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Assessing Cardiac Risks in Police Officers

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POLICE OCCUPATIONAL HEALTH REVIEW

Assessing cardiac risks in police officers

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SUMMARY. **It is necessary to assess the cardiovascular risks of police officers in order to address the issue of police officer safety, as well as the issue of public safety. In the past there has been a tendency to determine these risks according to the presence or absence of demonstrable ischaemia. Increased understanding of pathophysiology of coronary vascular disease leads to a different approach to this problem. This modified approach is discussed.**

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The state of a police officer's coronary arteries has obvious implications for the police officer's persona1 health. There are also implications for the public safety. These implications may be divided into two broad categories, the first being the ability to do the job, the second is the risk of sudden incapacitation.

There is a significant physical ability requirement to do police work. [1-3] A police officer may be required to run, to grapple with suspects or to forcibly restrain violent or combative individuals. There may be requirements to lift and carry objects or people. Clearly, the police officer who has active ischaemia on effort is at risk if expected to perform these sorts of tasks. Since the performance of such tasks is often intrinsic to the protection of the public safety, it follows that when the police officer cannot do the physical aspects of the job, the public safety is in jeopardy.

With respect to the risk for sudden incapacitation, the same sorts of arguments apply. A cardiac event that results in incapacitation is usually a manifestation of myocardial ischaemia, with or without infarction.[4] There is good evidence that exercise, particularly sudden onset of intense exertion, may increase the likelihood of a sudden cardiac event.[5] If a sudden cardiac event occurs during the performance of a task critical to the public safety, then the public safety is jeopardised. The easiest example of such a task is pursuit driving. In such circumstances, the sympathetic nervous response gives rise to physiological responses in the cardiovascular system that are very similar to the responses seen in exercise. Sudden incapacitation due to arrythmia might have disastrous consequences during high speed driving. Alternatively, if a police officer were to become suddenly incapacitated while protecting the public in some manner such as crowd control, the public would be unprotected. Similarly, the police officer or a co-worker might be in jeopardy.

For these reasons, it is important that when a police officer develops coronary disease, he or she must be carefully assessed to determine these risks in the context of the job that the police officer is doing. This context is crucially important. The impact of exertional angina on a police administrator's ability to safely do the job is clearly not the same as the impact on a general duty constable on patrol. This discussion is meant to apply to police officers doing operational, front line police work and may not be applicable to police specialists. A cardiac event, in this context, is seen as the clinical result of myocardial ischaemia whether or not the ischaemia is sufficient to produce infarction. When the police officer has undergone myocardial revascularisation the procedure may be interpreted as an attempt to correct ischaemia. For this reason the guidelines for return to work after an event, such as a myocardial infarction, may be applied to the police officer who has undergone myocardial revascularisation in an attempt to correct ischaemia. Physicians who count police officers among their patients, or who provide medical advice to a police force, are frequently tasked with the job of determining fitness to return to work for a police officer who has undergone a cardiac event.

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ASSESSMENT OF RISK FOR ISCHAEMIA

When trying to assess the risks associated with coronary disease it rapidly becomes apparent that there are two sorts of pathophysiology to consider. The process of coronary atherosclerosis with progressive narrowing of the coronary arteries by atheromatous plaque represents one pathophysiological process. This is the process that has traditionally been measured and quantified. There are reliable methods for assessing this process. The second process is that of 'plaque accident'.[6] The risk of plaque accident is harder to assess, but plaque accident is probably an important mechanism of sudden cardiac death[6,7] or incapacitation. It is this latter mechanism that is probably what we used to call 'acute coronary insufficiency'.

