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Resident's Corner

The residency year is half completed and a second round of certification examinations by The Royal College of Physicians and Surgeons has passed. There were both practice-eligible and residency-eligible candidates who took that examination, and the results are known to most Canadian emergency physicians. As with our U.S. counterparts, there is a significant failure rate, particularly among the practice-eligible individuals. The residency-trained candidates have had difficulty at approximately the same rate as residents from many other specialty programmes. Having heard about conversations which occurred after the examination process, it would seem we have seen the discipline mature and an appropriate bench mark be established. It is obvious from the discussions with staff physicians and residents across the country that this process has evolved to credential physicians who are competent, safe clinical managers with a good deal of complex depth in the areas of concern to Emergency Medicine. The process is searching for a sophisticated candidate with abilities to manage complex problems and act as a consultant in that capacity. It should hearten us all as residents to see the maturity of the process and the bench mark of the emergency physician become a more clear entity to all of us. This is an area which we should all feel free to discuss and review within our programmes in the light of the first and second set of examinations, keeping in mind the obvious criteria which goes into becoming a good emergency physician.

The examination process amongst many other topics is an excellent reason for as many of us as possible to think of attending the June meeting in Ottawa. I make a strong appeal for as many residents as possible from all the programmes across Canada to get together in Ottawa and sit down to discuss these and many other issues of concern to us. We would like to have a meeting similar to last year, where we sit down with the programme directors, with as many residents as possible, and work out problems which are common to all programmes, and common to our discipline. Some things which come to mind are the efficiency of teaching in the residency programmes, the preparation for examinations, and what we do with the months between July and December of the year we are to take the examinations. Another problem we should think about is the lack of onsite teaching in certain areas of Emergency Medicine in which we require in-depth knowledge, such as EMS. Again, I strongly suggest every effort be made to attend this meeting. I will also be in touch with you about a meeting in the mid-portion of the year, where perhaps we can establish a meaningful agenda for our get-together in June.

Best wishes to all of you for the new year.

Pauline Head MD
Chairman, Residents' Committee
C.A.E.P.

Unmatched group O packed cells: four years experience in a regional trauma unit

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Abstract

Unmatched group O whole blood ("universal donor blood") has been used extensively in the past for resuscitating patients in haemorrhagic shock, with conflicting reports about its safety. The introduction of blood component systems has now made group O unmatched packed red blood cells (G "O" UPRBCs) readily available for emergency use. A retrospective analysis was conducted of 76 Regional Trauma Unit (RTU) patients who received a total of 393 units of G "O" UPRBCs over a 4 year period. This represented 9.6% of all RTU patients who received blood during the period of study. Of 66 patients surviving their initial resuscitation, 8 developed weakly positive direct antiglobulin tests (7 of 8 demonstrated to be negative by 48 hours); six of these patients had received more than eight units of G "O" UPRBCs. There were no clinical complications detected. G "O" UPRBCs appear to be safe and efficient for emergency use in resuscitating the hypovolemic patient.

KEY WORDS: universal blood, Group "O" unmatched blood

Résumé

La grande utilisation par le passé de sang entier non-croisé du groupe O (sang du donneur universel) pour la réanimation des patients en état de choc hémorragique s'est accompagnée de rapports contradictoires quant à sa sécurité. L'avènement des systèmes des composantes sanguines a permis la disponibilité immédiate de culots globulaires non-croisés du group O pour les situations d'urgence. On a effectué une analyse rétrospective de 76 patients admis au Centre Régional de Trauma ayant reçu un total de 393 unités de culots globulaires non-croisés du groupe O au cours d'une période de quatre ans. Ce nombre représentait 9,6% de tous les patients traités au Centre Régional de Trauma ayant reçu du sang pendant la période d'étude. Parmi les 66 patients ayant survécu à la réanimation initiale, huit présentèrent des résultats faiblement positifs à la suite du test de Coombs direct (chez 7 de ces 8 patients, les résultats devinrent négatifs après 48 heures); six d'entre eux avaient reçu plus de huit unités de culots globulaires non-croisés du groupe O. Aucune complication clinique ne fut décelée. Les culots globulaires non-croisés du groupe O semblent être fiables et efficaces pour la réanimation du patient hypovolémique.

MOTS-CLÉS: sang universel, sang non-croisé du groupe O

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Introduction

Ottenberg in 1911 first suggested that persons with blood group "O" be used as "universal donors".[1] Group "O" cells, lacking A and B antigens do not produce hemolytic transfusion reactions secondary to anti-A or anti-B antibodies potentially present in the recipient. "Universal" whole blood given to patients with blood groups A, B or AB does however involve an incompatibility "on the minor side", since the plasma of the donor contains antibodies against red cell antigens which may be present in the recipient.[2] This fear of transfusing plasma with high titres of anti A and anti B antibodies, and the isolated reports of hemolytic transfusion reactions occurring in patients receiving high titre anti A and anti B blood, led to the routine screening of group "O" whole blood for levels of anti A and anti B isohemagglutinins.[3,4] Extensive use of group "O" whole blood in the Korean and Vietnam wars, proved its value in hypovolemic resuscitation, but reports of isolated non-fatal hemolytic reactions continued to appear despite routine isoagglutinin screening.[2,4,5,6,7,8]

The development of blood component systems has made red blood cell concentrates the standard RBC product available.[9] Group "O" packed red blood cells, with 200 mL of antibody containing plasma removed, are readily available for emergent use. This retrospective analysis was undertaken to study the frequency of and indications for Group "O" unmatched packed red blood cell transfusions, to determine if transfused antibody was detected during routine blood bank procedures and to note if clinical complications developed in patients receiving G "O" UPRBCs.

Materials and Methods

The Regional Trauma Unit (RTU) at Sunnybrook Medical Centre currently treats over 400 patients annually, the majority suffering blunt trauma (greater than 95%) and most (85%) referred from other surrounding hospitals. Patients are resuscitated by a trauma team, consisting of a trauma team leader (emergency physician or orthopaedic surgeon) and residents in anaesthesia, general surgery, neurosurgery and orthopaedic surgery, on their arrival.

A temperature controlled Blood Bank refrigerator in the Trauma Resuscitation room contains 8 units of O positive and 4 units of O negative packed red blood cells at all times. The blood stock is maintained and recycled by the Blood Bank staff and all blood is less than 10 days old. An equal amount of group "O" blood is held in the hospital Blood Bank as a reserve supply. The ABO group of all units of blood received from the Red Cross Blood Transfusion Service is repeated for confirmation. Group "O", Rh (d) negative blood is used only for females under the age of 40 years. The units of packed red blood cells are not routinely screened for isoagglutinins or isohemolysins. The decision to use unmatched group "O" packed red blood cells versus unmatched type specific, "immediate spin" direct cross-matched, abbreviated cross-matched or fully cross-matched blood is made by the trauma team leader based on the clinical condition of the patient. Any patient who has received more than 8 units of G "O" UPRBCs before type specific or cross-matched blood is available is maintained on group "O" blood because of the potential disadvantages of switching back to blood of their own group.[2,5,6]

Every blood sample sent to the blood bank for the issuance of blood components is analyzed for blood group, screened for abnormal antibodies and has a direct antiglobulin test (DAT or Direct Coombs Test) performed. The direct antiglobulin test is performed using antihuman rabbit serum (Ortho Registered) which will react with antibody or components of complement present on the red cells and cause agglutination. The DAT is scored from 0 (no aggregates) to 4+ (one solid aggregate) using the Grading System of the American Association of Blood Banks.[10]

The hospital charts, blood bank records and post mortem records when applicable, of all trauma patients receiving G "O" UPRBCs were retrospectively reviewed for a four year period between September 1st, 1979 and August 31st, 1983. For each patient studied the following data were recorded from their hospital and trauma records: age, sex, Injury Severity Score (ISS),[11] time from accident until arrival at the RTU, class of haemorrhage necessitating the use of G "O" UPRBCs (using an adaptation of the American College of Surgeons pulse and blood pressure determinants of Class I to IV haemorrhage),[12,13] blood products received prior to transfer, injuries accounting for the hypovolemic state and any complications caused by the use of G "O" UPRBCs. Data retrieved from the Blood Bank files included blood group, the number of units of G "O" UPRBCs transfused, the total blood products transfused over a 24 hour period, and any abnormalities noted on antibody screens DATs and cross matches performed up to one week following their hospital admission. The autopsy report was reviewed on any patient to die during the first week following admission.

