

# Indocid\*

(indomethacin, MSD Std.)

## Indications

INDOCID\* (indomethacin, MSD Std.) has been found effective in the symptomatic treatment of selected cases of rheumatoid arthritis, ankylosing (rheumatoid) spondylitis, gout, selected cases of severe osteoarthritis including degenerative disease of the hip. INDOCID\* should be used in those cases of severe osteoarthritis which do not respond to treatment with other drugs such as the salicylates. In these conditions it may on occasion replace other commonly used agents such as corticosteroids, salicylates, phenylbutazone-like compounds, and colchicine.

## Dosage Summary For Adults

**In chronic rheumatoid arthritis and ankylosing (rheumatoid) spondylitis:** Start with 25 mg b.i.d. or t.i.d. If response is inadequate, add 25 mg daily each week until an adequate response is obtained or a dosage of 150 to 200 mg is reached. **In acute rheumatoid arthritis and acute flares of chronic rheumatoid arthritis:** Start with 25 mg b.i.d. or t.i.d. If response is inadequate, add 25 mg each day until an adequate response is obtained or until a total daily dose of 150-200 mg is reached. Maintenance corticosteroids can often gradually be reduced 25 to 50 percent and completely discontinued over several weeks or months in some patients.

**In severe osteoarthritis and degenerative disease of the hip:** Start with 25 mg b.i.d. or t.i.d. If response is inadequate, increase the daily dosage by 25 mg at about weekly intervals until an adequate response is obtained or until a dosage of 150 to 200 mg is reached.

**In acute gout:** 50 mg t.i.d. until all signs and symptoms subside.

**INDOCID\* Suppositories:** 100 mg to 200 mg a day. May be administered one at bedtime, and if necessary one the following morning. Also may be used in combined administration with Capsules: 100 mg suppository at bedtime, supplemented the following day by 25 mg capsules as needed up to a total of 150 to 200 mg (capsules and suppositories) of indomethacin.

**Note:** In chronic disorders, it is important to start with low dosage and increase gradually for best results with fewer adverse reactions.

Always give INDOCID\* with food or immediately after meals or with antacid to reduce gastric irritation.

As with all drugs, the lowest possible effective dose should be utilized for each individual patient.

## Contraindications

Active peptic ulcer, gastritis, regional enteritis, ulcerative colitis, diverticulitis and if there is a recurrent history of G.I. lesions. Also contraindicated in patients allergic to a.s.a. or indomethacin. Safety of indomethacin for use in pregnancy or lactation has not been established. Indomethacin suppositories are contraindicated in subjects with a history of recent rectal bleeding.

## SHOULD NOT BE ADMINISTERED TO PEDIATRIC AGE GROUPS.

## Warning

Patients who experience dizziness, lightheadedness, or feelings of detachment on indomethacin should be cautioned against operating motor vehicles, machinery, climbing ladders, etc. Use cautiously in patients with psychiatric disturbances, epilepsy, or parkinsonism.

## Precautions

Indomethacin should be used with caution because of the possible occurrence of gastrointestinal reactions, the incidence of which may be decreased by giving the drug immediately after meals, with food or antacids. The risk of continuing therapy with indomethacin in the face of such symptoms must be weighed against the possible benefits to the individual patient. Indomethacin suppositories should be given with caution to patients with any anal or rectal pathology. Discontinue if G.I. bleeding occurs. Peptic ulcer has been reported. Hemorrhage and perforation have occurred in patients with history of peptic ulcer (see **Contraindications**) or in patients receiving steroids or salicylates concomitantly. In some patients there was no history of peptic ulcer or of other drugs being given. As a result of G.I. bleeding some patients may manifest anemia and, for this reason, appropriate blood determinations are recommended periodically. Headache may occur, usually early in treatment. Discontinue therapy if headache persists despite dosage reduction. In common with other drugs which have anti-inflamma-

tory, analgesic and antipyretic properties, indomethacin possesses the potential of masking the signs and symptoms which ordinarily accompany infectious disease. The physician must be alert to this possibility to avoid undue delay in initiating appropriate treatment of the infection. Indomethacin should be used with caution in patients with existing, but controlled infections. Where therapy is prolonged, ophthalmological examinations are desirable at periodic intervals (see **Eye Reactions**). Since advancing years appear to increase the possibility of adverse reactions, indomethacin should be used with greater care in the elderly. As with any drug, patients should be followed carefully to detect unusual manifestations of drug sensitivity.