With respect to coronary atherosclerosis that is stable or slowly progressing there are straightforward techniques that can be used to assess the adequacy of myocardial perfusion. The resting electrocardiogram is a commonly used tool but it has very poor predictive value.[8] If the cardiogram is very abnormal one can infer the presence of significant coronary disease in most cases, but the opposite is not true. A normal cardiogram is of little use in ruling out coronary disease.[8,9] Exercise, or treadmill, cardiography is somewhat more useful and a positive treadmill test is a reasonably good indicator of significant coronary occlusion. Once again, however, a negative test helps to rule out major obstruction to perfusion at the time of the test.[9] It certainly does not rule out significant coronary disease which has yet to become sufficient to cause myocardial ischaemia. Stress thallium testing has many of the same drawbacks but is probably more sensitive and specific.[10]

Notwithstanding their limitations, however, these tests do have a very important place in the assessment of police officers suspected of suffering coronary disease. The presence of myocardial ischaemia at rest or on exertion is correlated with an increased risk of sudden incapacitation.[4] In addition, the presence of angina or angina1 equivalent symptoms will impact upon the police officers' ability to perform some of the tasks intrinsic to the job.

The Royal Canadian Mounted Police has developed a policy[1] which will restrict members who have evidence of active ischaemia from operational duties requiring heavy physical exertion or performance of tasks critical to the public safety. Evidence of active ischaemia is seen as a positive treadmill test, a positive stress thallium test or angina.

The difficulty that emerges from this approach is the problem of assessing an individual without signs of active ischaemia but with known coronary artery disease. This is the situation faced by the physician who is assessing a police officer who has enjoyed an apparent full recovery from a myocardial infarction, or who has undergone some form of myocardial revascularisation and is now symptom free.

It may be tempting to assume that the post infarct patient who is free of symptoms can be viewed as cured. Similarly, it may be tempting to see the post operative patient as someone in whom the problem has been 'fixed'. Unfortunately, this is rarely the case. Atherosclerosis is a metabolic disease which is widespread and progressive. To view this condition as an issue of 'blocked pipes' is an oversimplification.

ASSESSMENT OF RISK FOR PLAQUE ACCIDENT

While it is true that the final common pathway leading to clinical disease or death is mediated through the obstruction of arteries, it is the process whereby the arteries become obstructed that is the real disease. The coronary arteries may be gradually occluded by atherosclerotic plaque, or suddenly occluded by plaque accident. That the metabolic disease persists is apparent from the fact that most patients who undergo bypass grafting or angioplasty suffer recurrent disease.[9,11-14] In some cases, the disease is manifested by the gradual and progressive obliteration of the vascular lumen, but in other cases it is the sudden occurrence of vascular obstruction, through the mechanism of plaque accident, and thrombosis formation which precipitates clinical disease.

Techniques for the assessment of progressive atherosclerosis have been discussed. We now turn our attention to the problem of trying to predict the likelihood of sudden plaque accident.

For many physicians in practice today, the 'gold standard' for determining the severity of coronary disease has always been the coronary angiogram. It is commonly perceived that high grade obstruction represented severe disease while low grade obstruction represented less severe disease. This model works reasonably well in terms of relatively slow growing lesions restricting myocardial perfusion. Indeed, this model explains exertional ischaemia nicely in terms of an unequal balance between supply and demand. Unfortunately, there is reason to believe that less obstructing lesions may represent more of a risk for plaque accident than some of the higher grade stenoses.[7,15-20] This leaves us in a difficult position when trying to provide advice to a police officer, or a police force, on the basis of an angiographic report. A high grade stenosis is clearly a danger but so, too, may be a

relatively mild obstruction caused by newer and less stable plaque. Indeed, the presence of coronary disease is usually determined, initially, by symptoms. The presence of active ischaemia is confirmed by treadmill or stress thallium, and the angiogram is useful to the surgeon or interventional cardiologist who is contemplating a revascularisation procedure.

In terms of predicting the likelihood of future plaque accident, once it is known that coronary disease exists, the angiogram may be of little benefit to the clinician.