Results

A total of 1224 patients were admitted to the RTU during the study period, 793 (65%) of whom received blood transfusions during the first 24 hours. Seventy-six patients (9.6% of those transfused) received a total of 393 units of G "O" UPRBCs, including 49 males and 27 females. The average age of these 76 patients was 33 years (range 13 to 70 years) and the average ISS was 39 (range 16 to 66). This compared to an average age of 32 years and an average ISS of 30.5 for the entire group of 1224 patients. Nine of the patients were brought directly to the RTU with an average transfer time of 22 minutes (range 12 to 37 minutes). Sixty-seven of the patients were transferred from other hospitals with an average time from injury until arrival at the RTU of 2.4 hours (range 1 to 6½ hours). Thirty-one patients had received blood products at another centre prior to transfer, all of which was either type specific or cross-matched blood.

The blood groups of the remaining 71 patients are outlined in Table I. Forty-three patients had non "O" blood groups. Five patients who arrived with class IV haemorrhages (3 of whom had absent vital signs) received G "O" UPRBCs without blood drawn for blood grouping and died during their initial resuscitation, without blood group identification. The class of hemorrhage defined before G "O" UPRBCs transfusion for the 76 patients is outlined in Table II. Sixty-two patients had suffered class III or class IV haemorrhages.

TABLE I: Blood group of 71 trauma patients receiving group "O" unmatched Packed Cells*

	A	B	O	AB
Rh [D] positive	25	6	25	0
Rh [D] negative	6	3	3	3
	31	9	28	3
Total [%]	(43.7)	(12.7)	(39.4)	(4.2)

*In 5 patients the blood group was never determined.

The quantity of G "O" UPRBCs transfused for each patient is outlined in Table III. Thirty-seven patients received type specific blood following G "O" UPRBCs. The total transfusion of packed cells over a 24 hour period is outlined in Table IV. Ninety-one percent of patients received more than 5 units and 58% of patients received more than 10 units of packed cells. The 7 patients who received less than 5 units of blood over a 24 hour period included the 5 patients with class I haemorrhage and 2 of the patients with class II haemorrhage.

The injuries determined to be responsible for emergency transfusion are outlined in Table V. Four of the

TABLE II: Class of hemorrhage prior to the administration of unmatched group "O" blood

Class of hemorrhage*	Class I (pulse < 100/min, b/p > 110/80)	Class II (pulse 100–120/min, b/p 90–110/60–80)	Class III (pulse 120–140/min, b/p 70–90/50–60)	Class IV (pulse > 140/min, b/p < 70 sys)
Number of patients	5	9	35	27†

*Criteria for class of hemorrhage adapted from the American College of Surgeons Classification.[12,13]
†Includes 3 patients admitted vital signs absent.

patients had suffered penetrating trauma, the remainder sustained blunt injuries. The injuries are divided into "primary injuries" (those injuries determined to be primarily responsible for the haemorrhagic state) and "secondary injuries" (injuries associated with blood loss but not felt to be primarily responsible for the haemorrhagic state). In 9 patients, combined primary injuries (both intra-abdominal injury and pelvic fracture) were present. In two patients, one with a transient airway obstruction and another with a severe head injury, no significant haemorrhagic cause could be found for the abnormal vital signs (both class I "haemorrhages"). Intra- abdominal bleeding and pelvic fractures accounted for the majority of primary injuries (83% of patients).

The patients were divided into three groups, according to their outcome. Group I included 10 patients who died in the Trauma Resuscitation Room, Group II included 10 patients who survived their initial resuscitation but who died less than 24 hours following their admission, and Group III included the remaining 56 patients who survived beyond a 24 hour period.

Three Group I patients arrived without vital signs and the remaining 7 patients arrived in class IV haemorrhage. In 5 of these patients, no blood group was determined (including the 3 patients admitted VSA) and the 5 remaining patients were all group O (4 Rh [D] positive and 1 Rh [D] negative). The initial antibody screens and direct antiglobulin tests were negative on the 5 patients studied: there were no further blood samples available for study on these group I patients. The autopsy reports were all reviewed and in each patient the injuries described accounted for the patient's death.

The 10 Group II patients are outlined in Table VI. Seven patients were non group O (4 group A and 3 group B) and of this group 5 developed weak (range ½ to 1½) positive DATs. In 4 of these 5 patients the DAT was demonstrated to be transiently positive, reverting to negative before death. The fifth patient had no blood sample tested after the weak (½ at 2½ hours) positive DAT. In all patients the initial screen and DAT were negative. The injuries described in the autopsy reports accounted for the patient's death in all cases. There were no clinical complications attributed to hemolytic transfusion reactions in any of these patients.

The 56 remaining patients (Group III) included 27 group A, 7 group B, 2 group AB and 20 group O

TABLE III: Units of G "O" UPRBs transfused in the first 24 hrs

Number of units transfused	1–2 units	3–5 units	6–8 units	>8 units
Number of patients	27	28	12	9

TABLE IV: Total number of units of blood transfused during the first 24 hours

Total number of units	<5 units	5–10 units	>10 units*
Number of patients	7	25	44

*range = 11–64 units.
average = 23.2 units.

TABLE V: Injuries determined to be responsible for unmatched group "O" blood administration

Type of injury	Number of patients with this injury as a primary injury†	Number of patients with this injury as a secondary injury
Intrathoracic	6	9
Intraabdominal (excluding retroperitoneal hematomas associated with pelvic fractures)	50	9
Musculoskeletal:		
–pelvic fractures	22	13
–other closed injuries	0	17
External (includes open #'s)	5	5
Uncertain* (n = 2)		

*One patient with a transient airway obstruction and another with a major head injury, neither of whom had a "significant" hemorrhage.
†In nine patients a combination of intraabdominal injuries and pelvic fractures were felt responsible for the hemorrhagic state.

patients. The records on these patients were all studied for a period of up to 7 days. Only three patients in this group developed positive DATs and these patients are outlined in Table VII. Two of these patients had received only group O blood during the first 24 hours, as they had received more than 8 units of G "O" UPRBCs before type specific blood was available. Patient I had a negative DAT at 24 and 48 hours; no further tests were performed. Patient II had a negative

TABLE VI: Group II patients (surviving < 24 hrs) (n = 10)

Patient	Age	Sex	Blood group	Number of G "O" UPRBCs	Total units of packed cells received	Class of hemorrhage	Direct antiglobulin test results
A	19	M	B neg	55	55	4	DAT positive (½) at 3 hrs DAT negative at 10 hrs
B	20	M	A neg	2	21	4	DAT positive (1½) at 3 hrs DAT negative at 6 hrs
C	18	M	B pos	21	21	3	DAT positive (½) at 2½ hrs (no further results available)
D	21	F	O pos	3	17	3	All DATs negative (n = 2)
E	27	M	O pos	6	24	4	All DATs negative (n = 2)
F	29	F	B neg	3	11	3	All DATs negative (n = 2)
G	20	F	O pos	4	36	4	All DATs negative (n = 3)
H	13	F	A pos	4	43	4	DAT negative on admission (no further results available)
I	56	F	A pos	18	18	4	DAT positive (1) at 2 hrs DAT negative at 6 hrs
J	59	F	B pos	28	28	3	DAT positive (1½) at 1½ hrs DAT negative at 8 hrs

DAT at 24, 48 and 96 hours and was successfully cross-matched on day one following his transfusion of 6 G "O" UPRBCs. Patient III had a negative DAT by 48 hours and, although receiving 17 units of G "O" UPRBCs in the first 24 hours, was successfully cross-matched and received group A blood by his fourth hospital day. None of the remaining 53 patients (including 33 non group O patients) developed any abnormality in their DAT, antibody screen or cross-match during the period of the study. No patient developed any clinical evidence of a hemolytic transfusion reaction.

A total of 9 patients received more than 8 units of G "O" UPRBCs, two of whom were in Group I and did not have their blood group identified or have DATs performed subsequent to receiving this blood and one of whom was blood group O positive. Five of the six remaining patients developed weak, transiently positive DATs. There were a total of 8 patients who were demonstrated to have positive DATs, representing 19% of the 43 non group O patients available for study. None of these patients demonstrated clinical evidence of hemolysis.