## Adverse Reactions

**Central Nervous System:** Commonly seen, headache (usually more severe in morning), dizziness, and lightheadedness. Infrequently observed: mental confusion, syncope, drowsiness, convulsions, coma, depression which may be severe, and other psychic disturbances, such as depersonalization. The severity of these effects may occasionally require cessation of therapy and rarely, admission to hospital. **Gastrointestinal:** include nausea, anorexia, vomiting, epigastric distress, abdominal pain, and diarrhea, which are not uncommon. Single or multiple ulceration of esophagus, stomach, duodenum or small intestine, perforation and hemorrhage have occurred. A few fatalities have been reported. Hemorrhage without obvious ulceration. Increased abdominal pain in patients with ulcerative colitis. Indomethacin has been suspected of precipitating the symptoms of ulcerative colitis or regional ileitis but causal relationship not proven. Rarely reported, intestinal ulceration followed by stenosis and obstruction. Least frequent reactions: ulcerative stomatitis, bleeding from sigmoid colon or diverticuli, perforation of pre-existing sigmoid lesions, e.g., diverticuli or carcinoma. With the use of indomethacin suppositories, pruritus ani, tenesmus, and irritation of the rectal mucosa reported occasionally; rectal bleeding rarely. However, sigmoidoscopic examination in a number of patients did not reveal any significant changes of rectal mucosa. **Hepatic:** Toxic hepatitis and jaundice of uncertain etiology, including severe and fatal cases. **Cardiovascular-Renal:** Infrequently, edema, elevation of blood pressure, and hematuria. **Dermatologic-Hypersensitivity:** Infrequently, pruritus, urticaria, angioneurotic edema, angitis, erythema nodosum, skin rashes, loss of hair, and acute respiratory distress including sudden dyspnea and asthma. **Hematologic Reactions:** Infrequently leukopenia, purpura and thrombocytopenia. Rarely agranulocytosis, hemolytic anemia, but definite relationship to drug not established. Anemia secondary to obvious or occult gastrointestinal bleeding. It is advisable to perform periodic blood counts (including platelet) in patients on long term therapy. If signs or symptoms of above reactions appear, discontinue drug and institute appropriate hematological investigations. **Ear Reactions:** Tinnitus infrequently, and deafness rarely. **Eye Reactions:** Retinal disturbances, including those of the macula, and corneal deposits have been observed. Some of these changes regressed after discontinuation of therapy. Infrequently, blurred vision, orbital and periorbital pain. **Miscellaneous:** Rarely, vaginal bleeding, hyperglycemia, glycosuria and peripheral neuropathy, and epistaxis.

## DETAILED INFORMATION AVAILABLE ON REQUEST.

### How Supplied

Ca 8662—INDOCID\* Capsules 25 mg each, are opaque, blue and white, imprinted with an MSD trademark and potency, and are supplied in bottles of 50 and 500.

Ca 8663—INDOCID\* Capsules 50 mg each, are opaque, blue and white, imprinted with an MSD trademark and potency, and are supplied in bottles of 50 and 250.

Ca 8711—INDOCID\* Suppositories 100 mg each, are white opaque suppositories, supplied in boxes of 12 or 30.

\*Trademark



**MERCK  
SHARP  
& DOHME** CANADA LIMITED

Pointe Claire, Quebec

(MC 941 a)

Can changing the rules and providing adequate face protectors reduce — even eliminate — eye and facial injuries in hockey players?

In 1974, a committee of the Canadian Ophthalmological Society (COS) was formed to study the incidence, types and causes of hockey eye injuries and to devise means of reducing such injuries. A retrospective study of the 1972-73 hockey season revealed eye injuries to 287 players, 20 of whom were legally blinded in one eye (unpublished brief to the COS Council). In 75% of cases the hockey stick was the cause of injury. Subsequently, a prospective study<sup>1</sup> during the 1974-75 hockey season identified a further 253 players with eye injuries, 42 of whom were legally blinded in one eye; the hockey stick accounted for 62% of these injuries.