How then, in the absence of objective evidence for high grade coronary obstruction, or for active ischaemia, is the police officer's risk for a future cardiac event estimated? Probably by assessing known risk factors. Indeed, there is some evidence to suggest that the well known risk factors for cardiac events exert their actions by modifying the risks for plaque accident.[15] There is also reason to believe that such risk factor modification may even cause regression of plaque with reduction of the risk for plaque accident.[15,21]

Beginning with the risk factor of hyperlipidaemia, or dyslipidaemia, there is now convincing evidence that plaque size and composition is correlated with the risk of plaque accident and coronary thrombus formation.[21,22] There is also good evidence that serum lipid levels correlate to the size and composition of plaque. More importantly, it appears that by reducing the size and lipid content of an atheromatous plaque, it is possible to reduce the risk of plaque accident and hence the risk of a cardiac event. Indeed, it is encouraging to note that even when obvious regression of plaque is not apparent, plaque stabilization may occur.[21,22]

Hypertension is a well known risk factor for acute cardiac events.[9,23] This risk factor may act by affecting the fibrous cap of the atheromatous plaque. The cap tension, or circumferential wall tension, is given by Laplace's law. The higher the blood pressure, the higher the cap tension.[15,24] Similarly, blood pressure induces both circumferential tension and radial compression of the surrounding vessel wall.[15,24] Other important haemodynamic factors may include plaque fatigue as well as circumferential bending and longitudinal flexion. Simply stated, the greater the pressure, and the greater the haemodynamic turbulence, the greater the chance of cracking the fibrous cap and exposing the thrombogenic fatty core of the plaque to the circulating blood.

Cigarette smoking is a well known risk factor for sudden cardiac events.[9,25,26] It is also known that smoking cessation results in a rapid reduction of cardiac risk.[26] This action of cigarettes may be mediated by vasospasm or by increasing the risk of plaque accident. Certainly, smoking seems to promote lipid oxidization[27] and create more extracellular lipid in the plaques.[28] Smoking also impairs endothelial function.[29] It is probably through some combination of these actions that cigarette smoking increases the risk of plaque accident.

Exercise, in the context of risk for cardiac events, requires careful examination. A small fraction of myocardial infarctions are associated with exercise[5,30,31] and, as previously indicated, physical exertion is an intrinsic part of police work. The mechanism for this effect may be haemodynamic and similar to that discussed for hypertension. On the other hand, there is evidence that regular exercise may retard plaque progression and may provide protection against sudden cardiac events.[30] It is possible that this phenomenon is mediated by the action of exercise on high density lipoprotein cholesterol, but it seems more likely that it is manifest by eliminating the triggering effect of sudden vigorous exertion.[5,30] Therefore, regular exercise is a desirable lifestyle choice while sudden exertion, in a police officer who does not exercise regularly, may be risky.

Diabetes is a serious risk factor for atherosclerotic heart disease.[9] There is evidence to suggest that some, at least, of the microvascular changes associated with diabetes can be controlled, or even reversed, by rigid control of blood sugar.[32] Left ventricular dysfunction where the ejection fraction is less than 50% is associated with a poor prognosis.[33]

The role of beta-blockers and angiotensin converting enzyme (ACE) inhibitors is also worth examining. After a myocardial infarction, the risk of reinfarction can be significantly lowered with beta-blocking medication.[34] Similarly, ACE activity may contribute to the development of coronary disease[35] and ACE inhibition seems to reduce the risk of cardiac events.[35]

It is apparent from this discussion that a number of risk factors for sudden cardiac events may be identified. The only risk factors that appear not to be modifiable are family history, age and, perhaps, left ventricular dysfunction.

ASSESSMENT OF THE INDIVIDUAL POLICE OFFICER

The foregoing provides a framework upon which can be built a decision about an individual police officer. If a police officer has suffered a myocardial infarction then it is clear that the individual is at high risk for plaque accident. Since any meaningful moderation of risk must take time, then it would seem reasonable that we must begin by disallowing the police officer from operational work until there has been time to alter the risk of plaque accident. Studies of plaque regression have examined time frames from just over 1 year[36] to 5 years or

more.[37,38] Until such time as a definitive study is performed which quantifies the rate of regression or stabilization, 1 year would appear to be an appropriate waiting period. This argument can be applied to the police officer who has undergone myocardial revascularisation either by coronary artery bypass grafting or by angioplasty. The risk of reocclusion is greatest during the first year after the procedure.[9] One year would appear to be an appropriate waiting period.