Discussion

Group O "universal" whole blood has been used extensively since Ottenberg first recognized its use in 1911. Ottenberg noted that the donor plasma antibodies (anti A and anti B) would be diluted and neutralized and should pose no immediate problem in the recipient.[1] The majority of experience since this time has been with military use.

During the Second World War, Kendrick noted that group O blood was given to many thousands of patients in the Mediterranean and European regions without any reported reactions.[4] Prior to April of 1944, group O whole blood was not screened for titres of anti A or anti B isoagglutinins. At this time, a group A patient who had been transfused with group O whole blood developed a non-fatal hemolytic transfusion reaction. An investigation into this case revealed that the

TABLE VII: Group II patients who developed positive direct antiglobulin tests

Patient	Age	Sex	Blood group	Number of G "O" UPRBs	Total units of packed red cells received in first 24 hrs	Class of hemorrhage	Direct antiglobulin test results
1	21	F	B pos	13	13	3	DAT positive (1½) at 7 hrs DAT negative at 24, 48 and 72 hrs (no further cross-matches performed
2	23	M	A pos	6	14	3	DAT positive (½) at 1½ hrs DAT negative at 4 hrs
3	33	M	A neg	17	17	3	DAT positive (½) at 18 hrs and (½) at 24 hrs DAT negative at 48 hrs (uncomplicated cross-match on Day 4)

plasma in the donor agglutinated the recipient's red cells in a dilution of 1:80,000. It was at this time that the military started to screen all group O whole blood and any units with a saline isoagglutinin titre of greater than 1:250 were used for group O recipients only.[3,4]

During the Korean war, only group O Rh Positive blood was sent into the combat zone, all of which had previously been screened. Over 60,000 transfusions were given and no clinical hemolytic reactions were encountered that were ascribed to group O blood. Four patients were admitted to the renal failure unit with major hemolytic transfusion reactions, each of whom received type specific blood which had been locally procured; at least two of these reactions were secondary to blood grouping errors. In Korea, the use of group O "universal" whole blood with A and B isoagglutinin titres less than 1:200 proved to be a safe experience.[2]

In Vietnam, both universal and type specific blood were used for emergent transfusion.[6] Over 100,000 "universal" donor transfusions were given without a single reported transfusion death.[7] There were 24 hemolytic transfusion reactions, all but one of which was secondary to administrative errors involving the use of group specific blood.[5] The 9 reported transfusion deaths during the Vietnam war all occurred in patients receiving type specific blood.[7,14] The main problem with type specific blood occurred when many multiply-injured patients arrived simultaneously at one centre, as this led to clinical errors involving the administration of "type specific" blood to incorrect patients.[7,8] The Israeli army, for several years, sent only group O blood to combat zones, 50% of which was whole blood and 50% of which was packed red blood cells and none of which was screened. Based on their experience, no significant problems were encountered with the use of this untitred "universal" donor blood.[3]

Antibodies present in the plasma of group O patients can be of differing types and origins. Naturally occurring antibodies are found in the serum of an individual who has never been transfused or pregnant with a foetus carrying the relevant antigen. Virtually all naturally occurring anti A and B antibodies are either wholly or in part IgM, and are called isohemagglutinins. These naturally occurring anti A and anti B antibodies are, with the rare exception, universally present when the corresponding antigen (A or B) is absent, and are responsible for the agglutination reaction used for isoagglutinin screening during the Korean and Vietnam wars. Following "immunization", as may occur with the transfusion of foreign blood group antigens, pregnancy with a foetus bearing a foreign blood group antigen, or by injection of horse serum or the nondialysable part of the influenza virus vaccine, "immune" antibodies may be produced. Immune anti A and anti B are predominantly IgM in A or B subjects, but may be largely IgG in group O subjects. In most blood group systems IgG antibodies will not agglutinate untreated red cells suspended in saline and are therefore described as "incomplete".[15]

The discovery of "dangerous" universal donors followed the hemolytic transfusion reactions described in 1944, and since this time the military experience has involved screening for isoagglutinins with dangerous donors defined as those with titres greater than 1:200 or 1:250.[2,5] However, these isoagglutinins may not be the most important type of antibody in determining the occurrence of a transfusion reaction. The single patient in Vietnam who developed a non-fatal hemolytic transfusion reaction from group O whole blood, was blood group A and received a group O unit with high titre anti-A in error.[5] Subsequent studies on this unit of blood revealed an isoagglutinin titre of 1:256 (considered to barely qualify as high titre) but a titre of incomplete (immune) anti A of 1:32,768.[5] Grove-Rasmussen emphasized that immune antibodies and hemolysin titres may be more important than isoagglutinin titres.[16] Others have demonstrated that titres of isoagglutinins do not always correlate with titres of immune antibodies or isolysins.[17,18] However, in evaluating a screening test of hemolysis, Smith and Monaghan found that when hemolysin titres were tested with red cells, 89% of 106 group O donors would have been rejected. Their recommendation was that group O packed red blood cells would be the product of choice and that routine testing for anti A and anti B abolished.[3]

Despite the sparsity of reports of significant hemolytic reactions, several authors have demonstrated that a selective, clinically insignificant destruction of recipient red cells can occur.[2,5,6] During the Korean war, 25 non group O patients receiving group O whole blood were studied for the presence of foreign antibodies, and 15 patients were demonstrated to have detectable immunoglobulins against A and B antigens. In most of these patients it was possible to demonstrate a selective destruction of recipient red cells but this "minor side reaction" was not found to be clinically harmful. These authors demonstrated that when isoagglutinins persisted in the plasma, it was impossible to obtain a negative cross-match with red cells of the patient's own group.[2] The presence and persistence of isoagglutinins may be related to the quantity of blood transfused, the strength of the antibody (anti B is weaker than anti A), concomitant blood loss by haemorrhage and the patient's capacity to remove or neutralize the antibody.[2,5,6] Anti A is more likely to be incomplete or hemolytic than anti B, and it also commonly devlops to a higher titre in the same patient.[2,6] The isoantigens A and B are not only present on the red blood cells or in the plasma, but are also present on some epithelial cells and on all endothelial cells lining the blood vessels.[19] In certain individuals who are secretors of blood group substances (determined by heredity), the A and B substances are present in other tissues and in the secretions. Crosby et al demonstrated when transfused antibodies were detected after one day, that all of these patients were non-secretors. The secretors seemed better able to neutralize the antibody, decreasing the degree of hemolysis as well as the persistance of the

antibody.[2]

This series represents civilian experience with the use of group O unmatched packed red blood cells. No clinical complications were ascribed to the use of G "O" UPRBCs. Eight patients out of the 43 non group O patients who had direct antiglobulin tests performed following the transfusion of G "O" UPRBCs developed weak, positive DATs. In 7 patients the DAT had reverted to negative by 48 hours' time; one patient died before a follow-up DAT could be performed. This 19% occurance of a positive DAT (in non group O patients) is higher than that reported in a recent review by Sohmer, who found that of 170 non group O patients who received an average 6 units of G "O" UPRBCs, only 5 patients developed serologic evidence of incompatibility and there were no reports of hemolytic transfusion reactions.[20]

Six of our eight patients who developed a transient, weakly positive direct antiglobulin test had received more than 8 units of G "O" UPRBCs and therefore were not switched back to their native blood group. The risk of giving type specific blood following large quantities of group O blood (the number of 4 or 5 units of whole blood was used during the Korean and Vietnam wars), has been well documented, the danger being that type specific blood may now be incompatable with the blood circulating in the patient.[2,5,6] Examining the Korean data, it is evident in one series that 8 of 11 patients who received more than 7 units of whole blood developed detectable isoagglutinin titres and this represented 8 out of the 13 patients with detectable levels. The "dose" of G "O" UPRBCs is obviously important in determining the detection of transfused antibodies, and it is for this reason that our current practise is not to use type specific blood following the administration of 8 or more units of G "O" UPRBCs.

In this series, the indications for emergency transfusion of G "O" UPRBCs were questionable in at least 7 patients (the five patients with class I haemorrhage and the two patients with class II haemorrhage who received less than 5 units of packed cells in the first 24 hours). A recent review by Blumberg and Bove, showed that in 10 out of 56 patients receiving either group O unmatched or type specific blood, the indications were questionable.[21] Despite its apparent safety G "O" UPRBCs should probably be reserved for those hemodynamically unstable patients with class III or IV haemorrhages.

