access Care'

80% of MCDDS patients were health care card holders, and thus by definition on low incomes.

The remaining 20% of patients were mostly drawn from the ranks of the 'working poor'. This seems a reasonable conclusion from the extensive dental service required by the population using the MCDDS, as separate studies have long shown that the bulk of dental disease is amongst low income earners. Also, the pattern of treatment delivered is consistent with low income people who have been previously unable to access care *(please see attached publication from the Australian and New Zealand Journal of Public Health)*.

It is impossible to imagine, that millionaires have waited for the MCDDS to deliver treatment for pain, extractions, new dentures, fillings and advanced restorative work. Millionaires have in general good dental health, and if it is the case that some millionaires have accessed the MCDDS, it is almost certain that they will have made little call upon the scheme, as they will have had little need.

Children have been eligible for MCDDS service, and have used the scheme in appreciable numbers, so that Government is simply incorrect when claiming that children have not had access.

Regarding the fact that low income earners are excluded from the MCDDS unless suffering chronic disease, it has always been possible for the Government to extend eligibility for the MCDDS on a means tested basis, and this has been the specific

recommendation from APOH.

Closing the MCDDS seems an irrational approach to expanding eligibility for access to MCDDS services to the poor.

4.c. Response to the Government Argument that 'The MCDDS was Extensively Rorted'

While the Government has made repeated announcement of widespread rorting of the MCDDS, only 65 dentists have been investigated and no dentists have been prosecuted. The Minister acknowledges that most cases involve administrative error and not fraudulent use of public funds.

Further, only 1,000 complaints have been made over 2,000,000 courses of care for 1,500,000 patients - **A Complaint Rate of 1 Per 1,5000 Patients**, which is substantially less than would be expected had there been widespread abuse.

In addition, and as noted elsewhere in this document, spending in the MCDDS has never reached maximum potential ($4,250 per patient in the first year of operation), but was maximal at only $2,202 per patient in the first year. Had there been widespread rorting, then expenditure would have approached $4,250 instead.

Also as noted elsewhere in this document, spending in the MCDDS reduced as patients moved from initial high cost treatment to low cost maintenance therapy, so that in the last year, average cost of treatment has been $1,171 per patient. Were the system widely rorted, then expenditure would have been maximal at $2,125 and would not have dropped.

It is difficult to understand why Government has publicly harassed clinicians for administrative errors, in large part resulting from Government refusal to simplify MCDDS administration.

As such, any objective evaluation of the available data, indicates that any rorting that may have occurred must have been limited to a handful of clinicians, and Government claims of widespread rorting are numerically insupportable.

4.d. Response to the Government Argument that 'Past MCDDS Patients will be Cared For in Future by Increased Funding for Public Dental Services'

Proposed increased Federal funding of public dental services represents a 33% increase in funding across the Nation.

Although sounding significant, this will not make significant difference to public dental patients because the public dental system is unable to attract additional workforce regardless of funding invested. Private practice is by comparison much more lucrative relative to public dental service, and also provides opportunity for more varied work because the public service is focused on basic and emergency style care.

Two lines of evidence support this conclusion.

Firstly, the Keating Government tried this approach, and was unable to establish lasting change.

Secondly, as seen in Table 1 and Figure 1 below, there is highly variable public dental funding across Australia, such that Tasmania spends twice as much per citizen on public dentistry than NSW. Nonetheless, despite much more money being available for public dentistry in all states and territories compared with NSW, public patients are no better off and public waiting lists, waiting times, and quality of care are comparable across all states and territories.

After the increased funding is made available, NSW and ACT will still be spending at levels lower compared with current expenditure in NT, TAS, QLD, and SA, while VIC will only just have reached current spending levels in QLD.

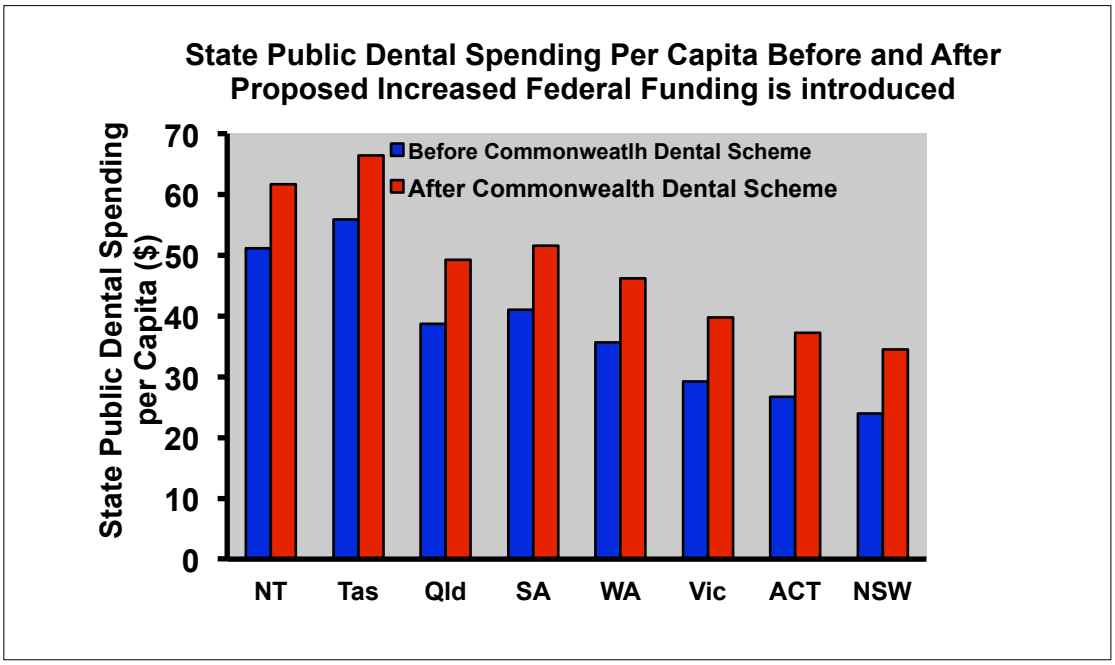
The absence of any discernable betterment for public dental patients in states with greater spending levels demonstrates that increased public dental spending will have little effect.

Also, please note that the public dental service attempts to treat up to 50% of the population eligible for public dentistry, but with only 10% of the workforce, is chronically unable to cope and trapped in emergency care.

It seems worth noting, that dental caries and periodontitis are the two main dental diseases managed in both private and public dental practice, but that the public dental services only rarely deliver the intense preventive service required by periodontitis patients, and instead systematically resort to treatment by extraction only and denture manufacture.

Notably, the MCDDS has had the effect of reducing public dental waiting lists in NSW by about one third, because public patients could move from the waiting list into private practice funded by Medicare. Closure of the MCDDS will simply push these patients back into the public system, which will again be unable to cope and result in longer waiting lists.

State	Current Spending ($)	Current Per Capita Spending ($)	Additional $ Per Year	Final Proposed $ Per Year	% Increase	Proposed Per Capita Spending ($)
NT	10,580,000	51.19	2,173,734	12,753,734	20.55	61.70
Tas	27,323,000	55.89	5,141,453	32,464,453	18.82	66.40
Qld	156,950,000	38.72	42,627,054	199,577,054	27.16	49.24
SA	63,816,622	41.05	16,349,800	80,166,422	25.62	51.56
WA	73,125,000	35.66	21,568,023	94,693,023	29.49	46.17
Vic	148,900,000	29.24	53,546,201	202,446,201	35.96	39.76
ACT	8,773,000	26.68	3,457,782	12,230,782	39.41	37.20
NSW	163,500,000	23.95	71,802,619	235,302,619	43.92	34.46
Total	652967622	37.80	216,666,667	869,634,289	33.18	48.31



4.e. Response to the Government Argument That 'The MCDDS Expenditure Has Blown Out'

It seems important to put the MCDDS in historical perspective. When first established by then Health Minister Abbott, in the dying days of the Howard Government, APOH was publicly critical that: a) the scheme was excessively bureaucratic requiring the preparation of complex care plans and letters of referral and reporting; b) that medical practitioners were acting as gate-keepers but in general were poorly informed on dental disease; and c) that it was limited to people with chronic disease and not available to the wider population with often similar needs.

After voicing these objections in the media, APOH's chairman (H Zoellner) received a phone call from Mr Abbott's office, explaining that the long term intention was to open the scheme up to be universal and without these bureaucratic limitations, but that in the first instance, a trial of dentistry in Medicare was needed. This was because it was not possible to properly anticipate: demand, costs, the correctness

or otherwise of the schedule, possible rorts, and any other unforeseeable difficulties. It was explained that the enhanced primary care scheme provided a convenient administrative box within which to trial dental Medicare and identify problems. Approaches to dealing with problems could then be themselves trialed within the MCDDS. Once the MCDDS could be seen to be working properly, then a sensible roll-out, costed on the basis of MCDDS experience, could be planned for the rest of the community.

It was also clear that the emphasis on 'chronic disease' was not of great importance to the previous government, other than that it did provide some clinical justification at targeting earliest dental Medicare access to people with medical need of dental service. Instead, 'chronic disease' was a convenient Enhanced Primary Care Scheme limitation, ensuring that any possible difficulties in the MCDDS would be limited to a smaller community rather than spread across the entire Nation.

Although this seemed a sensible approach, and was subsequently supported by APOH once it was explained, it is also clear that at its inception, it was not possible to properly anticipate actual costs, and that the previous Government did expect to have to adjust planned expenditure as the scheme took hold.

For these reasons, it is irrelevant to refer to the successful use of the scheme as having resulted in a 'cost blow-out'. The initial planned expenditure was experimental, and had the previous Government been retained, budget expenditure would have been adjusted in line with community need and improved regulation.

It is deeply unfortunate that the Government has not used the opportunity of the MCDDS as a trial to address difficulties in dental Medicare, but has instead simply sought to close the trial.

4.f. Response to Government Argument That: 'The MCDDS was Wasteful of Public Funds'

While dental service is surgical in nature and thus inherently expensive to deliver, it is also the case that costs can be contained and better clinical outcomes achieved through application of appropriate administrative procedures.

The Government has long had advice from APOH and others, to implement improved administrative procedures, with the intention of both containing costs and ensuring that all clinical procedures supported by the MCDDS are in accordance with current clinical best practice.

Specific changes recommended have been for: 1) Simplification of referral and reporting processes, to reduce the cost of entry to the MCDDS as well as of ongoing patient management; 2) Establishment of clear clinical guidelines for diagnosis and treatment planning, comparable to those established in Medicine and which if applied to the MCDDS would ensure that expensive services are only delivered where clinically appropriate; 3) Implementation of a pre-approval process for crowns, bridges and implants similar to that long established in the Department

of Verteran's Affairs, so as to ensure that when these clinical services are delivered in the MCDDS, that this is only in accordance with the clinical guidelines for treatment planning as suggested above.

APOH estimates that had these simple measures been implemented, savings in the order of 30 % of past expenditure would have been achieved, together with improved clinical outcomes.

Unfortunately, Government has not made any changes to MCDDS administration over the last 4.5 years, and thus must itself accept responsibility for an estimated $900M unnecessary expenditure during that time.

Separately, MCDDS expenditure must be seen in light of downstream health and cost benefits to 1 million diabetics for whom there is clear evidence linking dental infection with worse diabetic disease, as well as for another 1 million further citizens suffering across a wide range of further chronic diseases.

It is difficult to understand why the Government insists that it's own maladministration of the MCDDS constitutes justification for closure.

4.g. Response to the Government Argument That: 'The MCDDS Expenditure of $900M per Year was Unsustainable'.

While the most recent yearly expenditure in the MCDDS was approximately $900M, for the following reasons, it is incorrect to suggest that this cohort of patients will draw indefinitely upon Government revenue at that rate. Instead, long term expenditure for this scheme would have been very much lower at $340M per year. The reasons for this are outlined below.

Firstly, patients entering the scheme had extensive dental disease and thus needed extensive and often expensive work performed. The average expenditure per patient when the MCDDS commenced was $2,202, reflecting the needs of a population long denied access to dental services.