Before allowing a police officer to return to work after a cardiac event we must determine whether there is active myocardial ischaemia, either at rest or on exertion. This may be determined by means of a treadmill stress test or a stress thallium test which must be negative for ischaemia.

Similarly, since ischaemia may sometimes manifest as arrythmia, a 72 hour Holter monitor should be performed which rules out arrhythmias that are associated with sudden incapacitation, in order to ensure that there are no serious ventricular arrhythmias occurring. It must be ensured that risk factors for plaque accident have been reduced to an absolute minimum. It would seem reasonable to require, before certifying a police officer fit for duty after a cardiac event: that blood pressure be reasonably well controlled; that dyslipidaemia be reasonably well controlled (this often requires significant weight loss); that the police officer not smoke; and that the police officer has been certified as safe to exercise by a cardiologist and has undergone a graduated exercise program (usually through a cardiac rehabilitation program, but certainly as directed by a physician) and is now exercising regularly.

Finally, we must assess the two remaining risk factors. Since diabetes of sufficient severity to cause end organ effects is associated with severe small vessel disease and diffuse coronary disease, diabetes in conjunction with coronary disease should preclude return to operational front line policing. Similarly, since left ventricular failure is associated with a dramatic worsening of the prognosis, police officers with clinical evidence of congestive heart failure, or a measured ejection fraction of less than 50%, should not be returned to front line operational policing.

RESIDUAL RISKS

The foregoing analysis is designed to minimise risks rather than to provide a mathematical formula that quantifies risk. The latter has been published for use with airline pilots.[39] Airline pilots, however, do not usually have to perform activities of maximal exercise and are invariably accompanied by copilots. The recommended exclusions outlined above are designed to reduce the risk to the police officer, and to the public, of a sudden cardiac event.

Once the risk has been effectively reduced to the absolute minimum, and all of the foregoing criteria have been met, the knowledge is still present that the police officer does not have perfect coronary arteries and probably still carries a risk for sudden incapacitation which is somewhat higher than a person with perfectly clean coronaries. In order to accept this risk to the public the degree of magnitude of the risk must be determined.

The Pennsylvania State report to the United States [40] provides an illustrative example of how to assess the risk that sudden incapacitation will take place at a time when the police officer is in a situation where the public safety may be affected. In this analysis, the author assumes a population of males between the ages of 35 and 65 and a 5% attack rate for a cardiac event. By a series of probability multiplications, the author comes to the final absolute risk estimate, that a sudden cardiac event would occur during a critical moment, of 0.00875. The risk is interpreted as indicating that the risk of an adverse event at a critical moment is 1 per 25 years per 500 police officers. This is used as an argument against compulsory retirement at the age of 65 for police and fire personnel on the grounds of cardiac risks.

One cannot be sure what the risks would be for a police officer who has suffered a cardiac event and has passed the very stringent requirements outlined above, but it seems reasonable to assume that the order of magnitude of the risk would be similar. Assuming it is double, it must then be decided whether such a very small risk is acceptable or whether all persons with any level of coronary disease should be excluded from employment as public safety officers. This position is seen as extreme. It would seem reasonable, however, to allow a police officer to return to operational police work one year after a cardiac event if the police officer has met all the requirements outlined above and summarized in the Table. Since there remains a significant risk of disease progression, it would further seem reasonable to ensure that these requirements be met on an annual basis in order to continue front line operational policing. It would also be incumbent on the police officer to report any cardiac symptoms and to be assessed regularly by a cardiologist cognizant of the public safety implications.