This study has only examined the acute complications of giving unmatched blood and it should be noted that massively transfused patients may become sensitized to many other blood group antigens which may interfere with subsequent cross-matching procedures.[2,15,22] The risk, however, would not be greater with type O blood than with type specific unmatched blood, usually considered the alternative to group O unmatched packed red blood cells. In the acute setting potential complications also exist with individuals sensitized to other red blood cell antigens (Rh Fy(a) K, Jk(a)[15]; however, except for extravascular Rh reactions, an equal risk would exist with type specific blood. In this group of patients, all initial antibody screens were negative and no acute complications related to the "minor" blood group antigens were detected. It should also be noted that group O packed cells used during this study were not screened for anti-A and anti-B titres. The use of packed red blood cells reduces the volume of plasma transfused but the remote possibility of transfusing high titres of anti-A and anti-B in the remaining plasma exists when screening procedures are not performed.

Certain authors have stated a preference for emergent type specific blood; the choice depends on the state of the patient and the time required to obtain type specific blood. The use of type specific blood requires accurate patient identification and increased work for the Blood Bank personnel. Human error continues to be the major cause of untoward reactions to blood transfusions, most often the result of clerical mistakes.[8,23] Meticulous clinical procedures are vital to the use of blood products and in the setting of trauma involves the Red Cross Blood Transfusion Service, the Trauma Team and the Blood Bank staff. All blood received from the Red Cross at our institution is double checked for ABO group and only Group O packed cells are permitted in the Trauma Resuscitation Room blood refrigerator. The Vietnam experience demonstrates the potential complications with the emergent use of unmatched type specific blood. Goldfinger has emphasized the risk of using emergent type specific blood and fells it is more prudent to use group O packed red blood cells until proper typing of the patient can be accomplished under more controlled conditions.[23]

Resuscitation of the hypovolemic patient frequently requires a combination of crystalloid solution and blood component therapy. The decision to use unmatched Group O, unmatched type specific or fully cross-matched blood will depend on the clinical status of each individual patient. The use of group O unmatched packed red blood cells virtually eliminates the danger of fatal hemolytic transfusion reactions.[3,24] The only true "universal" group O donor blood is washed, packed red cells or deglyceralized frozen red cells from which the plasma is removed and replaced with normal saline.[3] In the future erythrocyte substitutes may be available for safe and routine clinical use.[25] Until this time, the emergent use of G "O" UPRBCs remains a clinically safe and efficient means for resuscitation.

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A review of the numbers

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Introduction

In the day to day practice of Emergency Medicine, there are a variety of formulae which must be recalled and applied on a regular basis. These include such things as A-a Gradients, acid-base rules, anion and osmolal gap calculations and many numbers which must be used in isolation and applied in various situations. The purpose of this review is to recall some of these formulae, their derivation and usefulness.

Introduction

Dans sa pratique courante, le médecin d'urgence est appelé à utiliser régulièrement une variété de formules. Celles-ci comprennent les gradients A-a, les règles de l'équilibre acido-basique, les calculs du trou anionique et osmolaire et de nombreux calculs qui doivent être utilisés isolément et appliqués dans plusieurs situations. Cet article a pour but la révision de certaines de ces formules, leur dérivation et leur utilité.

A-a Gradient

The alveolar arterial oxygen gradient (a-a Gradient) is simply the difference between the average partial pressure of oxygen in the alveoli and that in the arterial blood.

#1

A-a Gradient

$$A\text{-}a = P_IO_2 - (PaCO_2 \times 1.2) - PaO_2$$

This value is expressed in millimetres of mercury (mm Hg.) Ideally, when ventilation and perfusion are completely normal there would be little or no difference between these values. Practically speaking, a difference of ten to fifteen mm Hg is accepted as normal[1] in the young healthy patient. Any process which leads to an imbalance between ventilation and perfusion (V/Q mismatch) – i.e. ventilation of underperfused lung or perfusion of unventilated lung – will lead to an increase in the A-a gradient.

The A-a gradient is calculated using the formula in Box #1.[2] In this formula, P_IO_2 is the partial pressure of inspired oxygen at the local altitude, the pCO_2 and pO_2 are measured in the arterial blood, and the value 1.2 is the inverse of the respiratory quotient, 0.8. The value of the P_IO_2 is calculated as:

[Local barometric pressure] =

– [partial pressure of water vapour in inspired air]

× [fraction of O_2 in inspired air (F_IO_2)]

The local barometric pressure varies with altitude and an average value can be used. The partial pressure of water vapor in inspired air at the terminal bronchioles is essentially constant at 47 mm Hg. The F_IO_2 in room air is about 21%. Thus at sea level in room air, the value of P_IO_2 is:

$$(760 - 47) \times 0.21 = 150$$

This value, when calculated for the local barometric pressure, can be memorized and used in every calculation of A-a gradient. The values are then simply substituted into the formula and the calculation made. Thus, again in room air at sea level, the calculation for a patient with a pCO_2 of 40 and a pO_2 of 90 would be:

$$P_IO_2 - (pCO_2 \times 1.2) - paO_2 =$$
$$150 - (40 \times 1.2) - 90 =$$
$$150 - (48) - 90 = 12.$$

A computerized nomogram has been developed by Allingham[3] which allows the rapid determination of A-a gradients simply by finding the intersection of pO_2 and pCO_2 on a graph generated for local altitude.

TABLE I: Allingham's chart

This nomogram also offers some basic assistance in interpretation.

Anion Gap

The Anion Gap is generally expressed as the difference between measured cations (sodium (Na) being the only one of importance) and measured anions (of which chloride, (Cl^-) and (HCO_3^-) are the major components). However, to better understand anion gap, one must recall that the body is maintained in an isoelectric state, i.e. Cations = Anions. If we then include the sodium, chloride and bicarbonate we can express this relationship as follows:

#2

Anion Gap

$$\text{Anion Gap} = Na^+ - (Cl^- + HCO_3^-)$$

Na^+ + unmeasured cation or, alternatively,

$= Cl^- + HCO_3^- +$ Unmeasured anion

This can then be mathematically recorded to obtain:[4]

$Na^+ - (Cl^- + HCO_3) =$ Unmeasured anion – Unmeasured cation

= Anion Gap

Thus, it can be seen that the anion gap is determined by the unmeasured ions and that the calculation we make is valid because of the isoelectric principle. The normal value is taken as 12 ± 4.[4]

With this principle in mind then, it can be seen that the anion gap can be changed by only two mechanisms: Change the concentration of unmeasured cation or

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change in the concentration of unmeasured anions. As well, the possibility of errors in the measurement of Na^+, Cl^-, or HCO_3^- should be kept in mind. Therefore, to increase the gap, one can increase the unmeasured anions or decrease unmeasured cations; to decrease the gap the unmeasured cations can be increased or the unmeasured anions can be decreased.

An increase in the anion gap is rarely due to a decrease in unmeasured cations, as significant depressions of potassium (K), calcium (Ca), or magnesium (Mg) are unusual, and large enough depressions to give a significant change in the anion gap would be incompatible with life. (If all three of these cations were decreased simultaneously, one could see an increased anion gap). Therefore, an increased anion gap is commonly due to an increased concentration of unmeasured anions. The most frequently seen causes of this can be seen in Box #3.

#3

Causes of Elevated Anion Gap

1. Increased Organic Acids
   - —lactic acidosis
   - —ketoacidosis
2. Increased Anions of Inorganic Acids
   - —sulfate
   - —Phosphate
     - —eg. – in uremic acidosis
3. Ingestions
   - —ASA
   - —Ethylene glycol
   - —Methanol
   - —Paraldehyde
   - —Occasionally with penicillin, carbenicillin

A decreased anion gap may be due to an increase in the normal body cations (K, Mg, Ca) or abnormal cations (Li, gammaglobulins), but is most commonly caused by a decrease in anions in the form of hypoalbuminemia.