However, once initial treatment is delivered, patients move onto low-cost maintenance therapy, so that despite only 30% of recent patients having already had an initial course of care, the average cost per patient has dropped to just $1,171, about half of initial expenditure. This outcome of reducing cost per patient was predicted at the commencement of the MCDDS, while the cost per patient would have continued to drop had the scheme continued.

In addition, one third of expenditure in the MCDDS was for new dentures. However, new dentures could only be issued once every 7 years, so that on this basis alone, expenditure would be expected to drop by one third over the next two years as all dentures would have been made for eligible patients.

Finally, taking into account the above statistics and projecting forward, the anticipated MCDDS cost should have been $340M per year. This to sustain the oral

health of all eligible patients in maintenance therapy.

Considering savings to be achieved through better systemic health outcomes, with reduced hospitalizations, improved diabetic outcomes, and reduced PBS antibiotic and analgesic costs, the $340M yearly MCDDS investment would likely result in savings greater than actual expenditure.

4.h. Response to the Government Argument That: 'More People (Children) Will Be Treated Under the Government's Proposed Scheme Than Are Treated Under the MCDDS'

Parliamentary Members with a medical or military background will be familiar with the concept of 'triage', used to determine priority of treatment where clinical resources are limited. By this widely used approach, patients with greatest need and potential benefit are assigned a higher priority over those with minimal need.

Child dental health is very good in Australia, so that treatment needs for this population are low. The dental treatment needs of those who access the MCDDS are, however, very high. Similarly, the health impact of treatment in the MCDDS is very high. By way of example, please see several de-identified emails received from MCDDS patients attached. These demonstrate obvious significant clinical need in this group.

On this basis, when applying 'triage principles' to determine where limited funding would have the greatest impact, an estimated 2 million MCDDS eligible patients should be prioritized over the 3.2 million estimated eligible children with generally good dental health.

Please note, that this document does not argue against child Dental Medicare, and is in support the Government's initiative to provide dental care to children via Medicare.

This document argues that both children, and people with chronic disease, should receive care, but that if resources are too limited to support both groups, that the largest clinical need and benefit is amongst the chronically ill.

4.i. Response to the Government Assumption that 'Federal Funding of Oral Disease Should be Fundamentally Separate to That for Disease of the Rest of the Body'

It is an historical anachronism, that Medicare includes comprehensive care of all parts of the human body, except the oral cavity. There is no clear medical reason why this should be the case, as disease of the oral cavity has comparable effect on the health and wellbeing of humans to that of most other parts of the body.

Infact, the neurological, psychological and emotional value of the oral cavity is such, that there is disproportionate suffering for disease of the mouth compared with most other body parts.

While Government intention to include children's dentistry under Medicare is laudable, it is not clear why this should be on a means-tested basis whereas the remainder of Medicare is not means tested.

Similarly, the logic of restricting access to dental care on the basis of age seems spurious, since dental disease affects people of all ages. The case often argued that 'if children are treated then they will have good teeth as adults', is not well supported by the facts, since it is common for young adults leaving home to neglect dental health due to limited income, irrespective of earlier childhood care.

Also, it is unclear why dental Medicare for children should be limited to basic service only, whereas the remainder of Medicare is for comprehensive care.

The MCDDS was more closely adapted to the wider structure of Medicare than the Government's current plan for children's dentistry, because the MCDDS was not restricted with regard to age, income or limited to basic care.

4.j. A Note on Closure of the MCDDS and Lost Opportunity to Prepare Australia for Oncoming Medium and Long Term High Service Needs in the Aging Population

The ageing demographic and distribution of dental disease is such, that within the next 10 years, Australia will face 'a tsunami' of unfunded dental disease. As little as 40 years ago, most people over the age of 60 had full dentures, but this is no longer the case, with most aged people having teeth that require ongoing maintenance therapy. With the baby boomer population increasingly moving into retirement, demand on the public dental services will soon increase well beyond the current excessive levels.

The MCDDS provided opportunity for Australia to establish funding mechanisms capable of managing the oncoming bulge in demand for dental services. Dissolution of the MCDDS, however, and replacement with a limited child dental service and some public dental spending, does not seem to better prepare Australia for this oncoming difficulty, but instead seems a clear step backward.

It is APOH's duty to alert the Senate that Australia is rapidly running out of time to establish mechanisms similar to the MCDDS, capable of delivering comprehensive dental care to the ageing population.

4.k. A Note on Highly Diverse Uptake of the MCDDS Across State and Territory Jurisdictions: Disadvantage of Citizens According to Jurisdiction

It is APOH's further responsibility, to alert Senators representing constituents where uptake of the MCDDS has been low, that this is the result of Government refusal to advertise eligibility.

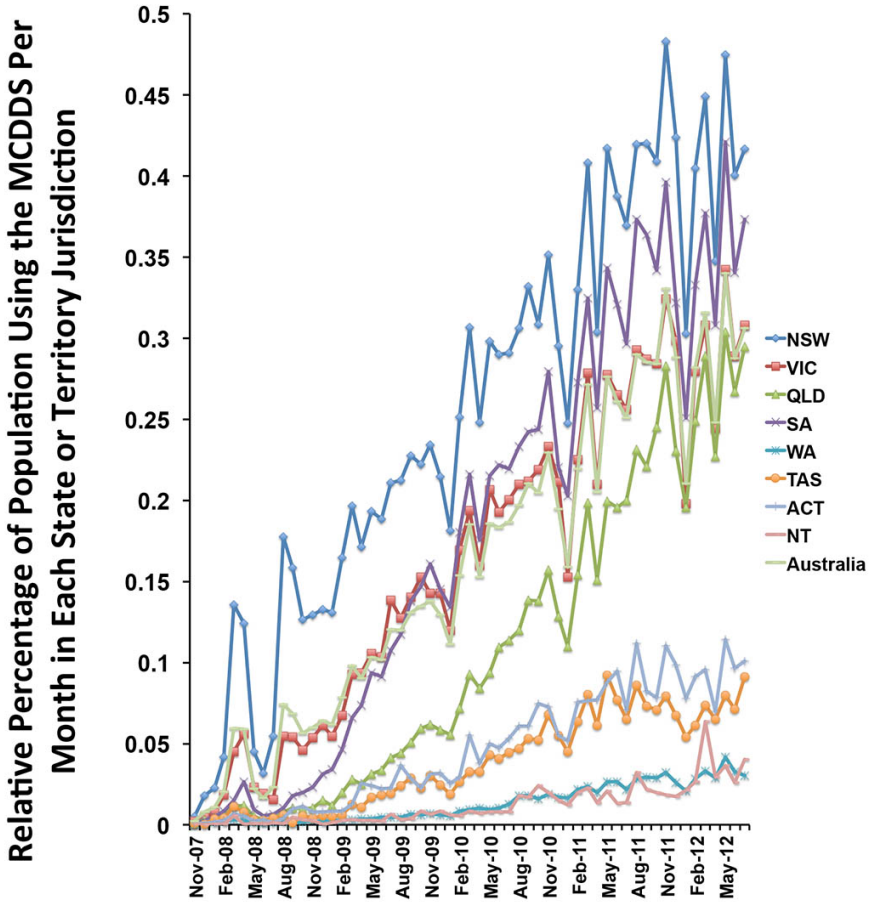
Because Government did not advertised existence of the MCDDS to eligible

patients, and the only advertising has been unofficial via health contacts of APOH and NSW Health, there has been highly variable use of the MCDDS across Australia.

The graph below shows the relative percentage of population in each state and territory jurisdiction that has used the MCDDS in each month since the scheme was established. While NSW had rapid uptake of the scheme, other states and territories had only much later use, and WA, TAS, NT and ACT have had almost no uptake of the scheme.

A diabetic person in WA for example, has clearly been disadvantaged relative to a diabetic person in NSW, and worse health outcomes in that state can be expected. The role of Senators in protecting the relative interests of their Sate constituents seems challenged by these statistics.

Graph showing the relative percentage of population using the MCDDS across state and territory jurisdictions. Low uptake in WA, TAS, ACT and NT reflect failed advertising of the scheme to eligible patients.



Appended Documents:

- Zoellner H. Oral problems in patients with diabetes. Diabetes Management Journal. 2006. 14: 18-19. *(An invited review outlining the main relationships between dental infection and diabetes)*

- Zoellner H. Gingivitis and the heart. Cardiology in General Practice. 2006 3: 28. *(An invited review outlining the main relationships between gingival and periodontal disease and heart disease)*

- Zoellner H. Dental infection and vascular disease. Seminars in Thrombosis and Haemostasis. 2011 37: 181-192. *(An invited review of the literature relating vascular disease to dental infection, which was subjected to peer review prior to publication)*

- Palfreeman V and Zoellner H. Description of comprehensive dental services supported by the Medicare Chronic Disease Dental Scheme in the first 23 months of operation. Australian and New Zealand Journal of Public Health. 2012 36: 69-75. *(A peer reviewed research publication in which the pattern of service delivery under the MCDDS was analyzed and described in detail)*

Oral Problems in Patients with Diabetes

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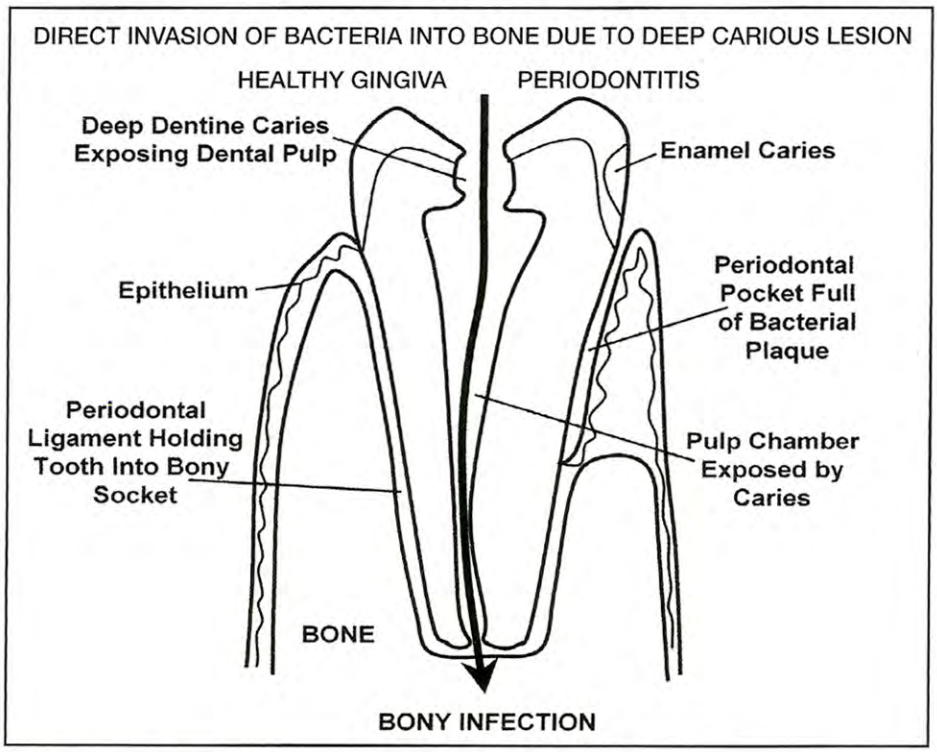


The Personal Impact of Oral Disease

As a major organ for eating, speech, facial expression and sexual intimacy, the mouth plays an important role in life. Oral pain is disproportionately intense, not only because of the large sensory cortical area devoted to the mouth, but also because oral deformity causes great mental anguish. In our society, to have a row of blackened, pus draining, missing and frankly stinking teeth embarrasses a person, undermines their self-esteem, and even reinforces unemployment.

Cruel Assumptions

Some false and cruel assumptions are:

- that those suffering have only themselves to blame;
- caries is not a life-threatening disease;
- oral health has little impact on general health;
- dental services are essentially cosmetic.