Presentation of a report on that study (phase I) to the COS annual meeting, in June 1975 was followed by a panel discussion with nonmedical experts. This aroused interest in the news media (see appendix), and publication of the study resulted in further publicity. The COS hockey eye injury committee recommended that all amateur hockey players wear face protectors and that parents be urged to provide adequate protective equipment and proper coaching for their children. Concurrently, Dr. Paul Vinger, in Boston, recommended that all hockey players wear a full-face protective mask affixed to an adequate helmet.<sup>2</sup>

The Canadian Amateur Hockey Association (CAHA) introduced new, high-sticking rules for the 1975-76 season,<sup>3</sup> and the Canadian Standards Association (CSA) technical committee on protective equipment for hockey and lacrosse players intensified efforts to

From the department of ophthalmology, The Hospital for Sick Children, Toronto.

Reprint requests to: Dr. T.J. Pashby, 20 Wynford Drive, Don Mills, Ont. M3C 1J4

## Eye injuries in Canadian hockey. Phase II

T.J. PASHBY, CRCS[C]

introduce standards for face protectors. A standard<sup>4</sup> will be available in print for the 1977-78 hockey season. Officials of the federal Department of Consumer and Corporate Affairs, have proposed new regulations under the Hazardous Products Act whereby a necessary requirement for the sale of masks would be that they meet the CSA standard.

Face protectors have become popular with young players, although they are

will be wearing them. The cost of wire-mesh masks starts at \$10; combination wire and Lexan masks cost from \$18; replacement Lexan portions cost \$5.

To determine the efficacy of face protectors and whether the new hockey rules are having any effect, data of eye injuries reported to the COS during the 1974-75 and 1976-77 seasons (1 Sept. to 15 May) were compared. The new CAHA rules penalizing high-sticking were introduced in time for the season between the study periods.

### Study data

Comparison of eye injuries during the two seasons showed a decline in reported eye injuries from 253 to 90 (Table I); only one type of injury (ruptured globe) did not decrease in incidence. Assessment of visual acuity after injury showed an encouraging reduction in both the number and the incidence of legally blinded eyes, from 37 (1 in 7 injured eyes) to 11 (1 in 8), as shown in Table II. Details of the injury were reported for eight players, seven of whom were wearing face protectors (non-CSA-approved models) at the time (Table III). Legal blindness was reported for no masked players other than goaltenders.

The hockey stick was the main injuring weapon, accounting for 54% of the 1974-75 injuries and 45% of those in 1976-77 (Table IV); this slight decrease was countered by an increased proportion of puck-induced injuries.

The age distribution of players with eye injuries (Table V) demonstrated a significant shift from younger to older age groups. This shift was even more dramatic in relation to players rendered legally blind in one eye, from 33% to 9% in children up to 15 years old.

### Discussion

Almost certainly the reduction in incidence of hockey eye injuries is great-

Changing and enforcement of rules by the Canadian Amateur Hockey Association (CAHA) and provision of adequate face protectors by the Canadian Standards Association (CSA) technical committee have resulted in a marked reduction in the number of eye injuries suffered by amateur hockey players in Canada. This is evident from comparison of the types, severity and causes of eye injuries, their effect on vision and the age distribution of the patients during the 1974-75 and 1976-77 hockey seasons. (Rule changes and the use of face protectors became effective in the intermediate season.) Dental and other facial injuries also can be reduced; in particular, injuries in goaltenders should be greatly reduced, if not eliminated, by a change to the CSA-approved wire-mesh mask. It seems certain that face protectors will become compulsory for all amateur hockey players registered with the CAHA. The cost of the necessary equipment and the inconvenience of wearing it are a small price to pay for the protection afforded.