#4

Causes of Decreased Anion Gap

1. Increased Cations
   - —hyperkalemia
   - —hypercalcemia
   - —hypermagnesemia
   - —elevated gammaglobulins
   - —elevated serum lithium
2. Decreased Anions
   - —hypoalbuminemia

Acid Base Rules

It is not within the scope of this paper to discuss fully the derivations of each of the important acid-base rules. However, one should recall that the major physiologic buffer system is the bicarbonate – carbonic acid pair, and that the pH is determined according to the Henderson – Hasselbach equation:

$$pH = pK + \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

The concentration of H_2CO_3 is proportional to the solubility constant of CO_2 times the pCO_2. This formula

NOMOGRAM FOR ALVEOLAR-ARTERIAL OXYGEN GRADIENTS, DENVER

PO2 \ PCO2	20	22	24	26	28	30	32	34	36	38	40	42	44	46	48	50	52	54	56	58	60
40.	57	55	53	50	48	45	43	41	38	36	33	31	29	26	24	21	19	17	14	12	9
42.	55	53	51	48	46	43	41	39	36	34	31	29	27	24	22	19	17	15	12	10	7
44.	53	51	49	46	44	41	39	37	34	32	29	27	25	22	D 20	17	15	13	10	8	5
46.	51	49	47	44	42	39	37	35	32	30	27	24	23	20	16	15	13	11	8	6	3
48.	49	47	45	42	40	37	35	33	30	28	25	23	21	18	16	13	11	9	6	4	1
50.	47	45	43	40	38	G 35	33	31	28	26	23	21	19	16	14	11	C 9	7	4	2	0
52.	45	43	41	38	36	33	31	29	26	24	21	19	17	14	12	9	7	5	2	0	0
54.	43	41	39	36	34	31	29	27	24	22	B 19	17	15	12	10	7	5	3	0	0	0
56.	41	39	37	34	32	29	27	25	22	20	17	15	13	10	8	5	3	1	0	0	0
58.	39	37	35	32	30	27	25	23	20	18	15	13	11	8	6	3	1	0	0	0	0
60.	37	35	33	30	28	25	23	21	18	16	13	11	9	6	4	1	0	0	0	0	0
62.	35	33	31	28	26	23	21	19	16	14	11	9	7	4	2	0	0	0	0	0	0
64.	33	31	29	26	24	21	19	17	14	12	9	7	5	2	0	0	0	0	0	0	0
66.	31	29	27	24	22	19	17	15	12	10	A 7	5	3	0	0	0	0	0	0	0	0
68.	29	27	25	F 22	20	17	15	13	10	8	5	1	1	0	0	0	0	0	0	0	0
70.	27	25	23	20	18	15	13	11	8	6	1	1	0	0	0	0	0	H 0	0	0	0
72.	25	23	21	18	16	13	11	9	6	4	1	0	0	0	0	0	0	0	0	0	0
74.	23	21	19	16	14	11	9	7	4	2	0	0	0	0	0	0	0	0	0	0	0
76.	21	19	17	14	12	9	7	5	2	0	0	0	0	0	0	0	0	0	0	0	0
78.	19	17	15	12	10	F 7	5	3	0	0	0	0	0	0	0	0	0	0	0	0	0
80.	17	15	13	10	8	5	3	1	0	0	0	0	0	0	0	0	0	0	0	0	0
82.	15	13	11	8	6	3	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0

'NORMAL' GRADIENTS:
NORMAL VENTILATION
A Age less than 45
B Age 45+, Chronic Lung Disease, Some Heavy Smokers
'NORMAL' GRADIENTS:
ALVEOLAR HYPOVENTILATION
C Age less than 45
D Age 45+, Chronic Lung Disease, Some Heavy Smokers
'NORMAL' GRADIENTS:
ALVEOLAR HYPERVENTILATION
E Age less than 45
F Age 45+, Chronic Lung Disease, Some Heavy Smokers
ABNORMAL GRADIENTS: SEVERE V/Q DISTURBANCE OR SHUNT
G All Persons
F
B Age less than 45 and no chronic
D lung problems
H UNLIKELY RESULT. RECHECK DATA

FORMULA – PIO2 – (PCO2 × 1.2) – PO2
PIO2 CALCULATED THIS ALTITUDE IS 121.4
ASSUMES CONSTANT BAROMETRIC PRESSURE OF 625
UNIVERSITY OF CALGARY, FACULTY OF MEDICINE
Revised from[3], used with permission

tells us that the pH is controlled by the kidneys (control of the concentration of HCO_3^-, the numerator) and the lungs (control of pCO_2, the denominator), and, therefore, that if one of these is not functioning properly, the other must compensate – i.e. in respiratory disturbances there will be metabolic compensation and in metabolic disturbances there will be respiratory compensation. It must also be remembered that while respiratory compensation may be seen very rapidly, metabolic compensation may take hours to days to develop. The rules are all derived from these basic underlying principles. These rules are found in Box #5 along with some other helpful interpretive data.

#5

"Acid Base Rules"

Δ pH	0.15 –	Δ HCO_2	10 mmol/1
Δ pH	0.1 –	Δ K^+	0.5 mmol/1
Δ pCO_2	10 torr –	Δ pH	0.08 in opposite direction
↑ pCO_2	10 –	↑ HCO_3	1 mmol/1 acutely
Δ pCO_2	10 –	Δ HCO_3	3 mmol/1 chronic
↓ pH	0.1 –	↓ K^+	0.5

Complete tables of the differential diagnosis of acid-base disturbnces can be found in many sources.[6]

Osmolal Gap

The Osmolal gap is defined as the difference between the measured osmolality and the calculated osmolality. Serum osmolality, in SI units, is calculated as 2Na + Glucose + BUN (blood urea nitrogen). The normal value is taken as 280 ± 15 (mosm.) milliosmols. The difference between this and the measured osmolality should not be greater than 10 mosm. This calculation (seen also in Box #6)

#6

Osmolal Gap

(2Na + Glucose + BUN) – measured osmolality

is useful in determining if there is an abnormal osmotically active particle present in the serum (see Box #7).

#7

Causes of Increase of Osmolal Gap

Ethanol
Methanol
Ethylene Glycol
Isopopyl Alcohol
Mannitol
Paraldelyde

These compounds are most commonly ingested substances or medications as can be seen from the list.

Bicarbonate Dose

Another calculation which may be useful is the estimation of the starting dose of bicarbonate. This can be calculated in several ways, but each method recognizes the potential problems of too rapid correction or over correction by calculating the estimated body deficit and dividing it by two to give the starting dose. These formulae can be found in Box #8.

#8

Bicarb Therapy

(mmol) millimol of Bicarb required

$$= 25 - [HCO_3^-] \text{ measured} \times (0.5 \times \text{wt. in kg.})$$

or

$$\text{mmol Bicarb required} = (\tfrac{1}{2}\, PCO_2 - [HCO_3]) \times (0.5 \times \text{wt.})$$

or

$$NaHCO_3 \text{ dose} = \frac{0.2 \times \text{body wt. (kg)} \times \text{base deficit}}{2}$$

Summary

The practice of Emergency Medicine today increasingly necessitates a variety of rapid arithmetic calculations.

The introduction of new standard units (S.I.) has complicated many of the formulae involved. This discussion has reviewed a number of the more commonly used formulae, their derivation and their implications.

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Non-ballistic penetrating craniocerebral injury: a case report and literature review

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Abstract

Presented is a case of a young male construction worker who suffered a significant non-ballistic penetrating craniocerebral injury with minimal clinical findings and no appreciable sequelae. The relevant available literature is briefly reviewed with emphasis on emergency management.

KEY WORDS: Non-missile (ballistic) penetrating
Craniocerebral injury

Résumé

On présente le cas d'un jeune travailleur de la construction ayant subi un traumatisme crânio-cérébral pénétrant non-balistique important dont les symptômes cliniques et les séquelles sont disproportionnellement minimes. On passe brièvement en revue la littérature pertinente disponible en soulignant le traitement d'urgence.

MOT-CLÉS: Traumatisme crânio-cérébral pénétrant non-balistique

Introduction

With the advent, popularization and availability of firearms, non ballistic penetrating craniocerebral injuries have become relatively rare. Nonetheless, the Emergency Physician must have a basic understanding of the pathophysiology and principles of management when confronted with the entity. The patient may present with a wide spectrum of clinical findings. This case represents one end of that spectrum.

Case Presentation:

F.D. A 33 year old construction worker was brought to our Emergency Department by ambulance. He complained of a small puncture wound sustained at the construction site a few minutes earlier when a small nail struck his left parieto-occipital skull (Figure 1). The gravity of the injury was better appreciated when a co-worker brought in the patient's helmet which had been removed at the construction site. The safety

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Fig. 1 – *Patient's scalp*

helmet was impaled by a 14 cm spike which had been dropped from the 17th floor (Figure 2). The spike had penetrated the helmet a full 5 cm (Figure 3). There had been no appreciable loss of consciousness nor did our patient have any complaints aside from the local trauma. He was a healthy individual with no past medical history, medications or allergies.