These assumptions must be challenged, because they support an anachronistic exclusion of oral health from the wider health system. Nonetheless, caries is the most common infectious disease in man. With the ageing population, demand for dental services is to increase by 20% and 29% in the private and public sectors respectively by 2010, while training positions for dentists have been reduced, instead of increased, over the last 25 years.[1]

Bad News for People with Diabetes

This is bad news for patients who have diabetes, for whom infections are generally more severe. The problems include dental abscesses, periodontitis and oral candidosis. Worse still, there is a positive feedback loop between diabetes and oral infection, with periodontitis seeming to increase the severity of diabetes and contributing to poorer glycaemic control.[2-5] Also, correlation between poor control of diabetes and caries is reported, although this is still controversial.[3-5] There are many anecdotal accounts of life-threatening dental infections in patients with diabetes. Potentially fatal Ludwig's angina from dental infection is fairly frequently seen in most major hospitals, while there are about 32,000 preventable hospitalisations for dental treatment in Australia per year.[1]

Definitive Treatment of Dental Infection is Surgical

A common history of patients with life-threatening dental infection is that if they are unable to access or afford dental treatment, they seek antibiotics from their medical practitioner. After one or two courses of antibiotics, however, a spreading uncontrolled infection necessitates hospitalisation to manage both sepsis and airway. Unfortunately, the definitive treatment of dental infection is almost invariably surgical, so antibiotic therapy alone is both ineffective and potentially dangerous.[6,7] The reasons for this become clear when the pathogenesis of dental infection is examined.

The Common Dental Infections – Caries, Periodontitis and Increased Susceptibility in Diabetes

For caries to develop, four separate factors must each be satisfied:

1. bacterial plaque must accumulate;
2. fermentable carbohydrate must be present for acid production by the bacteria;
3. the tooth surface must be susceptible to caries;
4. there must be sufficient time for the lesion to develop.[6,7]

Saliva dilutes fermentable carbohydrates, has buffers and ions that neutralise acid and re-mineralise enamel and saliva also has anti-bacterial agents. The differences

between individuals regarding saliva, dental morphology and fluoride experience generate variable susceptibility to caries. Patients who have diabetes may have an increased risk of caries due to the reduced salivary flow from diabetes and medications used to treat concomitant disease. Additionally, the diet contributing to the Type 2 diabetes may also drive dental caries.[8] These factors, together with the life-long nature of diabetes, may increase the risk of caries that exists in many patients with diabetes.[5, 8]

The early enamel lesion is essentially porous, and acid creates a delicate honeycomb of enamel. The nature of caries changes dramatically once acid dissolution reaches the underlying collagenous dentine. The softened dentine flexes, so that the overlying enamel breaks down and bacteria enter the actual body of the tooth for the first time. Instead of relying upon the human diet for nutrition, these organisms now derive their nutrition through degradation and destruction of the dentine matrix. Colonies of bacteria invade and destroy the dentine to eventually infect the vascular dental pulp. Because the dental pulp connects to the underlying bone through a small opening at the end of the root, bacteria eventually spread into the underlying bone to cause chronic infection. At this point, bacteria, saliva and even food debris have direct access to the bone.

Destruction of bone and replacement by masses of inflammatory granulation tissue occurs. Dental cysts may also form through proliferation of embryonic epithelium and these common abnormalities can cause significant deformity and weaken the jaws. The destructive bony lesions are usually surprisingly painless, but often present as acute dental abscesses. Spread of pus and infection into the floor of the mouth or into the para and retro-pharyngeal spaces may be life threatening. Spread to the maxillary sinuses from dental infection is also common, and may present as acute or chronic sinusitis.[6, 7, 9]

Periodontitis is also caused by bacterial plaque, with bacteria driving a destructive inflammatory response in the adjacent gingival tissues, forming deep "pockets" between the tooth and the remaining soft tissues. Stripping of the tooth's anchorage results in loosening and eventual loss.[6, 7] It is interesting that the widespread perivascular hyaline deposits of diabetes also occur in periodontitis, and it has been proposed that this contributes to development of the disease.[10] This, together with reduced neutrophil function, may be the basis for the increased periodontitis noted in patients with diabetes.[6, 10] On occasion, abscesses may form from periodontal pockets.[6, 7]

It is clear that definitive treatment of dental infection involves removal of the source of infection, which usually means removing either the tooth or the infected dental pulp tissue by root canal therapy. Treatment with antibiotics alone only encourages the development of resistant organisms, which are often aggressive gram negative anaerobes.[6, 7]

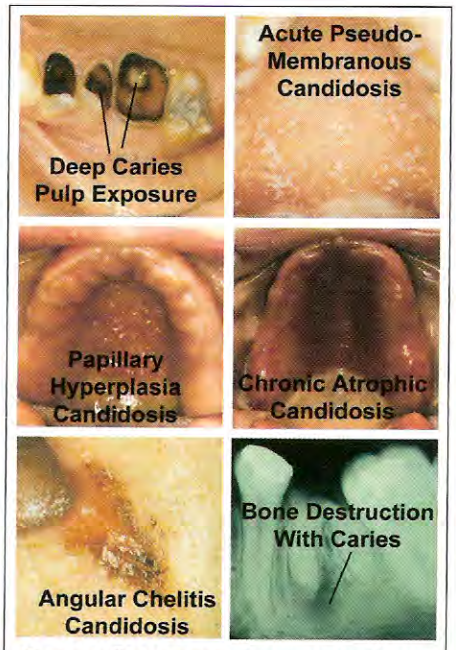
Oral Candidosis, Lichenoid Reactions and Sialosis are Increased in Diabetes

The traditional thrush lesion, with multiple white curd-like colonies of candida, is readily recognised in the immuno-compromised patient. Nonetheless, oral candidosis more often presents as erythematous lesions of the mucosa, and is often associated with denture wearing. Depapillation of the tongue (with or without pain), or angular chelitis, presenting as crusting sore lesions at the corners of the lips, are further presentations of oral candidosis. Significantly, all of these forms of oral candidosis are common in patients with diabetes and require different strategies for management, dependent upon the location of lesions and the presence or absence of dentures. Lichenoid reactions to some oral hypoglycaemic drugs may occur and usually present as white lace-like lesions on the buccal mucosa. Swelling of the parotid glands is another oral manifestation of diabetes that is seen in some patients.[6]

Prevention, Access to Dental Care and Medicare

It seems fair to suggest that regular preventive dental treatment is an important but neglected part of the management of diabetes. Similarly, because some form of physical intervention is usually required to manage dental infection, it is most important to ensure that patients with acute dental infection are seen by appropriately qualified dental practitioners and not simply treated with antibiotics.

There seems no moral justification for Medicare to exclude dental preventive treatment, caries control, pain control and the management of dental infection. At the very least, those who are most at risk, such as people with diabetes, should receive the necessary support. It is time for our health officials to deal with this growing problem.



Some Common Presentations of Caries and Oral Candidosis

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Gingivitis and the Heart

CHRONIC GINGIVITIS IS A PRECURSOR TO DESTRUCTIVE PERIODONTITIS

Over 800 bacterial species live in the mouth and many of these congregate at the gingival margin in a thick bacterial plaque, causing chronic gingivitis. Although not usually painful, gingivitis does cause swelling, bleeding and a steady exudative ooze. Up to 20% of patients with gingivitis progress to periodontitis, with destruction of the bony attachment, periodontal pocket formation and eventual loss of mobile teeth. Calcification of plaque is common, forming irregular darkened adherent masses. There are surprising relationships between gingivitis, periodontitis and the heart.[1-4]

THE RELATIONSHIP BETWEEN PERIODONTITIS AND ATHEROSCLEROSIS

Atherosclerosis and consequent ischaemic heart disease are increased with periodontitis, while gingivitis likely has a similar effect. Although the mechanism remains unclear, seeding of oral bacteria into arteries and increased circulating inflammatory mediators seem to be important.[3] Additionally, diabetes predisposes to periodontitis, while periodontitis itself appears to exacerbate diabetes.[4]

INFECTIVE ENDOCARDITIS AND ANTIBIOTIC PROPHYLAXIS

Up to half of sub-acute or chronic infective endocarditis (IE) cases are due to oral organisms, apparently seeding the endocardium following bacteraemia. Many dental procedures produce transient bacteraemia. Patients at risk of IE have long received antibiotic prophylaxis for dental procedures. However, this is controversial because fewer than 15% of patients with IE due to oral bacteria have a history of dental treatment. Antibiotic prophylaxis is currently recommended for patients with significant IE risk due to prosthetic heart valves, previous IE, rheumatic fever with valvular dysfunction, most congenital cardiac malformations, surgical systemic pulmonary shunts, hypertrophic cardiomyopathy or mitral valve prolapse with valvular regurgitation. To avoid the emergence of antibiotic resistant organisms and a subsequent antibiotic resistant bacteraemic shower, prophylaxis should start just one hour before dental treatment, with treatment plans designed to minimise such appointments, separated by at least three week intervals. Oral Amoxicillin (2g) is usually used, or Clindamycin (600mg) in cases of Penicillin allergy.[2]

PROTECTION FROM INFECTIVE ENDOCARDITIS THROUGH PREVENTING GINGIVITIS

Most cases of IE from oral bacteria seem to arise from frequent transient bacteraemia generated by eating and tooth brushing. Bacteraemia is more severe where there is gingivitis, because the bacterial plaque is quite literally rubbed into the haemorrhagic inflamed gingiva. For this reason, it is important for patients at risk of IE to be free of gingivitis and periodontitis.

MANAGEMENT OF GINGIVITIS AND PERIODONTITIS

Daily mechanical removal of plaque cures gingivitis within three weeks, while periodontitis is improved but also often requires additional surgical management. Calcified deposits must first be removed by dental clinicians, because calculus traps bacterial plaque against the gingiva to prevent resolution of inflammation. Chlorhexidine mouthwashes can often initially control plaque and inflammation sufficiently to permit subsequent safe regular mechanical cleansing with a toothbrush. Proper use of tooth brushes and inter-dental cleaning aids is surprisingly difficult, and patients require careful tutelage. Any dentist can remove calculus and provide oral hygiene instruction, but dental hygienists are exceptionally skilled, and specialist periodontists are invaluable for the surgical management of periodontitis.

DESQUAMATIVE GINGIVITIS IN RESPONSE TO SOME CARDIAC DRUGS

Lichen planus often presents in the oral cavity and tends to have a longer course compared with disease primarily affecting skin. Some oral variants cause troubling discomfort and a peculiarity of this is desquamative gingivitis, with atrophy and or loss of the gingival epithelium. Many cases of oral lichen planus appear to be lichenoid reactions to drugs, including ACE inhibitors and Beta blockers.[1,2]

DENTAL MEDICARE AND PREVENTION

Medicare has recently been expanded to provide over $2,000 of dental service via extended care plans where chronic disease may be affected by a dental condition. People with diabetes and those at risk of IE will be particular beneficiaries and I urge the medical profession to embrace the opportunity offered by this initiative.

References available at request. No conflict of interest declared. ❤

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Dental Infection and Vascular Disease

Hans Zoellner, B.D.S., Ph.D.[1]

ABSTRACT

Periodontitis is a chronic inflammatory response to bacterial plaque in which the anchoring bone and soft tissues supporting teeth are destroyed, resulting in tooth mobility and loss. Dental caries involves the spread of infection from the dentine to the vascular dental pulp and periapical bony tissues, before involvement of adjacent soft tissues and spreading sepsis. Several case-controlled, cross-sectional, and cohort studies report correlation between periodontitis and increased cardiovascular, cerebrovascular, and peripheral artery disease, as determined by clinical disease, angiography, ultrasonography, and reduced flow-mediated dilation. Some studies report a similar relationship of atherosclerosis with periapical infection and potentially also with coronal caries, and this review identifies the need to investigate these associations further. Smoking and cadmium exposure are epidemiologically confounding environmental risk factors shared by atherosclerosis and periodontitis. Further complicating epidemiological studies are the risk factors for both atherosclerosis and periodontitis, with which periodontitis appears to have separate positive feedback relationships. These include diabetes, increased plasma lipid levels, hypertension, and white blood cell count. Animal and human intervention studies provide some direct support of a causal role for periodontitis in atherosclerosis, and possible mechanisms include bacterial invasion of arteries, specific atherogenic properties of oral bacteria, the acute phase response, and cytokine polymorphisms.