not mandatory here as they are in the US (Fig. 1); by May 1977, 300 000 had been sold in Canada. Approximately 600 000 players are registered with the CAHA, and it is estimated that there are another 300 000 not registered. Thus it appears that one of every three amateur players now wears a face protector. It is estimated that by October 1977 two-thirds of all players

Table I—Types of reported eye injuries sustained while playing hockey

	1974-75 (253 reports)	1976-77 (90 reports)
Soft-tissue damage	180	65
Orbital fracture	15	6
Corneal abrasion	49	15
Corneal laceration	13	1
Iris damage	75	25
Hyphema	106	34
Traumatic glaucoma	17	5
Lens damage	26	4
Vitreous hemorrhage	25	9
Macular damage	31	12
Choroidal damage	16	3
Retinal damage	19	7
Optic-nerve damage	2	0
Ruptured globe	4	4
Total	576	181

Table II—Visual acuity after eye injury

Visual acuity	1974-75	1976-77
20/20	122 (67%)	49 (67%)
20/40	41 (23%)	21 (29%)
20/60	10 (5%)	1 (1%)
20/100	9 (5%)	2 (3%)
Subtotal	182	73
Legal blindness (unilateral)		
20/200	6	3
<20/200	24	4
No light perception	7	4
Subtotal	37 (15%)	11 (12%)
Total reported/total cases	219/253	84/90

Table III—Injuries sustained by amateur hockey players wearing non-CSA-approved face protectors\* during the 1976-77 season

Player	Mask	Cause of injury	Injury	Visual acuity
Goaltender	Molded	Puck	Cataract	20/200
Goaltender	Molded	Puck	Hyphema	LP
Goaltender	Molded	Puck	Macular edema	20/20
Goaltender	Molded	Puck	Malar fracture	20/20
Skater	Large aperture	Stick	Soft-tissue injury	20/20
Skater	Large aperture	Fell	Broken tooth; cut lip	20/20
Skater	Large aperture	Stick-blade	Facial laceration	20/20

\*One additional goaltender, who was not wearing a mask, sustained hyphema and glaucoma as a result of injury with a stick; his visual acuity is 20/200.

Table IV—Causes of eye injuries

Cause	1974-75		1976-77	
	Total	Unilaterally legally blind	Total	Unilaterally legally blind
Stick	137	20 (54%)	50	5 (45.5%)
Puck	68	13 (35%)	32	5 (45.5%)
Other	15	1 (3%)	6	1 (9%)
Not stated	33	3	2	0
Total reported	253	37 (15%)	90	11 (12%)

Table V—Age distribution of the injured players

Age (yr)	1974-75		1976-77	
	Total	Unilaterally legally blind	Total	Unilaterally legally blind
< 11	28	5	10	0
11-15	76	6	18	1
16-20	56	9	29	5
> 20	69	13	33	5
Not stated	24	4	0	0
Total	253	37	90	11

er than recorded in this study: in view of the COS members' increasing awareness of the problem, there is no doubt that a far greater percentage of injuries was reported for the second season. The small but definite reduction in stick-induced blinding injuries may reflect the introduction and enforcement of new CAHA high-sticking rules during the 1975-76 season. The shift of injury incidence from the younger to the older group of players almost certainly reflects the readier acceptance of face protectors by younger players, either by choice or parental persuasion.

The overall trend to lower incidence of eye injuries in hockey players has been paralleled at The Hospital for Sick Children, Toronto (Table VI), but in some other centres the toll is still high. Ophthalmologists at the University of Montreal report 14 eyes rendered legally blind as a result of retinal detachment during amateur hockey; none of the players was wearing a face protector. However, the authors state that

vision in most of these eyes could have been saved had treatment been instituted sooner, emphasizing the need for intraocular examination of players with extraocular injuries.<sup>5</sup>

Proof of the value of face protectors in preventing facial injury is provided by Dr. David Dickson (personal communication), president of the Brampton Minor Hockey League. This league provides hockey for 2200 boys aged 8 to 20 years, who are required to wear an adequate face protector. All hockey injuries suffered at the Brampton Arena that require hospital attention are directed to Peel Memorial Hospital. Between 15 Sept. 1976 and 5 Jan. 1977

Table VI—Eye injuries treated at The Hospital for Sick Children, Toronto, 1973-75

	Total	Sustained during hockey	
1973	453	52	(11.5%)
1974	574	48	(8.4%)
1975	699	38	(5.4%)

# Apresoline®

(hydralazine hydrochloride)

## antihypertensive

**INDICATIONS:** Various forms of hypertension alone or as an adjunct in: fixed essential hypertension, whether of benign or malignant character; hypertensive toxemias of pregnancy, pre-eclampsia and eclampsia.