Physical examination, when he was seen at 0915 hours, revealed an alert well looking young male in no distress and hemodynamically stable. His vital signs were BP 140/90, HR 84, RR 16. Examination of the scalp revealed a 1 cm puncture wound and hematoma over the left parieto-occipital scalp. There was no active bleeding. Neurological exam demonstrated a patient that was alert, coherent and orientated in three spheres. Speech was normal. Cranial nerves II to XII were intact. In particular, pupils were 3 mm; equal and reactive. Extraoccular movements, fundoscopic examination and visual fields by confrontation were normal. Tympanic membranes as well as the mouth, tongue and oropharynx were normal. The neck was supple with full range of motion and the C-spine was non tender. The motor system was normal with respect to tone and power. Co-ordination was intact as was sensation to light touch and pinprick. Gait was normal and reflexes were bilaterally brisk and symmetrical with down-

Fig. 2 – *Impaling object*

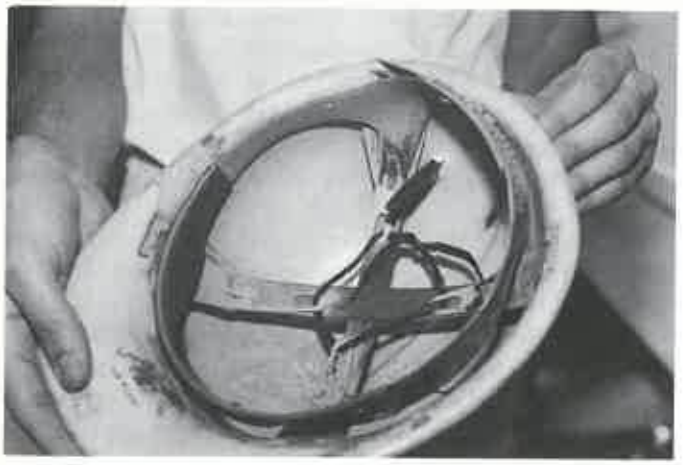


Fig. 3 – *Penetration of spike*

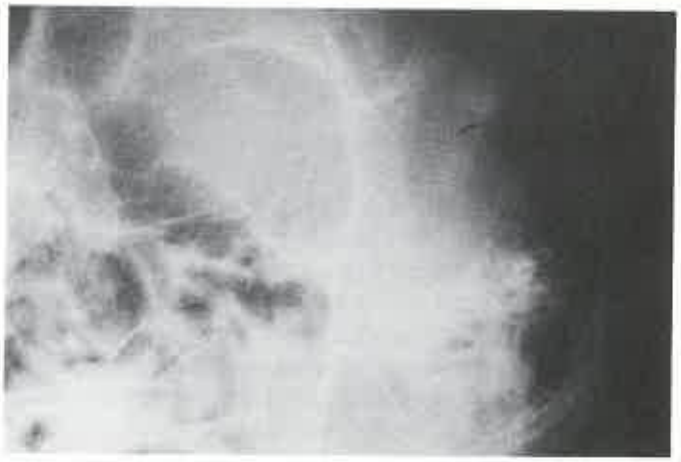


Fig. 5 – *X-ray oblique skull*

going toes. The rest of his examination was equally unremarkable.

Following his physical assessment he was taken for plain radiographic studies at 1000 hours. These demonstrated a depressed left parieto-occipital fracture (Figure 4 and 5). At 1030 hours a CT scan was performed which revealed a well circumscribed hematoma in the left parietal region associated with the penetrating injury to the vault (Figure 6).

At 1100 hours the patient was given 1 cc Tetanus Toxoid, 2 grams of cefazolin and a neurosurgical consultation was requested. At 1400 hours the patient was taken to the operating room. An S-shaped scalp incision incorporating the puncture wound was made and this revealed a galeal hematoma and rectangular puncture of the bony skull. A craniectomy was performed revealing a dural tear which was incised in a cruciate manner uncovering a mass of necrotic brain and intracerebral hematoma. These were evacuated under suction and bleeding cortical vessels were cauterized to achieve hemostasis. The area was irrigated with bacitracin, the dura repaired and the wound closed. cefazolin was continued intra and post-operatively and DILANTIN® was started.

Post-operatively a right homonymous hemianopsia was demonstrated on neurological examination. This was transient, and resolved completely by the third post operative day. Our patient was discharged home a week post-injury on oral KEFLEX® and DILANTIN®; neurologically intact.

Discussion

The advent of firearms has made non-ballistic penetrating injuries to the skull and brain a relative rarity.[1,2,3] This phenomenon is clearly reflected by the paucity of literature on the subject. The majority of cases recently reported come from South Africa where the Third World component of the population in conjunction with strict gun control laws have made stab wounds to the skull a rather common occurrence.[3] The bulk of the remaining literature involves accidental penetration in children,[2] isolated case reports or antedates the popularization of firearms.[4,5]

De Villiers[1] reviewed his experience over a twenty year period (1953–1973) and collected 95 cases of penetrating craniocerebral injuries. Typically, the victim is a young male and is more than twice as likely to be injured on the left (74%) rather than the right side (29%) owing to the preponderance of right handed assailants. The offending object in 75 of the 95 cases was a knife. This is consistent with subsequent observations.[3] Pencils and other wooden objects are rarely

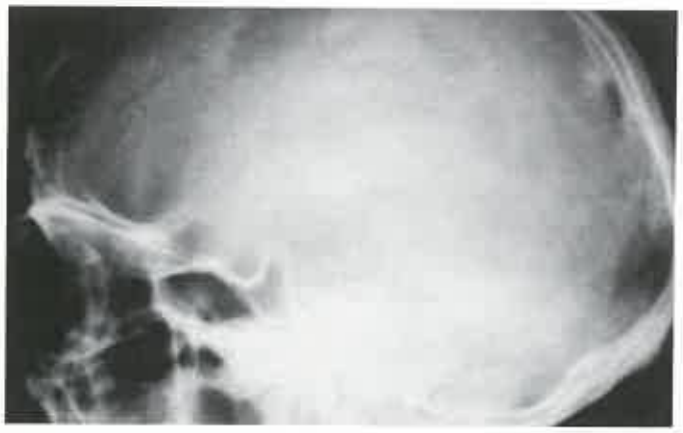


Fig. 4 – *X-ray lateral skull parieto-occipital slot fracture*

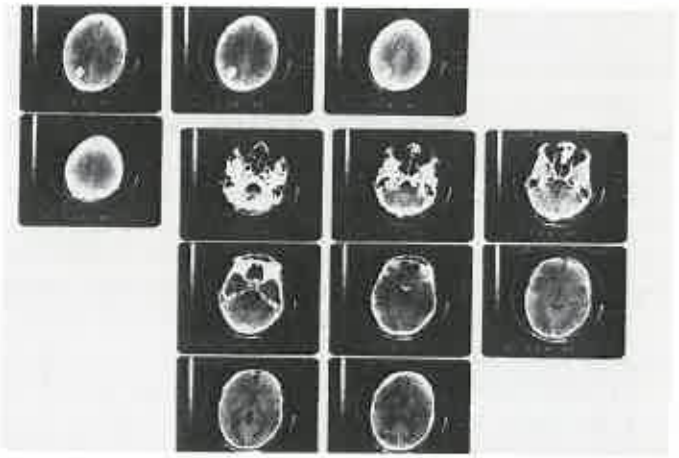


Fig. 6 – *CT Scan – intracerebral hematoma*

responsible for penetrating craniocerebral injuries. The significance of these organic materials is nonetheless important since they represent a high proportion of retained (missed) intracranial foreign bodies,[1,6,7] higher incidence of infective complications[7] and negative roentgenographic studies.[6,7]

The organic penetrating foreign bodies have a higher incidence of suppurative complications[7,8,9] and are often not appreciated on plain film examinations of the skull.[7] On C.T. scan, wood has a very low attenuation value (less than water) and may be misinterpreted as an intracerebral pneumocoele associated with penetration.[7]

Patients with penetrating craniocerebral injuries can be divided into three broad groups based on their clinical presentation. The first group includes those injuries which produce a rapidly fatal outcome and usually involve a massive intracranial hemorrhage, rapidly expanding intracranial hematoma and transtentorial herniation. In De Villier's series, 12 of 16 deaths fell into this group. Two others were late deaths due to infection and the other two involved transection of major vessels in addition to medullary or pontine structures. The second group involves patients with major neurological deficits and variable deterioration due to expanding intracerebral hematoma. Hemiplegia often associated with dysphasia due to the preponderance of left sided lesions is a common feature.