KEYWORDS: Atherosclerosis, periodontal disease, periapical infection, epidemiology, intervention studies

PERIODONTAL DISEASE, CARIES, AND PERIAPICAL INFECTION

The mouths of most vertebrates are armed with highly calcified teeth for the purposes of defense and masticating food. Although vital for these important physiological functions, by piercing the mucosal barrier from their bony anchorage, teeth also provide a unique opportunity for bacterial invasion (Fig. 1). Plaque bacteria accumulating at the gingival margin perpetually threaten invasion of the soft tissues and bone, and the epithelial attachment, gingiva, and periodontal ligament are evolved to protect from this. If plaque is permitted to irritate the gingivae for any prolonged period, a chronic inflammatory response develops, *gingivitis*, and is characterized by swelling, redness, and susceptibility to bleed. Gingivitis is a successful response to the bacterial onslaught, but on occasion it may progress to periodontal disease in which the soft tissues and bone are destroyed.[1] Most adults have some mild manifestations of periodontal disease, but in 5 to 20% of the population severe

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Coagulopathies and Thrombosis: Usual and Unusual Causes and Associations, Part IV; Guest Editors, Giuseppe Lippi, M.D., Emmanuel J. Favaloro, Ph.D., M.A.I.M.S., and Massimo Franchini, M.D.

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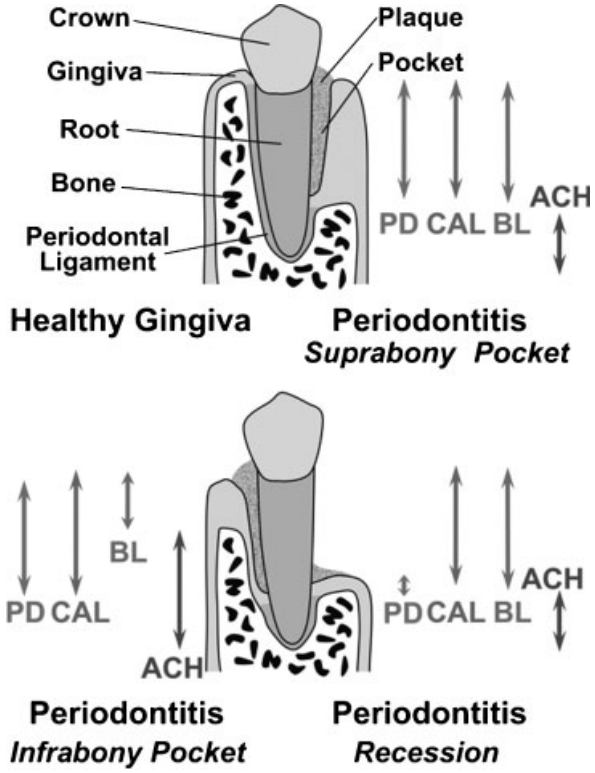

Figure 1 Diagram of anatomical variants of periodontal disease illustrating the numerical measures used to record severity of disease. In health, the gingiva attaches at the cemento-enamel junction where the crown and root meet. The root is anchored into the adjacent alveolar bone via a collagenous periodontal ligament, but this attachment is destroyed when bacterial plaque causes periodontal disease. Most often, suprabony periodontal pockets form in which the periodontal ligament is destroyed and the alveolar bone lost, but with retention of soft tissues to form a deep periodontal pocket colonized by plaque bacteria. Occasionally, however, infrabony pockets form when bone and soft tissues are not lost, despite destruction of the periodontal ligament. When both bone and soft tissues are resorbed together with destruction of the periodontal ligament, the tissues are described as having undergone recession. Not illustrated are vertical defects in which the vertical bony contour changes rapidly, and furcation lesions where periodontitis reaches the branch points of multirooted teeth. Graduated probes are used to measure probing depth (PD) and clinical attachment loss (CAL); radiographs are used to evaluate alveolar crest height (ACH) and bone loss (BL). Notably, the biological significance of these measures clearly depends on the precise anatomical form of periodontitis involved, which has relevance when interpreting published reports.

disease develops, during which teeth become loose and fall out (Fig. 1). Periodontal tissue destruction occurs in bursts independently across the mouth, seemingly due to the accumulation of vascular basement membrane and interstitial amyloid deposits that restrict neutrophil emigration.[2–5] Destruction of the periodontal ligament is the only constant anatomical feature of periodontal disease (Fig. 1) and may be due to direct bacterial invasion, bacterial or host enzyme activity, downgrowth of the epithelial attachment, or proliferation of epithelial embryonic root sheath remnants.[1,5,6] Depending on the pattern of soft and hard tissue resorption, periodontal pockets form and trap highly irritant gram-negative anaerobic bacterial plaque against the remnant tissues, establishing a chronic inflammatory state that may persist for decades (Fig. 1). Although hundreds of bacterial species combine to form the periodontal microbial flora, *Porphyromonas gingivalis* and *Aggegatibacter actinomycetemcomitans* (formerly *Actinobacillus actinomycetemcomitans)* appear particularly important in periodontal disease.[1] Irregular calcification of plaque often occurs and adds to the anatomical difficulty of removing the bacterial irritant that drives periodontal disease. Management of periodontitis involves improved oral hygiene, the removal of plaque and calcified deposits from teeth by careful debridement of periodontal pockets, and, where appropriate, surgical reconstruction to obliterate pocket spaces and restore attachment.

Dental caries is the most prevalent infectious disease in humans and is initiated by attack of the crown by bacterial acids followed by bacterial invasion of dentine and the vascular dental pulp (Fig. 2). The periapical foramen restricts vascular supply to the dental pulp, which limits the inflammatory response such that untreated caries often progresses to pulp necrosis and gangrene. Gangrenous material escaping through the periapical foramen infects the periapical tissues, resulting variously in acute or chronic abscesses, replacement of resorbed bone with chronic inflammatory granulation tissue, or epithelial cysts from stimulated remnants of the embryonic root sheath (Fig. 2). Although distinct pathological entities, these different periapical lesions may progress from one form to another, and spread of infection into the adjacent soft tissues with potentially life-threatening sepsis are the final stages in the natural history of dental caries (Fig. 2).[1] The surgical management of caries involves removal of infected hard tissues and restoration with fillings. In cases where the pulp is necrotic and there is periapical infection, the gangrenous pulp tissues must be removed either by extraction or root canal therapy. In root canal therapy, a series of delicate files is used to remove necrotic material before obliterating the pulp chamber with a root canal filling (Fig. 2).[1]

THE ASSOCIATION OF DENTAL INFECTION WITH VASCULAR DISEASE

Initial Reports That Dental Infection Correlates with Myocardial Infarction and Stroke

Widespread interest in the relationship between dental infection and atherosclerosis was initiated by two

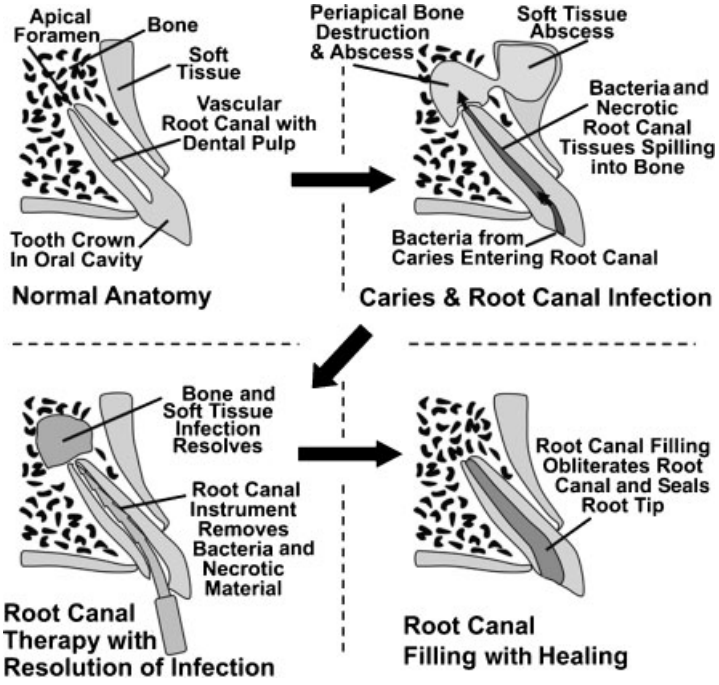

Figure 2 Diagram illustrating the natural history of dental caries and periapical lesions, as well as treatment of periapical lesions by root canal therapy. The tooth is anchored in alveolar bone, with only its crown penetrating into the oral cavity through the mucosal soft tissues. The root canal containing the vascular dental pulp receives its circulation via a delicate apical foramen at the root apex. Bacteria enter the tooth during dental caries and invade the root canal containing the dental pulp. The inability to mount an effective inflammatory response in the dental pulp results in necrosis and gangrene of the pulp, and this necrotic bacterial mix spills into the periapical bony tissue via the apical foramen to cause periapical infection. Periapical infection usually results in bone loss with replacement by chronic inflammatory granulation tissue, and these lesions may also form cysts (not shown) or develop into abscesses as illustrated. Spread of infection into the adjacent soft tissues is common and may progress to widespread sepsis. Treatment of periapical infection requires either extraction or root canal therapy using appropriately delicate files to remove the necrotic material. Once periapical infection has resolved during root canal therapy, the root canal is filled to seal the root tip and prevent recurrent infection.

separate publications in 1989, investigating first acute myocardial infarction[7] and then stroke.[8] Earlier work had indicated a correlation of atherosclerotic disease with a range of infections,[9–13] and this, together with a clinical impression that patients with myocardial infarcts had poor teeth, led to the question of a possible role for dental infection in vascular disease.[7]

An important challenge for these early studies was to develop a meaningful numerical measure for overall dental infection, and to this end a "total dental index" was established taking into account the number of carious lesions, periodontal pocket depth, visibly apparent pus in gingival pockets, the presence of vertical bone loss, the number of periapical lesions, and pericoronitis.[7,8] Total dental indexes were determined for patients with either acute myocardial infarction[7] or stroke[8] and compared with case-matched controls. The association of dental infection with vascular disease was found to be independent of known risk factors for atherosclerosis, establishing a need to consider dental infection itself as a possible independent risk factor.[7,8]

Periodontitis and Cardiovascular Disease

Since these initial important reports correlating a broad range of oral infective processes with vascular disease,[7,8] most further efforts have been directed toward the study of periodontitis. Several similar case-controlled studies have demonstrated a positive correlation between periodontitis and acute myocardial infarction,[14–17] clinically apparent coronary heart disease,[18,19] and both clinical and subclinical coronary artery disease detected by angiography.[20–22] Cross-sectional studies in which associations between variables are evaluated within single populations have similarly supported a relationship between periodontitis and cardiovascular disease.[22–28] Cohort studies in which individuals with and without potentially predisposing conditions are followed over time are generally considered to provide stronger evidence for associations than either case-controlled or cross-sectional analyses,[29] and several such longitudinal cohort studies also support an association between periodontitis and coronary heart disease.[30–35]

The specific criteria for periodontal disease used in these studies and found to correlate with coronary heart disease have varied greatly and include bleeding on

probing,[14,19–21,26] pocket depth,[14–17,19–22] clinical attachment loss,[17,19,21,25,27,28] bone loss or alveolar crest height,[15–19,24,26] the worst pocket depth,[23,24] the number or presence of periodontal pockets,[18,30,31,34,35] the number of vertical periodontal defects,[18] involvement of furcation areas between molar roots,[26] the extent of plaque or calculus deposits,[20,27,30] self-reported bleeding gums,[23] a total dental index,[32] and the sum of scores for missing teeth, apical lesions, caries, and marginal bone loss.[33]

There has been similar diversity among these studies in the specific criteria used for cardiovascular disease, which include acute myocardial infarction,[14–16,28] survival of earlier myocardial infarction,[17,35] clinically confirmed chronic coronary artery disease and/or angina,[14,18,19,24,25,27,28,32,35] angiographically confirmed coronary artery disease,[20–22] the extent of multiple vessel disease as determined by angiography,[27] self-reported cardiovascular disease,[23,26] admission to hospital for cardiovascular disease,[25,28,30] and finally death from cardiovascular disease.[24,28,30–35]

This diversity, expressed not only in the features of periodontal disease considered for analysis, but also in the criteria for cardiovascular disease, may account for some of the variability seen among studies in the strength of association between these two conditions. Nonetheless, a recent meta-analysis of a range of case-controlled and cross-sectional studies strongly supports the relationship between cardiovascular disease and periodontitis, with a pooled odds ratio [OR] of 2.35 (95% confidence interval [CI], 1.87 to 2.96).[36]

Throughout, investigators have made significant effort to control for potentially confounding variables including age and gender,[14–16,18–23,25–27,30–35] smoking,[14,16,18–20,22,23,25,26,28,30–32,34,35] alcohol consumption,[19,20,28,30,34,35] body mass index,[18,19,25,28,30,32,34,35] physical activity,[19,20,30] race or ethnicity,[25,30] educational level,[18–20,23,25,30,35] socioeconomic status,[19,20,23,30,32,35] marital status,[30,35] place of birth,[18] locality,[20] blood pressure and hypertension,[25,26,28,30–32,34,35] diabetes mellitus,[18,22,25,28,30–32,34,35] blood glucose,[25,34,35] total serum cholesterol,[14,19,22,25,30–32,34,35] serum triglyceride,[14,19,25,32,34,35] serum low-density lipoprotein (LDL),[25,32,34] and serum high-density lipoprotein (HDL).[14,22,25,32,34,35] Although not all studies considered all potential confounding factors, taken as a whole, these studies do suggest that the association of periodontitis with cardiovascular disease is independent of other commonly recognized risk factors.