**CONTRAINDICATIONS:** Hypersensitivity to hydralazine; coronary artery disease and mitral valvular rheumatic heart disease.

**ADVERSE REACTIONS:** Side effects are usually transitory and reversible with reduction of the dosage. Tachycardia, headache, palpitation, dizziness, weakness, nausea, vomiting and postural hypotension. Less frequently, numbness and tingling of the extremities, flushing, depression, nasal congestion, lachrymation, conjunctivitis, edema, tremors, dyspnea, anginal symptoms, skin rash, drug fever, muscle cramps, giant urticaria and a lupus-like syndrome (arthralgia) in some cases following administration for long periods.

Peripheral neuritis, evidenced by paresthesias, numbness and tingling has been observed. Published evidence suggests an antipyridoxine effect and addition of pyridoxine to the regimen should be implemented if symptoms develop. Blood dyscrasias, consisting of reduction in hemoglobin and red cell count, leukopenia, agranulocytosis and purpura have been rarely reported. If such abnormalities develop, discontinue therapy.

**PRECAUTIONS:** Myocardial stimulation produced by APRESOLINE can cause anginal attacks and ECG changes of myocardial ischemia. The drug has been implicated in the production of myocardial infarction. It must, therefore, be used with caution in patients with suspected coronary artery disease. Also, use cautiously in the presence of advanced renal damage and recent coronary or cerebral ischemia.

**Use in Pregnancy:** APRESOLINE should be used in pregnancy only when, in the opinion of the physician, its use is deemed essential to the welfare of the patient (toxemia of pregnancy).

### DOSAGE

#### Hypertension:

Orally: In general, after initiating therapy gradually increase dosage, adjusting according to individual response. As a single agent, initially 10 mg four times daily, increasing slowly to a maximum practical dosage of 200 mg daily. In combination with other hypotensive agents, lower dosages of APRESOLINE will be appropriate.

When there is urgent need, therapy in the hospitalized patient may be initiated intravenously or intramuscularly. Usual dose is 20-40 mg, repeated as necessary. Certain patients, especially those with marked renal damage, may require a lower dose. Pressure may begin to fall within a few minutes after injection, with an average maximal decrease occurring in 10 to 80 minutes. Most patients can be transferred to oral APRESOLINE within 24 to 48 hours.

#### Toxemia of Pregnancy

- Early toxemia and hypertension of pregnancy; one 10 mg tablet orally, four times daily, slowly increasing the dosage up to 400 mg per day, or until a therapeutic result is obtained.
- Late toxemia and pre-eclampsia: 20 to 40 mg intramuscularly or by slow, direct intravenous injection or infusion. Repeat as necessary.

### SUPPLIED

All forms contain hydralazine hydrochloride. Tablets of 10 mg (yellow scored) Tablets of 25 mg (blue coated) Tablets of 50 mg (pink coated) Ampoules of 1 ml aqueous solution containing 20 mg Bottles of 100 and 500. Ampoule package size of 10. Full information available on request.