The last group demonstrate transient or minimal neurological abnormalities without focality and no demonstrable intracranial hemorrhage. These tend to do well without surgical intervention.

The initial level of consciousness appears to be the best single predictive factor of outcome, with no reported cases of survivors among those presenting in coma.[3] Mortality correlates well with the extent of vascular injury and bleeding. Kieck[3] reviewed 109 transcranial stab wounds, 74 of which had angiography (56 transcranial; 18 transorbital) and found 26 patients with vascular lesions. This represents 20% of the whole group and 33% of those angiogrammed. Five patients developed carotid-cavernous fistulae, four of them 48 to 72 hours after injury, and in one patient, upon removal of the knife blade. In addition, eleven patients developed aneurysms. Of these, eight were at the base of the brain, and involved a delay in development, multiplicity of aneurysms and a high mortality associated with rupture.

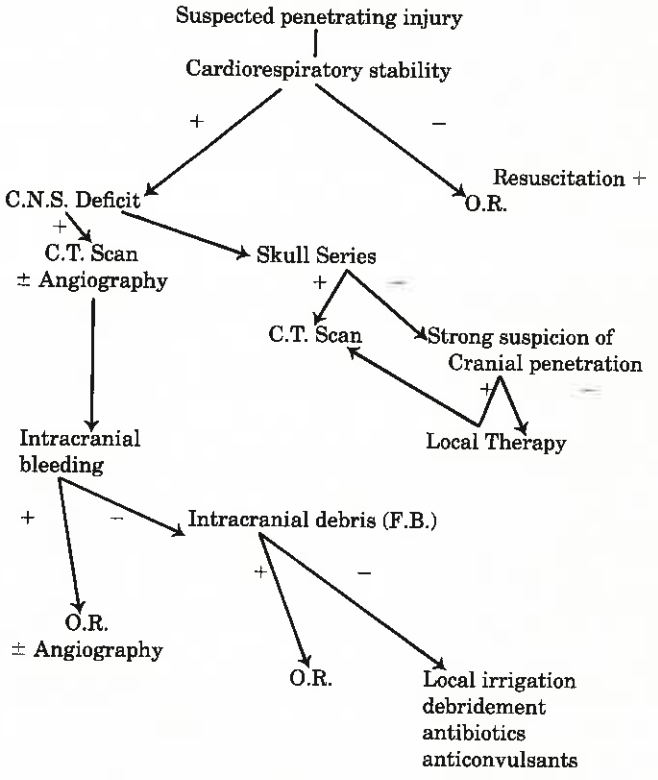
Because of the uniformly fatal outcome of those patients presenting in coma, De Villier has questioned the value of surgical intervention. Despite the availability of CT scanning, this concern has been echoed by others in the more recent literature,[3] and heroic neurosurgical intervention remains controversial with the important focus for the Emergency Physician being that of cardiopulmonary stabilization and early neurosurgical consultation.

It should be emphasized that the entrance wound (as was the case with our patient) may be trivial and misleading. The presence of a "slot fracture" (slot like defect in the skull) is diagnostic of a stab wound but no radiographic abnormality may be detected on standard skull views even in cases of known penetration of the skull.[1,3] The vascular damage may be on the opposite side if the offending object has crossed the midline.[3] There is a high incidence of delayed vascular lesions; the majority of which occur at the base of the brain in contrast to a more peripheral location following blunt trauma.[3]

In many cases, the impaling object has been removed. If, however, it has remained in situ; temptation to remove it should be resisted.[8] While in place, the offending object may be exerting a tamponade effect on any transected vascular structure.[3] In addition, its removal may injure nearby vascular structures narrowly missed upon entry with disastrous consequences.[1] Leaving the foreign object in place may also allow CT scan or angiography to demonstrate a transgression of the midline which may have therapeutic implications.

Penetrating craniocerebral injuries in children warrant some discussion. The types of instrument causing injury tend to show a wider variation in children, and there is a more even distribution of right and left sided injuries.[1] Due to the thinness of the incompletely ossified pediatric skull and the fragility of the orbital roof, seemingly minor injuries may be associated with violation of the dura and brain.[2] Penetrating orbitocranial injuries are also associated with a higher morbidity and mortality than penetrating injuries elsewhere on the calvarium.[10,11] There is a number of reports in the literature of child abuse and infanticide using sewing needles and other sharp objects introduced though fontanelles and orbits.[12,13] It is imperative to maintain a high index of suspicion when presented

DIAGRAM I: Management protocol for suspected penetrating craniocerebral injuries.



with a child (particularly a small infant) with a potential penetrating craniocerebral injury. Because of the widely discrepant variations in presentation of these injuries, some flexibility in management is mandated. A suggested management protocol is outlined in diagram I.

Summary

There are a few management principles which bear further emphasis. Attention to and definitive management of the airway and cardiovascular system should precede specific management of the penetrating cranial injury. The nature of the entry/scalp wound does not correlate with intracranial pathology and routine plain skull films may be negative despite penetration. This mandates oblique and other less conventional views when penetration of the skull is strongly suspected.

A high index of suspicion is key to detecting injuries in patients with minimal clinical findings. As was the case with our patient, the mechanism of injury and the nature of offending object should alert the physician to the potential penetration. The age of the patient is also an important consideration.

Wood and other organic materials may be difficult to detect radiographically, have a higher incidence of retained/missed fragments and infective complications.[7,8,9] Tetanus Toxoid and early antibiotic coverage should be considered in all penetrating injuries and especially in those of organic objects.[2,9]

The offending object, if still in place, should only be removed under operative control where the potential ensuing consequences can be definitively managed.

The major cause of death from these injuries is intracranial hemorrhage and those patients presenting in coma have a uniformly fatal prognosis.[1,3] Aggressive neurosurgical intervention in these cases is controversial.

Angiography plays a key role in these injuries despite the advent of C.T. scanning and should be considered early in those patients with suspected vascular damage[1,3] as well as 2 weeks post-injury to identify latent vascular injuries.[3]

Craniotomy is the specific method of management with a "look and see" approach.[1,2] Burr holes and Mannitol have been singularly disappointing in temporizing when intracranial hemorrhage is present.[1] Neurosurgical consultation should be obtained as soon as penetration of the cranium is suspected.

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Editorial

Whither P.A.S.G.

Peter Lane MD

The shock pants debate is again heating up, and it illustrates both the strengths and the weaknesses of Emergency Medicine research to date. Until quite recently, the vast majority of reports in the literature consisted of either case reports or retrospective series. These began with the all-too-familiar works of Dr. G. Crile,[1] through the early reports from Viet Nam,[2] and eventually to civilian Emergency Department and prehospital[3,4] uses of the garment. Most of these reports documented anecdotal beneficial effects when used in cases of hypovolemic shock.[4,5] It was on these early reports that many Emergency Physicians based their decisions to start using the garment clinically.

More recently, a number of papers have looked at the garment experimentally, either in animal shock models or in normovolemic human volunteers. Some of these have evaluated the mechanism of action,[6,7,8,9] while others have looked at some of the side effects.[10,11,12] An excellent study by Chisolm and Clark[13] evaluated tissue compartment pressures in normovolemic volunteers at different inflation pressures. We have recently reported a case of bilateral compartment syndromes necessitating amputations after use of the garment.[14] This is truly a disastrous complication, and gives us reason to reevaluate our use of the device.

Holcroft et al[15] have shown, in a rather elegant study using baboons, that the shock pants both impede and augment venous return, in part because of distortion of the retroperitoneal and intraabdominal veins. Again, this is causing us to rethink both the possible mechanisms of action as well as the contraindications to use of the garment.

Perhaps the most disconcerting yet significant report recently is that of Mackersie et al.[16] Until this report, there had been no controlled trial of the use of the garment in any clinical setting. The study evaluated results in two groups of trauma patients, one with and one without the pneumatic antishock garment (PASG) applied in the prehospital setting. While some may quibble that the study groups were not strictly comparable and assignment to a treatment group was neither random nor blind, the groups were comparable in terms of Trauma Scores, transport times, and IV success rates. A total of 161 patients were included in the study, and analysis showed that the PASG did not result in significant clinical improvement.