The Relationship Between Periodontitis, Stroke, and Subclinical Atherosclerosis

Since the initial report in 1989,[8] several further case-controlled studies,[8,37–41] cross-sectional studies,[24,42,43] and cohort studies[44–47] have demonstrated positive and independent correlation between periodontitis and stroke. Consistent with this is a similar correlation between periodontitis and transient ischemic attack in case-controlled[38,48] and cross-sectional studies.[43] There is also an association between periodontitis and peripheral vascular disease.[49,50]

Although overt atherosclerotic disease is clinically important, it is scientifically interesting that subclinical atherosclerosis, as revealed by coronary angiography,[51] altered carotid intima-media thickness,[52,53] or ultrasonographically or radiographically revealed carotid atherosclerotic plaques,[54–56] also correlate with periodontitis in case-controlled[52] and cross-sectional studies,[51,53–56] independent of separate confounding risk factors.

Tooth Loss Correlates with Atherosclerotic Disease

Tooth loss is an important clinical outcome of dental disease, and it is also much easier to record data on missing teeth than on periodontitis. For these reasons, it has been valuable to consider coronary heart disease with regard to tooth loss,[21,23,25,27,46,57–59] edentulism,[18,34,35] or denture wearing,[18] independent of periodontitis in case-controlled,[18] cross-sectional,[26,60,61] and cohort studies.[46,57–59] Stroke[25,43,46] and transient ischemic attack[43] also correlate with tooth loss[46] and edentulism[25,43] in cross-sectional[25,43] and cohort studies.[46]

The association of tooth loss with atherosclerosis may be a statistical epiphenomenon reflecting dental infection sufficiently serious to result in extraction. However, the possibility remains that tooth loss itself influences propensity for vascular disease, independent of preceding dental infection. Possible mechanisms for such a direct effect of tooth loss include consumption of a proatherogenic diet with reduced chewing efficiency and chronic denture-associated candidal infection.

Periapical Infection and Atherosclerosis

It is important to recall that caries and consequent periapical infection is the main cause of tooth loss.[1,62,63] Recalling also that the earliest reports in this area evaluated overall dental infection including caries and periapical lesions,[7,8] it seems that investigators have neglected possible associations of atherosclerosis with caries and periapical infection relative to periodontitis. Nonetheless, some case-matched,[64] cross-sectional,[37,56] and cohort studies[32,33] have been performed supporting a similar independent relationship between periapical infection and coronary artery disease,[32,33,64] stroke,[37] and, to a lesser extent, subclinical carotid athoerosclerosis[56] to that seen in periodontitis.

Studies relating atherosclerosis to periapical infection suggest it is not any particular quality of periodontitis

driving the correlation with atherosclerosis, but rather it is the total infective load that may be important. Supporting this idea is a dose–response relationship between increasing myocardial infarction and an increasing number of periapical lesions,[64] as well as an inverse relationship between the number of completed root canal therapies and myocardial infarction.[64] In addition, a cross-sectional study in which periodontal disease and periapical lesions were considered separately demonstrated independent association of these two dental infections with stroke.[37]

Although it seems likely that periapical infection rather than coronal caries is the most important aspect of caries relating to atherosclerosis, this is an assumption unsupported by published data, and it is problematic that no reports have described the individual relationship with atherosclerosis of coronal caries, periapical infection, and periodontitis. Moreover, although there are several distinct pathological forms of periapical lesion (Fig. 2),[1] the effect of this diversity on the association with atherosclerosis has not been studied. There is both need and scope for significantly more research investigating the relationship of atherosclerosis with both coronal caries and periapical infection.

Factors Complicating Interpretation of the Relationship Between Vascular Disease and Dental Infection

Although all of the published studies investigating the relationship between atherosclerosis and dental infection describe attempts to control for the effect of known separate risk factors for vascular disease, the possibility remains that dental infection and atherosclerosis share as yet unidentified risk factors.

Smoking[65–69] and cadmium exposure[70] are known environmental risk factors shared by atherosclerosis and periodontitis, and they can in principle be readily controlled for in studies investigating the relationship between the two diseases.

Disease-associated risk factors shared by atherosclerosis and periodontitis are surprisingly difficult to disentangle because of apparent feedback relationships between periodontitis and the risk factors involved. For example, diabetes mellitus is a risk factor for both atherosclerosis and periodontitis,[71–73] but treatment of periodontal disease appears to improve diabetic control,[74,75] making it difficult to control for the effect of diabetes in studies examining periodontal and vascular disease. Similarly, hypertension,[76–78] high white blood cell count[76,79–82] elevated total plasma cholesterol,[82–86] raised plasma LDL levels,[82–84] elevated very low-density lipoprotein (VLDL),[86] raised fatty acid,[86] and increased plasma triglyceride levels,[83,85–88] as well as reduced plasma HDL levels,[82,89] are also well-established risk factors for both atherosclerosis and periodontitis but improve with periodontal therapy,[90–96] suggesting a feedback relationship between periodontitis and these risk factors. Although most studies seek to control for the overlap of such shared risk factors, there seems to be a need for any epidemiological data linking atherosclerosis with periodontal disease to be interpreted in a highly conservative manner.

A common observation made across many of the studies currently reviewed is that the association between periodontitis and atherosclerotic changes is stronger in younger people[19,35,40,47] and sometimes not seen in older subjects.[19,35] One reasonable explanation for this may be the cumulative nature of periodontal disease and tooth loss, such that as subjects age there are fewer teeth available for periodontitis to act on.[25]

The specific periodontal variable measured has a bearing on study outcomes, with one case-controlled study, for example, reporting different ORs for myocardial infarction dependent on whether pocket depth (OR: 2.19; 95% CI, 1.66 to 2.89), clinical attachment loss (OR: 1.46; 95% CI, 1.26 to 1.69), alveolar crest height (OR: 1.3; 95% CI, 1.14 to 1.49), or the number of missing teeth (OR: 1.04; 95% CI, 1.02 to 1.07) was used to perform the calculation.[17] Although at first confusing, this differential outcome might be explained in context of the active inflammation inherent to all deep pockets, which would be greatly reduced when there is recession (Fig. 1). Although such differential results indicate a need for clarification of the most biologically relevant periodontal measures for future studies,[17,97] they may also provide guidance as to possible pathological mechanisms.

The complicating factors just outlined may account for several case-controlled,[98,99] cross-sectional,[100] and cohort studies[101–105] in which no clear association between dental infection and atherosclerosis was seen. In light of the previously described complexities, it is difficult to achieve certainty from epidemiological observations alone of a causal relationship between dental infection and atherosclerosis. Fortunately, some data are available from both human intervention studies and animal experiments that can inform interpretation of epidemiological studies.

Animal Studies Supporting a Causal Relationship of Periodontitis for Atherosclerosis

A variety of animal model systems have been used to study the relationship between periodontal disease and atherosclerosis including New Zealand rabbits fed a high-fat diet,[97,106,107] transgenic ApoE-deficient mice,[108–114] and pigs.[115]

The ability of periodontopathic organisms to increase atherosclerosis is well demonstrated in experiments where intravenous infusion of *P. gingivalis*,[108,111,115] or *A. actinomycetemcomitans*,[113,114] in

ApoE-deficient mice[108,111,113,114] or pigs[115] increases the severity of atherosclerotic lesions. The potentially causal role of periodontitis is, however, more directly demonstrated in experiments where atherosclerosis is made more severe upon induction of periodontitis by oral infection with *P. gingivalis* in rabbits fed a high-fat diet[97,106] or in apolipoprotein E (ApoE)-deficient mice.[109,110]

P. gingivalis DNA has been detected in aortic mouse atherosclerotic lesions,[109,110] as well as in both aortae and coronary arteries of pigs,[115] supporting a role for direct bacterial invasion. The importance of bacterial cell attachment to the arterial wall is illustrated by reduced atherogenic activity for mice of a fimbrial-deficient strain of *P. gingivalis*.[110]

Immunization of apolipoprotein E–deficient mice against *P. gingivalis* is protective against atherosclerosis exacerbated by *P. gingivalis*,[110–112,116] further supporting a causative role for oral bacterial infection in vascular disease.

Human Intervention Studies for Atherosclerosis by Treating Periodontal Disease

The most convincing evidence of a causal role for periodontal disease in human atherosclerosis would be clinical intervention trials demonstrating protection against vascular disease by treating periodontitis.[29,117] Making this difficult, however, is that both periodontitis and atherosclerosis progress over many decades, and both diseases also have only limited potential for recovery. As a consequence, it is recognized that intervention trials may not readily detect an effect of periodontal treatment on atherosclerosis outcomes.[29,97]

A range of serum biomarkers correlate with increased atherosclerosis including increased C-reactive protein (CRP), fibrinogen, interleukin(IL)-6, total cholesterol, and LDL, as well as reduced HDL.[118–123] Reduction in response to treatment for periodontitis of circulating levels of CRP,[90,93–95,124–129] IL-6,[90,95,124,125,128–131] fibrinogen,[93,94,128] total cholesterol,[90] and LDL,[130] as well as increased HDL,[95] supports a role for periodontal disease in atherosclerosis. Also, treatment of periodontitis is reported to reduce circulating $CD4^+HLA\text{-}DR^+$, $CD4^+CD44^+$, and $CD4^+CD49d^+$ T cells suggested as contributing to atherogenesis.[94] Significant for the potential therapeutic value of periodontal interventions is that these changes in T cells as well as improvements in circulating levels of CRP and fibrinogen are largely lost by 12 months post-treatment, apparently reflecting recurrence of periodontal disease.[94] Periodontal treatment failed, however, to reduce fibrinogen levels in a recent study, although a surprising increase in hematocrit and hemoglobin was reported.[132]

Endothelial dysfunction as reflected by reduced flow-mediated dilation of the brachial artery is considered a reasonable indicator for early atherosclerotic disease,[133] and improved flow-mediated dilation following treatment for periodontitis,[126,131,134–137] without improvement in responsiveness to nitroglycerin,[126,131,134,135] is consistent with a causative role for this oral disease in atherosclerosis.

Anatomical demonstration of improved atherosclerosis following treatment of human periodontitis seems currently limited to a single report of reduced carotid intima-media thickness together with the previously mentioned changes in CRP, fibrinogen, and lymphocyte populations.[94]

A large multicenter randomized controlled Periodontitis and Vascular Events (PAVE) trial is currently underway, evaluating the effect on cardiovascular events of periodontal therapy as compared with "community dental care." Preliminary results thus far reported are equivocal, with no statistically significant improvement in serious adverse events between the two groups studied, although a modest trend toward improved vascular outcomes is seen.[138,139] The PAVE trial, however, is at a very early stage, and it may be too early for statistically meaningful data to have emerged.