**C I B A**

Dorval, Que. H9S 1B1

C-7008

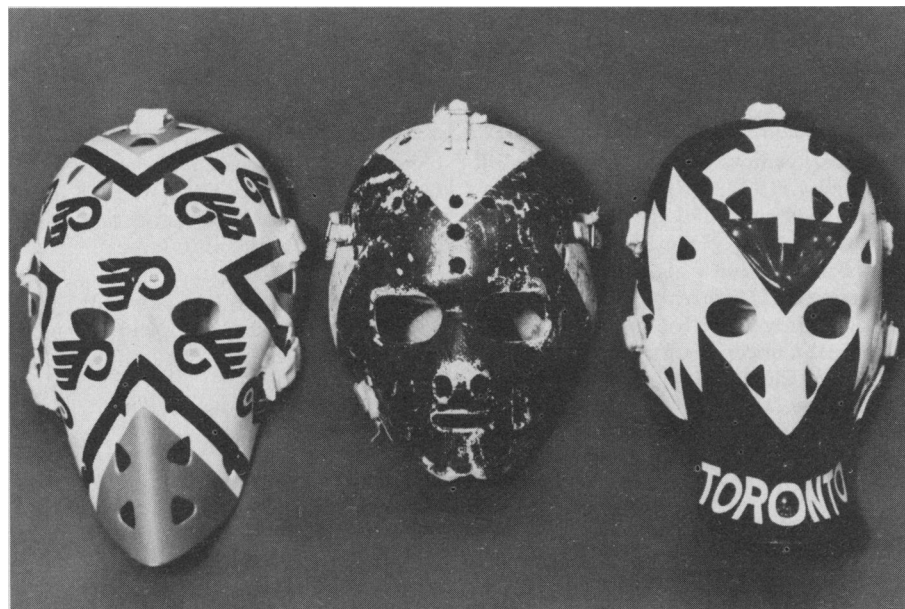


**FIG. 1**—Adequate face protectors now in use in Canada and the US: combination Lexan and wire mesh, left; wire mesh, right.

138 hockey injuries were treated there — including 65 facial injuries to players wearing no facial protection, but none among the 2200 (masked) boys. A further benefit relates to the teeth. Two dental surgeons (A.W.S. Wood and F. Pulfer) who are active members of the CSA technical committee for

protective equipment for hockey and lacrosse players have helped to establish standards for dental protection with the new full-face protectors. This has overcome the inadequacy of some of the old-type tooth protectors. A report by otolaryngologists at the universities of Michigan and Minnesota detailed

the numbers of lacerations, fractures and broken teeth in four age groups;<sup>6</sup> the players were members of three amateur associations and the National Hockey League (NHL). All the injuries were much more numerous in the NHL players — for example, the incidence of broken teeth was 3 per 100 players in the youngest league but 2.2 per player in the NHL.



**FIG. 2**—Professional goaltenders' masks: an older model with large apertures at centre; newer, modified models with smaller eye apertures, left and right.

### Conclusion

Obviously, goaltenders are at the greatest risk of serious eye injury (Table III). The CSA-approved wire-mesh masks provide much better protection for these players, and the professionals seem to be coming around to this opinion (three NHL goaltenders wearing molded masks suffered severe eye injuries during the 1976-77 season). At present, some molded models are being modified by reducing the size of the eye openings (Fig. 2). This is a sound safety measure pending wider acceptance of the greater protection afforded by wire-mesh masks.

If all players are introduced to face protectors at an early age they will become accustomed to them as part of their hockey equipment. It is hoped that protectors that do not meet the

new CSA penetration standard will disappear from the market.

### Acknowledgements

Credit is due to the CAHA for rule changes, to the CSA for its work in evaluating the need for protection and to members of the Canadian Ophthalmological Society who submitted reports on eye injuries they treated. Credit is due also to the manufacturers who modified and produced protectors that the CSA committee considers safe.

The author thanks Miss Helen Haffey, director of medical records, for providing data on hockey injuries treated at The Hospital for Sick Children and medical publications department for help in preparing this report.

### References

1. PASHBY TJ, PASHBY RC, CHISHOLM LDJ, et al: Eye injuries in Canadian hockey. *Can Med Assoc J* 113: 663, 1975
2. VINGER PF: Ocular injuries in hockey. *Arch Ophthalmol* 94: 74, 1976
3. CANADIAN AMATEUR HOCKEY ASSOCIATION: *Hockey Rules 1975-76*, Toronto, Cr Life Ins, 1975

Support for the campaign to make approved face-protectors compulsory for amateur hockey players has come from all segments of the community. Particularly outstanding contributions have been made by the following.