In summary, a process has been proposed for the determination of fitness for duty in a police officer who has suffered a cardiac event. This process requires careful exclusion of active ischaemia at rest or on exertion. This process also requires the scrupulous control of risks for

Table. Criteria for return to, or continuation of, front line operational police duties after cardiac event or myocardial revascularisation (annual requirement)

1. Police officer wants to return to operational duty.
2. 1 year has elapsed since cardiac event or last cardiac symptom, myocardial revascularisation or any clinical evidence of active cardiac disease.
3. Treadmill test or stress thallium negative.
4. 72-hour Holter does not indicate malignant arrythmia.
5. Ejection fraction greater than 50%. no clinical heart failure.
6. The police officerhoes not suffer diabetes of sufficient severity or duration that microvascular complications are deemed probable.
7. Blood pressure is controlled in the normal range, such control documented.
8. Dys/hyperlipidaemia controlled, such control documented.
9. Non smoker for more than one year
10. Has been certified fit, by a cardiologist, for maximal unmonitored exercise, has completed a physician supervised program of graduated exercise, is now exercising regularly and has passed fitness and/or physical ability test in police forces where this is applicable.

plaque accident as well as the exclusion of known conditions that impart a poor cardiac prognosis. The police officer who returns to duty after a cardiac event must be closely monitored and the continued ability to meet return-to-duty criteria should be assessed annually. The process is summarised in the 'ten commandments' listed in the Table.

References

1. Trottier A, Brown J. Police Health 1994: A Physician's Guide for the Assessment of Police Officers. Canada Communications Group, 1993
2. Farenholtz D, Rhodes E C. Police officer physical abilities test compared to measures of physical fitness. Can J Sport Sci 1992; 17: 228-233
3. Maher P J. Police physical ability tests: can they be valid? Public Personnel; Management Journal 1984; 13: 173-183
4. Davies M J, Thomas A C. Plaque fissuring – the cause of acute myocardial infarction, sudden ischaemic death and crescendo angina. Br Heart J 1985; 53: 363-376
5. Willich S N, Lewis M, Lowe1 H, Arntz H-R, Schubert F, Schroder R. Physical exertion as a trigger for acute myocardial infarction. New England J Med 1993: 329: 1684-1690
6. Fuster V, Badimon L, Badimon J, Chesboro J H. The pathogenesis of coronary artery disease and the acute coronary syndromes. New Engl J Med 1992: 326: 242-250.310-318
7. Shah P K, Forrester J S. The pathophysiology of acute coronary syndromes. Am J Cardiol 1991; 68 (Suppl C): 16C-23C
8. Sox H C, Garber A M, Littenberg B. The resting electrocardiogram as a screening test. Ann Inter Med 1989; 111: 489-502
9. Selwyn A P, Braunwald E. Harrison's Principles of Internal Medicine (13th Ed). 1994; Ch. 203: 1079-1083
10. Come P C, Lee R T, Braunwald E. Harrison's Principles of Internal Medicine (13th Ed). 1994; Ch. 190: 971
11. Dargie H J. Late results following coronary artery bypass grafting. Eur Heart J 1992: 13 (Suppl H); 89-95
12. Weiner-D A, Ryan T J, Parson L, Fisher L D et al. J Am Coll Cardiol 1991; 18: 343-348
13. The V.A. coronary artery bypass surgery cooperative study group: eighteen-year follow up in the vetrans affairs cooperative study of coronary artery bypass surgery for stable angina. Circulation 1992; 86: 121-130
14. Ellis S G, Cowley M J, Disciascio G et al and the multivessel angioplasty prognosis study group. Circulation 1991; 83: 1905-1914
15. Falk E, Shah P K, Fuster V. Coronary plaque disruption. Circulation 1995; 92: 657-671
16. Alderman E L, Corley S D, Fisher S D et al. CASS participating investigators and staff. Five year angiographic follow up of factors associated with progression of coronary artery disease. J Am Coll Cardiol 1993; 22: 1141-1154
17. Nobuyoshi M, Tanakara M, Nosaka H et al. Progression of coronary atherosclerosis: is coronary spasm related to progression? J Am Coll Cardiol 1991; 18: 9044910
18. Giroud D, Li K M, Urban P, Meier B, Rutishauser W. Relation of the site of acute myocardial infarction to the most severe coronary arterial stenoses at prior angiography. Am J Cardiol 1992; 69: 7299732
19. Little W C, Constantinescu M, Applegate R Jet al. Can coronary angiography predict the site of subsequent myocardial infarction in patients with mild to moderate coronary artery disease? Circulation 1988; 78: 1157-l 166
20. Danchin N. Is myocardial revascularisation for tight coronary stenosis always necessary? Lancet 1993; 342: 224-225
21. Gotto A M. Lipid lowering, regression and coronary events. Circulation 1995; 92: 646-656
22. Brown B G, Zhao X-Q, Sacco D E, Albers J J. Lipid lowering and plaque regression: new insights into prevention of plaque disruption and clinical events in coronary disease. Circulation 1993; 87: 1781-1791
23. Stamler J, Stamler R, Neaton J D. Blood pressure systolic and diastolic, and cardiovascular risks. Arch Intern Med 1993; 153: 5988615
24. Lee R T, Kam R D. Vascular mechanics for the cardiologist. J Am Coll Cardiol 1994; 23: 128991295
25. Jonas M A, Oates J A, Ockene J K, Hennekens C H. Statement on smoking, and cardiovascular disease for health care professionals. American Heart Association Medical/Scientific Statement. Circulation 1992; 86: 16641669
26. Deckers J W, Aagema W R P, Huijibrechts I PA M, Erdman R A M, Boersma H, Roelandt J R T C. Ouittine smoking in patients with recently established coronary artery disease reduces mortality by over forty percent. Eur Heart J 1994; 15 (Abstract Suppl): 171
27. Morrow J D, Frei B, Longmire A W et al. Increase in circulating products of lipid oxidation in smokers. New Engl J Med 1995; 332: 1198-1203
28. Wissler R W. The PDAY collaborating investigators. New insights into the pathogenesis of atherosclerosis as revealed by PDAY. Atherosclerosis 1994; 108 (Suppl): S3-S20
29. Celermajer D S, Sorensen K E, Bull C, Robinson J, Deanfield J E. Endothelium dependent dilation in the systemic arteries of asymptomatic subjects relates to coronary risk factors and their interaction. J Am Coll Cardiol 1994: 24: 1468-1474
30. Mittleman M A, Maclure M, Tofler G H, Sherwood J B, Goldberg R J, Muller J E. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. New Engl J Med 1993; 329: 1677-1683
31. Curfman G D. Is exercise beneficial or hazardous to your heart? New Engl J Med 1993; 329: 1730-1731
32. The diabetes control and complications trial research group. The effect of intensive treatment on the development of long term complications of diabetes mellitus. New Enel J Med 1993; 329: 977-986
33. White H D, Norris R M, Brown M A, Brandt P W T, Whitlock R M, Wild C J. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation 1987; 76: 44-51
34. Yusuf S, Peto J, Lewis J, Collins R, Sleight P. Beta blockade