MECHANISMS LINKING DENTAL INFECTION TO VASCULAR DISEASE

Direct Bacterial Infection of the Arterial Wall

In periodontitis, expanded blood vessels are in intimate contact with an atrophic pocket epithelium so that bacterial plaque may be separated from circulating blood by as few as two cells.[140] Bacteremia is consequently frequent in periodontitis and provides a pathway for periodontal pathogens to access the arterial wall. DNA of *A. actinomycetemcomitans*,[141–143] *P. gingivalis*,[115,141–145] and other periodontopathic organisms[141–143,145] has been demonstrated in carotid,[141,144] coronary,[115,142,145] and aortic[115,143] endarterectomy specimens. DNA from streptococci implicated with caries and not periodontitis has also been found in atherosclerotic plaques,[143] suggesting that caries should perhaps be investigated as a potential independent risk factor for atherosclerosis.

The Virulence of Oral Bacteria for Atherosclerosis

Circulating antibody levels against a range of periodontopathic bacteria correlate with intima-medial wall thickness, and the plaque microbial load for these species is also proportionate to cardiovascular disease,[29] supporting an atherogenic role for these organisms. *P. gingivalis* seems particularly important as a potential microbial link between human periodontitis and atherosclerosis,

as indicated by correlation between circulating specific antibody and risk for stroke[146] and cardiovascular disease,[61,147,148] and antibody levels against *A. actinomycetemcomitans* are also correlated with increased coronary heart disease[61,149] and stroke.[150] Adhesion of *P. gingivalis* is strongly affected by the fimbrial genotypes expressed, and it is interesting that there is overrepresentation of some fimbrial forms in atherosclerotic lesions.[151] Streptococci, normally associated with caries rather than periodontal disease, also have potential to contribute to arterial vascular disease via thrombogenic activity,[152] and potentially prothrombotic von Willebrand factor correlates with levels of circulating antibody against *A. actinomycetemcomitans*.[153]

Lipopolysaccharide shed by gram-negative organisms such as *P. gingivalis* associates preferentially with VLDL and intermediate-density lipoproteins, and it has been suggested that these lipoproteins deliver lipopolysaccharide from periodontal lesions to atherosclerotic plaques.[154] A range of separate potentially atherogenic activities has been demonstrated for *P. gingivalis* in cell culture experiments including increased human endothelial apoptosis,[155] increased endothelial expression of monocyte chemoattractant protein-1,[155,156] stimulation of U-937 monocyte adhesion to aortic endothelium,[157] increased tissue factor and reduced tissue factor pathway inhibitor expression in human aortic endothelium,[158] and platelet aggregation.[159] Of interest is that increased levels of CRP are associated with elevated levels of *P. gingivalis* and *A. actinomycetemcomitans* in dental plaque.[160] However, in light of the large number of other species contributing to the microbial plaque of periodontitis lesions, it seems likely that there are many other important but as yet uninvestigated microbial activities of relevance for atherosclerosis. This is supported by correlation between hypertension and discrete patterns of oral microbial ecology considered causative for periodontitis.[161]

The Acute Phase Response in Periodontitis and Atherosclerosis

In the acute phase response, IL-6 released from inflamed tissues accesses the liver via the circulation to stimulate greatly increased synthesis of a range of plasma proteins including CRP and fibrinogen.[162] Because serum IL-6[129,163] and CRP[79–81,127,129,163–165] are elevated in periodontitis, and periodontal therapy reduces circulating IL-6,[124,125,127–129,163] CRP,[94,124,125,128,129,163] and fibrinogen levels,[94,128] the acute phase response is suggested as important in mediating the link between periodontal disease and atherosclerosis.[5,166,167]

IL-6 synthesis is highly inducible in cultured endothelium,[168] and it is noteworthy that IL-6 mRNA and protein is expressed by endothelium in inflamed periodontal tissues.[169,170] Supporting periodontal endothelium as a likely source for IL-6 is that these cells have a unique opportunity to secrete IL-6 directly into the circulation, and ultrastructural features of high endothelial-like venules in periodontitis are consistent with high synthetic activity.[5,171] Plasminogen activator inhibitor (PAI)-1 is also a major product of endothelium,[172] and raised plasma levels of this antifibrinolytic protein in periodontitis suggests a role in atherogenesis.[81,173] Also, a case-matched study demonstrates reduced circulating levels of antithrombotic protein C with periodontitis.[165]

Cytokine Polymorphisms in Periodontitis and Atherosclerosis

Polymorphism among inflammatory cytokines may account for individual differences in host response. Notably, polymorphisms in IL-1,[174,175] IL-6,[174] and tumor necrosis factor α[174] are associated with enhanced systemic changes in periodontal disease, including elevated serum IL-6 and CRP levels,[174] as well as increased susceptibility to cardiovascular disease.[175]

CONCLUSION

Despite uncertainties inherent in considering individual studies, a balanced reading of the current literature strongly supports the suggestion that periodontal disease contributes to atherosclerosis, and several plausible mechanisms are supported by published data. Nonetheless, the distinct roles of coronal caries and periapical infection in atherosclerosis remain unclear, as do the precise atherogenic mechanisms involved. The outcome of long-term intervention studies determining the effect of dental treatment on atherosclerosis will be of interest not only with regard to improved understanding of the systemic effects of dental disease but also from the perspective of providing a further therapeutic avenue for vascular health.

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Description of comprehensive dental services supported by the Medicare Chronic Disease Dental Scheme in the first 23 months of operation

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Abstract

Objective: Australia's Medicare universal insurance system has supported comprehensive dental service through the Chronic Disease Dental Scheme (CDDS) since November 2007. Public debate opposing CDDS includes claims of over-servicing, calls for expansion to universal eligibility, and government threat of closure. Here we examine CDDS services over the first 23 months of operation.

Methods: CDDS statistics on patient age, gender and item numbers claimed from November 2007 to December 2009 from Medicare were subjected to analysis.

Results: The distribution of 404,768 total CDDS patients varied across Australia from 3.6% of the population in NSW to 0.07% in NT, while uptake increased over time. The average patient had 7.58 dental treatments, and the most common were: direct restorations (2.27), preventive and periodontal services (1.46), diagnostic services (1.43), extractions (0.77), and new dentures (0.53). Crown and bridgework appeared over-represented (0.48).

Conclusion: Although data do suggest over-servicing in crown and bridgework, there also appears to be significant community need for the CDDS.

Implication: Clear guidelines for dental clinical diagnosis and treatment planning, as well as a pre-approval process for crown and bridgework is suggested to improve the CDDS, and this could form the basis for expansion to universal eligibility for dental Medicare.

Key words: dental, medicare, chronic disease, over-servicing, treatment planning

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There is evidence for association between dental infection and several systemic conditions including: central and peripheral vascular disease, diabetes, poor birth outcomes and aspiration pneumonia.[1-6] Although mechanisms are unclear, some animal and human intervention studies suggest a causative contribution of oral infection to these systemic conditions,[2,7-14] so that improved control of dental infection may reduce the burden of separate systemic disease. Dental infection also contributes substantially to preventable hospitalisation.[15,16] Despite these broad health implications, dental services are not included in Australia's Medicare universal health insurance system, and the cost of private dental services significantly limits access.[17]

In November 2007 the Enhanced Primary Care Dental Initiative, which provided limited dental funding, was expanded to become the Medicare Chronic Disease Dental Scheme (CDDS). Under the CDDS, eligible patients may receive Medicare benefits of up to $4,250 for eligible services provided under the CDDS during two consecutive calendar years. Eligibility for CDDS is determined by general medical practitioners, who identify the presence of a chronic systemic disease that may be adversely affected by the patient's dental condition. Medical practitioners are required to prepare multidisciplinary care plans including dental treatment, in order for patients to access the CDDS.

Although the Federal Government elected in December 2007 indicated cessation of CDDS, the scheme has continued with the support of the Senate. The public availability of data on CDDS service provision provides an unprecedented opportunity to examine patterns of private dental service heavily subsidised by the Australian Federal Government via Medicare.

A brief review of common dental disease and treatment is necessary. Caries involves bacterial invasion and destruction of tooth structure, and is treated by removal of infected tissue followed by 'filling' with dental restorations. In untreated caries, bacteria invade the vascular dental pulp with an eventual spread to bone at the root apex (Figure 1). The spread of infection to soft tissues may be followed by life-threatening sepsis. Restorations may be either direct into teeth, or alternatively 'indirect restorations' cemented into place after preparation using plaster models. Direct restorations may be of amalgam, mostly used in posterior teeth, or of adhesive tooth-coloured material. Although adhesive materials are aesthetically pleasing, amalgam has greater longevity.[18,19] When tooth structure is severely compromised, it becomes necessary to 'cut the tooth back' to a thimble shape to accept indirectly prepared crowns enclosing the entire tooth surface (Figure 1). Inlays are smaller indirect restorations of gold or porcelain. Most restorations eventually require replacement

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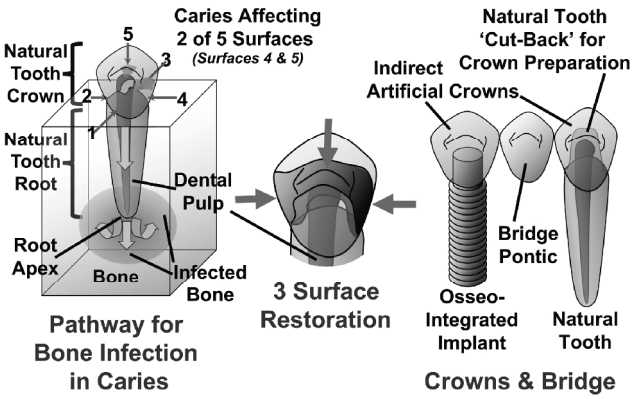

Figure 1: Diagrams illustrating: spread of caries infection to apical bone via the pulp; the 5 separate tooth surfaces susceptible to disease; a three surface restoration; osseointegrated implants; a bridge pontic; crowns; and the typical thimble-shaped preparation of a natural tooth needed to make a crown.

accompanied by additional loss of tooth structure, so that a conservative approach is preferred. If caries infection has spread to the pulp or bone, the tooth must either be extracted, or an endodontic procedure performed to remove infected dental pulp and obturate the pulp chamber with a 'root canal filling' (Figure 1).

In periodontitis, irritant plaque destroys the periodontal ligament and bony tooth support with eventual tooth loss. Treatment includes improved oral hygiene, curettage, and surgical tissue remodelling.

When only some teeth are lost, they may be replaced using either bridges or partial dentures. A bridge consists of a false tooth 'pontic' held in place by two crowns on either side of the offending gap. While bridges are permanently cemented into place, partial dentures are removable and fixed by clasps grasping natural teeth. Denture clasps may be part of a cast metal framework, or alternatively just wires embedded in a resin base. Partial dentures with a cast metal framework are generally preferred over those with an acrylic base because of greater comfort and longevity. While any ill-fitting partial denture may traumatise gingiva and act as a plaque and food trap, these problems seem less pronounced when cast metal frameworks rather than acrylic bases are used.

When all teeth are lost, replacement is with a full denture. Osseointegrated implants may be used to anchor full dentures, partial dentures and also crowns or bridges.[20]

General dental practitioners and specialists deliver the full range of dental services, while prosthetists with dental technician background and additional clinical training are able to make dentures and denture repairs.

There has been public discussion of possible over-servicing in the CDDS,[21-23] while the federally appointed National Health and Hospital Commission recently recommended a separate universal dental health insurance program,[24,25] and the Greens political party has announced a policy of expanding the CDDS to include the entire population.[26,27] This study aims to describe patterns of service delivered under the CDDS over the first 23 months of implementation, both in light of and to inform public discussion.

Methods

Collection of data

Data on services defined by specific item numbers between November 2007 and December 2009 was from the Medicare website (http://www.medicareaustralia.gov.au). Table 1 shows the item numbers used and grouped for analysis in this study. Item numbers were selected to avoid double counting, as item numbers for services intermediate in the restoration or root canal − endodontic therapy of individual teeth were excluded from the analysis, as were item numbers for intermediate steps or components of prosthetic, surgical and preventive services. Item numbers were grouped to facilitate analysis of treatment type, and were indicative of the number of: diagnostic; preventive and periodontal; extraction; general surgical; acute pain and dental emergency; chronic pain management; endodontic; direct restorative; indirect restorative; bridge; implant; orthodontic; denture; and denture repair services on a per tooth or patient basis. The Medicare website provides data using a similar but less comprehensive grouping, which has the disadvantage of 'double counting' on a per tooth or patient basis, but is nonetheless more helpful with regard to determining the total cost of treatment. For this reason, where costs are compared in the current study, these data were obtained directly from the Medicare website according to the grouping used by Medicare. Patient number was estimated from those item numbers used for diagnostic examination, while the study was limited to the first 23 months of the CDDS to minimise the likelihood of including patients returning after an initial course of care for maintenance therapy.