Nonmedical participants in the panel discussion at the 39th annual meeting of the COS in Ottawa in June 1975 were George Armstrong (Toronto Maple Leaf Hockey Club), Robert Nadin (referee-in-chief, CAHA), Thomas Lawson (CAHA), George Lynn (associate professor, school of industrial design, Carleton University) and Nora McCabe (sports writer, *The Globe and Mail*, Toronto).

Special mention is due to the Canadian Broadcasting Corporation for its national network television interview on "Hockey Night in Canada", arranged by Al Stewart and introduced by David Hodge; to Jenny Meldrum (CBC) on "Sports Beat", to Douglas Hall (CKVR, Barrie) on "Day Beat",

to Skyline Cablevision and CJOH-TV, Ottawa, for their interview that has been shown across Canada and to Henry Viney (CFCN, Calgary). Radio exposure was enthusiastically supplied by John Campbell (CKFM, Toronto), Harvey Mock (CFRW, Winnipeg), Bill Brady (CFPL, London) and Jan Hawton (CKLC, Kingston). Several newspaper articles appeared; two of these, by Jim Proudfoot (Toronto *Star* sports editor), created especially widespread interest and greatly stimulated the demand for safety measures. Support came from doctors across the country, including Drs Pierre Labelle, Darryl Green, David Dickson, Frank Buffam, Gordon Harris and B.D. Gain; also Donald Hayes, Ph D, department of kinesiology, University of Waterloo, and Drs Paul Vinger (Boston) and Earl Hoerner (Livingston, New Jersey), on the CSA technical committee on protective equipment for hockey and lacrosse players (chairman, Robert Smith; secretary, Linda LaVecchia).

4. CANADIAN STANDARDS ASSOCIATION: *Preliminary Standard (Z262-2), Eye and tooth protectors for hockey forwards and defencemen*, CSA, Rexdale, Ontario, 1977
5. ANTAKI S, LABELLE P, DUMAS J: Retinal

detachment following hockey injury. *Can Med Assoc J* 117: 245, 1977

6. WILSON K, CRAM B, RONTAL E, et al: Facial injuries in hockey players. *Minn Med* 60: 13, 1977

## OBITUARIES

**Brown**, John Reginald, Toronto; University of London (Eng), 1953; FRCP[C]; preventive medicine; professor of environmental health, preventive medicine and biostatistics, University of Toronto. Died July 30, aged 57; survived by wife Mahnya and son David.

**Calder**, Gordon, London, Ont.; University of Western Ontario, 1930; immunology/allergy and rheumatology. Died July 12, aged 70; survived by wife Glenna and daughter Mary Glenna.

**Campbell**, Sampson Hardie, Windsor, Ont.; McGill University, 1923; general practice. Died June 23; survived by wife Grace, daughter Cynthia, sister Florence and one grandson.

**Cantero**, Antonio, Montreal; McGill University, 1927; FRCS; FRCP[C]; internal medicine and gastroenterology; consultant, Notre Dame Hospital; Canadian Broadcasting Corporation, medical department (Montreal); research professor, University of Montreal and Montreal Cancer Institute; senior member, Canadian Medical Association. Died June 16, aged 74; survived by wife Fernande Kent, daughters Victoria and Michaeli.

**Clow**, Laurence Roberts, Kingston, Ont.; Queen's University, 1945; FRCS[C]; attending staff, obstetrics and gynecology department, Kingston General Hospital; courtesy staff, Hotel Dieu Hospital; associate professor, depart-

ment of obstetrics and gynecology, Queen's University. Died June 18, aged 55; survived by daughters Susan, Ann, Cathy, sons Robin and James.

**Curtin**, Agnes Ann, Rexdale, Ont.; University of Toronto, 1920; public health and general practice. Died May 28.

**Goldstein**, Dorothy A., Calgary; University of Edinburgh, 1958. Died June 22.

**James**, A. Ronald, Port Perry, Ont.; University of London, 1942; MRCS; LRCP; general practice. Died May 16, aged 61; survived by wife Marguerite Jean, daughters Lydia and Diana, sons Stephen, Tim, Adrian and Noel.