during and after myocardial infarction, an overview of the randomized trials. Prog Cardiovasc Dis 1985; 27: 335-371

35. Lonn E M, Yusuf S, Jha P et al. Emerging role of angiotensin converting enzyme inhibitors in cardiac and vascular protection. Circulation 1994; 90: 205662069

36. Tatami R, Inoue N, Itoh H et al. (for LARS investigators). Regression of coronary atherosclerosis by combined LDL apheresis and lipid lowering drug therapy in patients with familial hypercholesterolemia: a multicentre study. Atherosclerosis 1992; 95: 1-13

37. Brensike J F, Levy R I, Kelsey S F et al. Effects of therapy with cholestyramine on progression of coronary arteriosclerosis: results of the NHLBI type II. Coronary Intervention Study. Circulation 1984; 69: 313-324

38 Buchwald H, Varco R L, Matts J P et al (and the POSCH group). Effect of partial ileal bypass surgery on mortality and morbidity from coronary heart disease in patients with hypercholesterolemia: report on the program on the surgical control of hyperlipidemia (POSCH). New Engl J Med 1990; 323: 946-955

39. Wielgosz A T. Canadian Guidelines for the Assessment of Cardiovascular Fitness in Pilots, Flight Engineers and Air Traffic Controllers 1995. Canada: Civil Aviation Medicine Division 1995

40. Landy Frank J, Schaie K W, Buskirk E R et al. Alternatives to chronological age in determining standards of suitability for public safety jobs. Pennsylvania State University: The Aging and Public Safety Project, January 1992