Statistical analysis

Statistical analysis assessed data from those who have accessed CDDS services with reference to the wider population who are eligible, but who may not have not used the scheme. The chi-square test was used to evaluate the statistical significance of differences in proportion between groups,[28] and binomial analysis of proportion by approximation was applied in considering single statistics using the calculator available at http://faculty.vassar.edu/lowry/binomialX.html, assuming an expected proportion of 0.5. Confidence intervals (CI) are indicated at the 95% level and were calculated without correction for continuity.

Results

The distribution of CDDS patients across Australia

The total number of CDDS patients treated as well as the number of discrete treatments according to type is shown in Table 2. Of the Australian population, 1.8% accessed CDDS, although this varied between state jurisdictions ranging from a maximum of 3.6% of the New South Wales (NSW) population to 1.8% in Victoria; 1.4% in South Australia (SA); 0.57% in Queensland; 0.34% in the Australian Capital Territory (ACT); 0.28% in Tasmania; 0.08% in Western Australia (WA); and 0.07% in the Northern Territory (NT), with differences all statistically significant to $p<0.001$ with the exception of that between WA and NT. A separate table showing state-level data is available at the University of Sydney website (http://hdl.handle.net/2123/7744).

Table 1: Medicare Item Numbers used for analysis grouped according to the type of service delivered (X- is 85- for general dental practitioners, 86- for specialist dental practitioners, and 87- for prosthetist delivered services).

Item No. Suffix	Treatment Description
Patients Treated	
X-011, X-012, X-013, X-014, X-015	Comprehensive, Periodic, and Limited (Emergency) Oral Examination, and Consultations
Diagnostic Services	
X-022, X-025, X-031, X-035, X-036, X-037, X-038, X-039, X-051	Intraoral, Extraoral, Temporomandibular Joint, Cephalometric, Panoramic, Hand-Wrist and Skull Tomographic Radiology, and Biopsies
Preventive and Periodontal Services	
X-047, X-111, X-113, X-114, X-115, X-117, X-121, X-123, X-131, X-141, X-161, X-171, X-221, X-222, X-225, X-231, X-232, X-233, X-234, X-235, X-236, X-238, X-241, X-245	Caries Activity Testing, Scaling, Cleaning, Recontouring Restorations, Internal Bleaching, Remineralisation, Dietary Advice, Oral Hygiene Instruction, Fissure Sealing, Odontoplasty, Periodontal Recording, Root Planing and Curetage, Non-Surgical Periodontal Therapy, Soft Tissue and Bone Surgical Periodontal Therapy Including Grafts and Gingivectomy
Extractions	
X-311, X-314, X-316, X-322, X-323, X-324, X-326	Simple, Sectional and Surgical Extractions Including Removal of Root Fragments and Bone
General Surgical Procedures	
X-331, X-332, X-337, X-338, X-341, X-343, X-344, X-345, X-371, X-373, X-375, X-376, X-377, X-378, X-379, X-381, X-382, X-384, X-385, X-388, X-389, X-391, X-393, X-395	Bone and Soft Tissue Plastic Surgery including Grafts and Hyperplastic Tissue, Repositioning Muscle Attachment, Cysts and Tumours, Removal of Scars, Salivary Duct and Gland Surgery, Removal of Foreign Bodies, Cyst Marsupialisation, Surgical Exposure and Ligation of Teeth, Repositioning Teeth, Transplantation of Teeth or Tooth Bud, Isolation and Preservation of Neurovascular Tissue, and Surgery Involving the Maxillary Antrum
Acute Pain and Dental Emergencies	
X-213, X-386, X-387, X-392, X-412, X-419, X-911, X-927, X-986	Acute Periodontal Infection, Splinting Displaced Teeth, Drainage of Abscess, Endodontic Emergency, Palliative Care, Prescription
Chronic Pain Management	
X-165, X-394, X-926, X-965, X-966, X-968, X-971, X-972, X-981	Desensitisation, Surgery for Osteomyelitis, Occlusal Splint and Adjustment, Physiotherapy, Splinting and Stabilisation
Endodontic Services	
X-414, X-417, X-431, X-432, X-433, X-434, X-436, X-437, X-438, X-457	Pulpotomy, Peri-Radicular Surgery and Apicectomy, Apical Seal, Treatment of Perforation and Resorption
Direct Restorations (Amalgam and Adhesive)	
X-511 to 5, X-521 to 5, X-531 to 5, X-576	Posterior Amalgams, Posterior Adhesive and Anterior Adhesive Restorations with from 1 to 5 Surfaces, and Stainless Steel Crowns
Indirect Restorations (Crowns and Inlays)	
X-541 to 5, X-551 to 5, X-613, X-615, X-618	Indirect Inlays with 1 to 5 Surfaces, and Crowns (Metal, Porcelain, Veneered)
Bridge Pontics	
X-642, X-643	Pontics for Bridges (For Analysis 2 Crowns are Assumed Needed Per Pontic)
Osseointegrated Implants	
X-661, X-664, X-666, X-671, X-672, X-673, X-684, X-688	Implant Abbutments, Bars and Crowns Attached to Implants, Two and One Stage Implants
Orthodontic Services	
X-811, X-821, X-823, X-829, X-831	Removable and Fixed Appliances, Partial and Full Banding
Dentures	
X-711, X-712, X-719, X-721, X-722, X-727, X-728	Full Dentures, Partial Resin Dentures, and Partial Metal Framework Dentures
Denture Repairs	
X-741, X-743, X-744, X-745, X-746, X-751, X-752, X-753, X-761, X-762, X-763, X-764, X-765, X-767, X-768, X-769	Adjustment, Relining, Remodelling, Cleaning and Polishing, Reattaching and Replacing Teeth and Clasps, Repairing Broken Base, and Adding Teeth

The item numbers selected for analysis provide information at the level of services per tooth (extractions, endodontic services, direct restorations, indirect restorations) or per patient (diagnostic services, preventive and periodontal services, general surgical procedures, acute pain and dental emergencies, chronic pain management, bridge pontics, osseointegrated implants, orthodontic services, dentures, and denture repairs). The range of dental services supported by the CDDS is extensive and permits comprehensive dental care of patients

Table 2: The number of CDDS supported patients and services according to treatment type.

	General dental practitioner services	Specialist services	Prosthetist services	Total services for all practitioners
Patients treated				
Services	352,298	6,282	46,188	404,768
95% CI	351,879-352,717	6,128-6,436	45,792-46,584	-
Diagnostic services				
Services	568,015	9,122		577,137
95% CI	566,682-569,348	8,935-9,309		575,795-578,479
Preventive and periodontal services				
Services	581,224	8,456		589,680
95% CI	579,879-582,569	8,276-8,636		588,327-591,033
Extractions				
Services	305,281	6,587		311,868
95% CI	304,253-306,309	6,428-6,746		310,831-312,905
General surgical procedures				
Services	4,958	338		5,296
95% CI	4,820-5,096	302-374		5,153-5,439
Acute pain and dental emergencies				
Services	48,361	362		48,723
95% CI	47,933-48,789	325-399		48,294-49,152
Chronic pain management				
Services	23,677	388		24,065
95% CI	23,377-23,977	349-427		23,762-24,368
Endodontic Services				
Services	44,992	1,344		46,336
95% CI	44,579-45,405	1,272-1,416		45,917-46,755
Direct restorations (amalgam and adhesive)				
Services	916,834	1,631		918,465
95% CI	915,262-918,406	1,552-1,710		916,893-920,037
Indirect restorations (crowns and inlays)				
Services	193,261	293		193,554
95% CI	192,427-194,095	259-327		192,719-194,389
Bridge pontics				
Services	41,295	47		41,342
95% CI	40,899-41,691	34-60		40,946-41,738
Osseointegrated implants				
Services	2,657	1,559		4,216
95% CI	2,556-2,758	1,482-1,636		4,089-4,343
Orthodontic services				
Services	1,977	934		2,911
95% CI	1,890-2,064	874-994		2,805-3,017
Dentures				
Services	158,767	459	56,998	216,224
95% CI	158,007-159,527	417-501	56,534-57,462	215,345-217,103
Denture repairs				
Services	62,960	239	25,281	88,480
95% CI	62,473-63,447	209-269	24,971-25,591	87,905-89,055
Total number of individual procedures performed, per tooth – patient basis				
Services	2,954,259	31,759		3,068,297
95% CI	2,953,610-2,954,908	31,412-32,106		

The majority of CDDS patients were seen by general dental practitioners only, although prosthetists and dental specialist practitioners also provided an appreciable number of services.

The rate of CDDS uptake significantly increased towards the end of the study period (Figure 2). While the absolute percentage of state population using CDDS in the month of December 2009 in WA and NT remained much lower than all other states (0.006% and 0.008% respectively, $p<0.001$), there was increased use in Tasmania (0.02%), ACT (0.03%), Queensland (0.06%), Victoria (0.14%), and SA (0.15%) ($p<0.05$), and NSW residents had the highest percentage population use of CDDS per month at the end of the study period (0.21%) ($p<0.001$). Of note is a brief dip in CDDS use from April to June 2008.

The age and gender of CDDS patients

Figure 3 illustrates the number of CDDS patients according to age group, gender and whether seen by prosthetists or dentists. More women were treated than men by both prosthetists and dentists ($p<0.001$), while dentists treated substantially more patients compared with prosthetists ($p<0.001$) (Table 2, Figure 3). Although most patients were over the age of 54 ($p<0.001$), and prosthetists treated an older cohort compared with dentists ($p<0.001$), 1,449 patients were under the age of 15.

Patterns of service delivered under the CDDS

Similar patterns of treatment were seen across states and territories (data available at the University of Sydney website, http://hdl.handle.net/2123/7744). Of 7.580 average dental treatments per patient across Australia, the most common services were: direct restorations (2.269, 95% CI 2.265-2.273); preventive and periodontal services (1.457, 95% CI 1.455-1.462); diagnostic

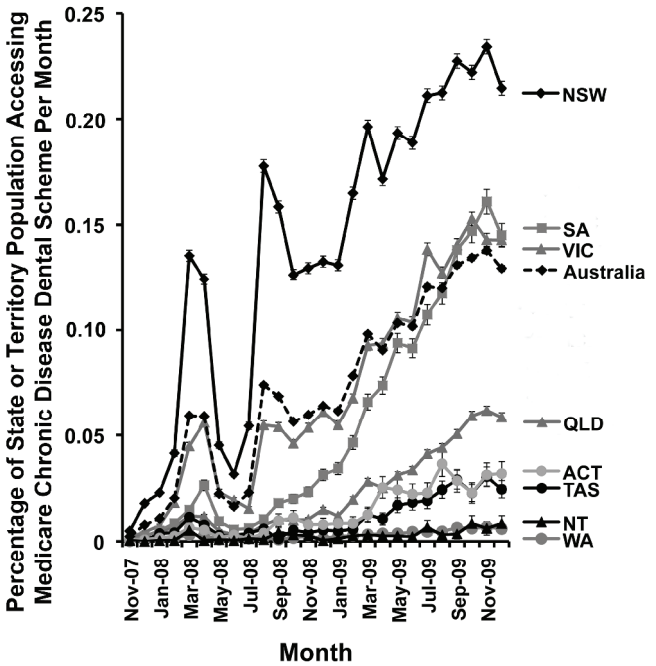

Figure 2: Graph showing the percentage of total state, territory or national population undergoing dental examination in the CDDS per month from November 2007 to December 2009. There was steadily increasing use of the CDDS, although uptake varied greatly between jurisdictions.

services (1.426, 95% CI 1.422-1.429); extractions (0.770, 95% CI 0.768-0.773); new dentures (0.534, 95% CI 0.532-0.536); and indirect restorations (0.478, 95% CI 0.476-0.480).

Among all restorations placed, there were proportionately more direct (82.6%, 95% CI 82.5-82.7) than indirect restorations (p<0.001) (Table 3). Comparing jurisdictions, however, while NSW (20.0%, 95% CI 19.9-20.1) and Tasmania (18.9%, 95% CI 17.6-20.2) had comparable rates of indirect restorations, these two states delivered proportionately more indirect restorations compared with other states and territories considered together (12.7%, 95% CI 12.6-12.8) (p<0.001, data available at the University of Sydney website, http://hdl.handle.net/2123/7744) where the proportion ranged from 16% in WA to 5% in NT. Separately, there were proportionately more crowns related to bridges in NSW and Victoria (43.9% of crowns, 95% CI 43.6-44.2), than in other states considered together (31.2% of crowns, 95% CI 30.5-31.9) (p<0.001, data available at the University of Sydney website, http://hdl.handle.net/2123/7744). Adhesive materials were used in the great majority of posterior restorations (92.1%, 95% CI 92.0-92.2) in preference to amalgam (p<0.001). Oral hygiene instruction was delivered to only 59,374 patients comprising 14.7% of all patients (95% CI 14.6-14.8). Most patients receiving dentures required partial (66.4%, 95% CI 66.2-66.6) as opposed to full dentures (p<0.001), while 68.0 % of full dentures (95% CI 67.6-68.4) and 45.2% of partial dentures (95% CI 44.9-45.5) were for the upper jaw (p<0.001). The great majority of partial dentures made had cast metal frameworks (81.4%, 95% CI 81.2-81.6) as opposed to resin bases (p<0.001).

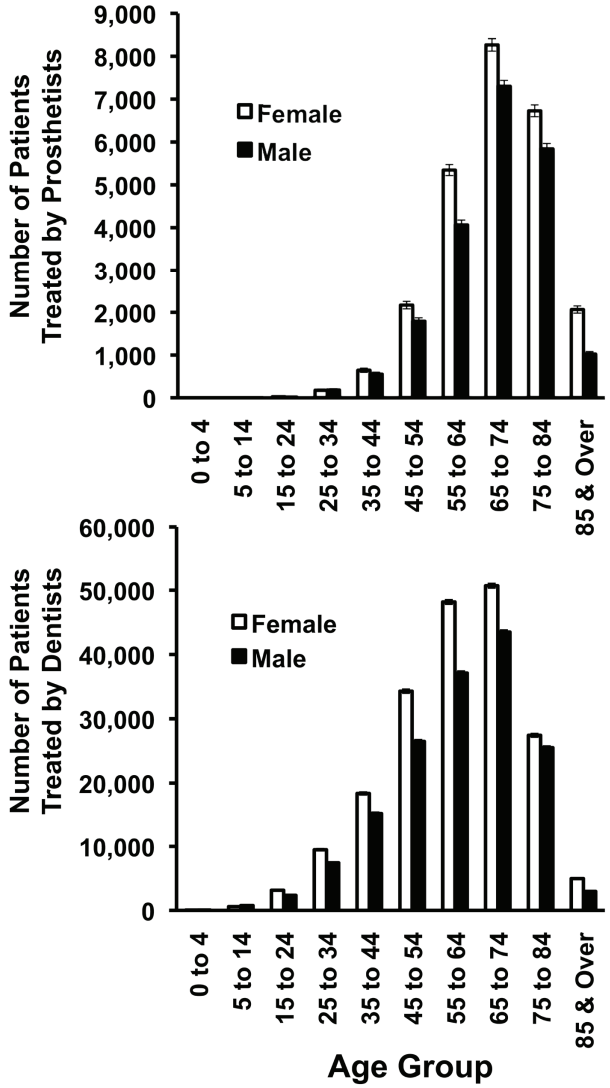

Figure 3: Histograms showing the number of patients treated by prosthetists as well as by general and specialist dental practitioners combined, according to age and sex. There was a slight female preponderance amongst patients in the CDDS, while patients seen by dentists were generally younger than those seen by prosthetists.

The cost of CDDS services

Total expenditure from November 2007 to December 2009 was $731,907,788, with an average expenditure per patient of $1,808. Considering the percentage of total expenditure according to treatment type, denture services (33.88%, CI ± 0.01%) accounted for the largest proportion of cost, followed by crown and bridge (30.32%, CI ± 0.01%), direct restorative (16.33%, CI ± 0.01%), diagnostic (4.94%, CI ± 0.01%), oral surgical (4.76%, CI ± 0.01%), endodontic (3.66%, CI ± 0.01%), preventive (2.80%, CI ± 0.01%), periodontic (2.20%, CI ± 0.01%), general (0.76%, CI ± 0.01%), and orthodontic (0.35%, CI ± 0.01%) services.

Discussion

There are recent reports of patterns of private dental service in Australia,[29-31] but these suffer from the necessary sampling limitations of mail surveys to busy clinicians. The data readily extracted on CDDS services, however, provides a patient sample size and level of detailed information on precise treatments delivered, which appears unprecedented for Australian private dental practice. The current study is only of clinicians and patients participating in the CDDS, so conclusions about patterns of private dental practice can not be readily made regarding wider practice beyond the scheme. Nonetheless, the current study does illustrate the potential power of Medicare records for analysis and practice monitoring, were the CDDS expanded to eventually include all citizens. Some over-estimation of patient numbers is likely in the current study, because many patients presenting to specialist dental clinicians will have also attended general dental surgeries. However, the comparatively small number of diagnostic examinations by specialists suggests the effect of this is negligible. Also, it is impossible to determine from the available statistics what proportion of patients presenting to prosthetists also attended general or specialist dental practitioners, but any 'double counting' effect of this is likely to be small because prosthetist patients comprised only 11% of total patients served. Most preventive services such as fluoride application, scaling teeth and oral hygiene instruction at least partly reverse existing disease and also prevent new lesions from arising. Separately, many operative dental procedures such as restorations and endodontic services simultaneously manage current disease and rehabilitate oral function. To facilitate numerical analysis in the current study,

Table 3: The average number of CDDS services per patient according to type of service delivered, expressed as both services per patient and the relative percentage of services per patient.

	Services per patient	95% CI	Relative % of services	95% CI
Diagnostic services	1.426	1.423 – 1.429	18.802	18.758 – 18.846
Preventive and periodontal services	1.459	1.456 – 1.462	19.237	19.193 – 19.281
Extractions	0.770	0.767 – 0.773	10.162	10.128 – 10.196
General surgical procedures	0.013	0.012 – 0.014	0.175	0.170 – 0.180
Acute pain and dental emergencies	0.120	0.119 – 0.121	1.588	1.574 – 1.602
Chronic pain management	0.059	0.058 – 0060	0.784	0.774 – 0.794
Endodontic services	0.114	0.113 – 0.115	1.510	1.496 – 1.524
Direct restorations (amalgam and adhesive)	2.269	2.265 – 2.273	29.927	29.876 – 29.978
Indirect restorations (crowns and inlays)	0.478	0.476 – 0.480	6.307	6.280 – 6.334
Bridge pontics	0.102	0.101 – 0.103	1.347	1.341 – 1.353
Osseointegrated implants	0.010	0.009 – 0.011	0.137	0.133 – 0.141
Orthodontic services	0.007	0.006 – 0.008	0.095	0.092 – 0.098
Dentures	0.534	0.532 – 0.536	7.045	7.016 – 7.074
Denture repairs	0.219	0.218 – 0.222	2.883	2.864 – 2.902
Total number of individual procedures performed on a per tooth – patient basis	7.582	–	100.000	–

The average CDDS patient received a wide range of routine and advanced dental services consistent with a backlog of untreated dental disease.

we have sought to avoid possible ambiguity by classifying treatment with primary regard to the operative service delivered, rather than with reference to the potentially preventive, disease management or rehabilitative overall clinical objectives. Unfortunately, data available through the Medicare Australia Website permits only determination of the mean number of services per patient, and not the specific proportion of patients who received any given treatment. We are, however, hopeful that the more detailed data needed to evaluate the distribution of services across the patient population may be made available by Medicare at some future time.

It is interesting to observe patterns of uptake of the CDDS across state and territory jurisdictions. Because of government intentions to discontinue CDDS services, there has been no formal advertising to the target population with chronic systemic disease of their eligibility. We speculate that the much higher uptake in NSW relative to other states reflects the combined effect of local CDDS promotion by a NSW-based oral health advocacy group (The Association for the Promotion of Oral Health) via local community groups, as well as active advice from NSW Health to potentially eligible patients to seek CDDS services. There was a marked reduction in new CDDS patients in May and June 2008 across all state and territory jurisdictions, followed by a steep growth in uptake of the scheme. The brief fall in patient numbers seems due to Federal Government announcements and letters to health professionals, enrolled patients and the media that the CDDS was closing, although subsequent Senate action prevented this from happening. It has been argued that the CDDS does not benefit children,[32] however, since comparatively few children suffer chronic systemic disease it is not surprising that the CDDS benefits mostly older people. It should be noted that children with chronic disease are also eligible for CDDS support and the current data indicate use of this scheme in such circumstances.

Data are consistent with a significant burden of untreated dental disease in CDDS patients, but in the absence of data on individual patients, it is impossible to be confident that services delivered were all appropriate to patient need. Indirect restorations including bridges do, at first sight, appear over-represented, and also account for a significant proportion of cost. Regulation of CDDS via a pre-approval process for crown and bridge services would seem reasonable to ensure these services are all clinically appropriate. Importantly, similar regulation has proven effective in dental services supported by the Department of Veteran's Affairs. Despite the high cost of indirect restorative procedures, we see that many teeth would be lost without such service and that any savings would be eroded by the further cost of replacement. In addition, we argue that exclusion of advanced dental service from this Medicare scheme would be inconsistent with the wider established principle that Medicare supports 'all' and not just 'basic' medical services.[21]

While it seems reasonable to question if all expenditure on indirect restorations is justified in terms of potential health outcomes, it is also clear that improving timely access to dental services offers significant health and cost benefits with regard to diabetes, vascular disease, infective endocarditis, aspiration pneumonia, and preventable hospitalisations.[1-16] Also, it is common for those unable to afford private dental service to seek immediate relief from dental abscesses and cellulitis from antibiotics and analgesics prescribed by medical general practitioners. By subsidising attendance for these medical services, Medicare has long provided indirect dental support, and it is particularly unfortunate that antibiotic therapy alone is ineffective because only dental surgical intervention can remove the cause of dental infection (Figure 1). A less wasteful and more effective use of Medicare funds would be to support access to the necessary dental services, at least at the time of acute pain, but preferably before infection spreads to bone and soft tissues. We also have concern that the medical prescription of antibiotics unsupported by dental surgical intervention increases the community load of antibiotic-resistant organisms, hastening

tread into the post-antibiotic era. Without access to more detailed Medicare data, it is not possible to properly evaluate the wider health and cost benefits of the CDDS, but further research in this area seems warranted.

Differences between states regarding the use of indirect restorations and bridges were marked in the current study, and may reflect differences in treatment needs and or cultures. Separately, in light of the longer clinical survival of amalgam as opposed to adhesive restorations,[18,19] the proportionately much greater use of adhesive posterior restorations over amalgam suggests a clinically inappropriate imbalance between the use of these materials. Similarly, the data indicate only very limited use of oral hygiene instruction, which should ideally be an important component of any treatment plan for patients suffering significant dental disease.

An apparently excessive variation in treatment planning has been of concern for some time,[33-36] although it is possible to reduce this with standardised training.[37] The variability in treatment planning seen across state jurisdictions in the current study may reflect the lack of national standards for dental diagnosis and treatment planning, and we suggest that application of such standards to the CDDS would improve outcomes. Clear guidelines regarding the suitability or otherwise of crown and bridgework for given clinical settings would also likely greatly reduce the costs of the CDDS.

The average cost of CDDS services was significantly less than the maximum permitted per patient, so current arrangements appear sufficiently generous to fund services required. With progression of patients from acute through to maintenance service, the per-patient cost of CDDS is expected to reduce. Establishment of pre-approval for some services, together with inclusion in the CDDS of dental therapists and hygienists able to deliver more preventively orientated services at lower cost would help contain expense, were the program expanded to include the entire Australian population.

Competing interests

The second author is the chairman of the Association for the Promotion of Oral Health, which has advocated strongly for retention and improved regulation of the CDDS, as well as for expansion to eventually include the entire Australian population.

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