This all offers us a sobering lesson. There is of course much work to be done before the precise role of this device is clarified. However, it should serve to warn us of the hazards of enthusiastically embracing a new modality until it has been thoroughly evaluated. It also should give us some cause for satisfaction that the field of Emergency Medicine is capable of the sort of rigorous questioning and reappraisal that leads to a better understanding of how to care for the ill and injured patient.

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Pesticide poisonings: don't let them bug you

MILTON TENENBEIN MD, FRCP(C)

The patient presenting to the emergency department because of an acute exposure to a pesticide is not an uncommon occurrence. If the history is clear and the symptom complex is typical (e.g. classic cholinergic syndrome after malathion ingestion) then the diagnosis and management are straightforward. But in most cases, the product's identity is unknown and there are no or vague clinical findings. Because there are many different pesticides, each with a varying degree of toxicity, the physician is commonly unsure of how best to handle the situation; but an orderly approach plus a basic understanding of the toxicities of the major pesticide classes will go a long way towards the solution of this problem.

A pesticide can be thought of as any substance capable of destroying an undesirable organism. In Canada, any product used for pest control is subject to the regulations of the Pest Control Products (PCP) Act. It requires that all pesticides must be registered with the federal Department of Agriculture. The intent of the registration process is to ensure product identification, safety and efficacy. This act defines a pest as any injurious, noxious or troublesome insect, fungus, bacterium, virus, weed or rodent. Products subject to the Food and Drug Act are exempt from the PCP Act. Examples would be antibiotics or antiseptics that are used in humans, domestic animals or in food processing industries.

Before considering the toxicology of these products, let us first consider a few pointers regarding the general approach to the problem. After ensuring that the patient is not at immediate risk (satisfactory vital signs and level of consciousness) the history is obtained. From the epidemiological point of view, most serious pesticide poisonings occur in rural areas or with those individuals involved with the manufacture or transport of these chemicals. The serious urban ones are usually due to suicidal gestures or to using the product contrary to package instructions. All three routes of absorption (gastrointestinal, respiratory and percutaneous) can result in systemic toxicity.

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From the Manitoba Poison Centre

Then attempt to identify the product. Identification is often impossible without having the pesticide container in hand. If the container has not been brought in, send someone for it or have the label read to you over the telephone. The following label information is helpful: product name, active ingredient, PCP Act registration number, product classification and toxicity information. If the container is absolutely unavailable, attempt to determine if the product is a herbicide, insecticide or rodenticide.

The registration number (which is four or five digits) can be used in difficult situations. Poison control centres can identify the product and provide treatment advice when provided with this number.

The product classification system has three categories: domestic, commercial and restricted. Classification is based upon relative toxicity. Domestic products can be purchased by lay people for use in and around their homes. Commercial products are intended for professionals engaged in commercial activities such as farming and extermination. Restricted products are extremely hazardous and are limited to very specialized activities. In practice, it is quite unlikely to suffer ill effects from a domestic product which has been used in accordance with label instructions. An example would be the aerosol insecticides available in most supermarkets for use around the home.

The PCP Act requires a poison warning symbol for any product with an LD50 of 2500 mg/kg or less. Thus the absence of such a symbol is helpful. More dangerous pesticides must also have treatment advice on their labels. Although this advice may be helpful, it is at times inaccurate.

In cases where the potential exists for a toxic exposure then the appropriate decontamination procedures must be instituted. Depending upon the circumstances, these may include induction of emesis, gastric lavage, activated charcoal or topical decontamination by continuous irrigation or showering.

In order to manage individual cses, one must have knowledge regarding the toxicity of the product. In 1984, there were 4,580 products registered under the PCP Act. These products were formulated from 511 different ingredients. Obviously a system of classification is required. One based upon intended use and chemical class is most useful from the clinical point of view (see the table). Consideration of all pesticides is beyond the scope of this review. The most common herbicides, insecticides and rodenticides along with some commonly encountered repellants will be considered.

HERBICIDES

The ideal pesticide would exert its injurious action upon the undesirable target organism and spare desirable nontarget organisms. Because of the vast differences between plant and animal physiology, herbicides can and have been developed with minimal to negligible toxicity for humans. A useful rule of thumb is that

PESTICIDES

1 HERBICIDES

A. Chlorophenoxy Compounds
- 2, 4 - D
- 2, 4, 5 - T
- mecoprop
- dicamba

B. Carbamate Herbicides
- triallate
- maneb
- cycloate

C. Bipyridium Compounds
- paraquat
- diquat

D. Substituted Ureas
- monuron
- diuron

E. Triazines
- atrazine
- simazine
- prometon

2 INSECTICIDES

A. Organophosphates
- parathion
- malathion
- dichlorvos
- diazinon

B. Carbamates
- carbofuran
- propoxur
- carbaryl
- aldicarb

C. Organochlorines
- lindane
- methoxychlor
- dieldrin
- DDT

D. Pyrethrins and Pyrethroids
- pyrethrum
- pyrethrin
- allethrin

3 RODENTICIDES
- anticoagulants
- strychnine
- red squill

4 REPELLANTS
- N,N-diethyl-m-toluamide
- paradichlorobenzene
- naphthalene

except for the uncommon massive intentional overdose of a concentrated herbicide, toxicity other than transient gastrointestinal disturbances is unlikely. The important exception to this rule is the bipyridium group of herbicides, examples being paraquat and diquat. These herbicides are extremely toxic. Very small ingestions can be fatal.

Chlorophenoxy Compounds

The chlorophenoxy acids, salts and esters are the most common herbicides. They are found in most over the counter products sold for domestic weed control. Their mechanism of action is interesting. They function as plant growth hormones, causing the organism to overgrow its nutrient supply. They do not exert similar actions in humans in whom they have very low toxicity. In fact, 2,4-D has been used therapeutically in humans for the treatment of coccidiomycosis. Routine exposures (defined as accidental childhood ingestions or exposures experienced during the routine use of these products) are unlikely to result in anything other than transient irritation of skin, eyes, gastrointestinal or respiratory linings. an intentional ingestion of a large amount of undiluted product can result in metabolic acidosis, myotonia, myoglobinuria, coma and convulsions. Overdoses of this extent are extremely uncommon.

Carbamate Herbicides

Carbamate herbicides differ from carbamate insecticides in that they do not have anticholinesterase activity, thus exposures do not result in cholinergic symptoms. The likelihood of systemic toxicity is minimal. Gastrointestinal upset may occur. Thiram, which is used as a fungicide, is interesting because it inhibits aldehyde dehydrogenase. Antabuse-like reactions can occur if ethanol is ingested after exposures to large amounts of thiram. Such patients should be advised to avoid alcohol for 1–2 weeks.

Substituted Ureas and Triazines

The substituted ureas and the triazines are relatively nontoxic. They are not commonly used in Canada and no human cases of poisonings have been reported from either of these groups. Because of the metabolism of the ureas, there is at least a theoretical risk for methemoglobinemia.

Bipyridium Compounds

The bipyridium compounds, paraquat and diquat, are extremely toxic. Fatality can result from the ingestion of as little as 10–15 ml of the concentrate. All exposures should be considered as potentially fatal. Toxicity has also been documented by inhalation and percutaneous absorption. Except for a few dilute granular formulations, these herbicides are all classified as commercial or restricted products. Because of these restrictions, poisoning is uncommon in Canada.

The bipyridium herbicides promote the transformation of molecular oxygen into superoxide radicals which in turn leads to peroxidation of lipid cell membranes. Virtually all tissues of the body can be affected. Death can occur within 24 hours. In these situations necrosis of esophagus, lungs, liver, kidneys, heart and other organs is usually found. In milder cases, the patient may experience pulmonary toxicity beginning 1–2 weeks following the exposure. This may also be fatal. Late pulmonary toxicity occurs because these herbicides are selectively concentrated in the lungs.

Treatment of bipyridium herbicide poisoning is supportive. Administer adsorbents as soon as possible after ingestion. A 7% solution of bentonite in water is felt to be the most efficient, but if unavailable, use activated charcoal. If both are unavailable, have your patient eat dirt (top soil). Avoid supplemental oxygen. In some cases induced hypoxia has been used as a therapeutic approach. In most cases, charcoal hemoperfusion is used as it is generally felt to hasten the excretion of this toxin.

INSECTICIDES

As mentioned earlier, the ideal pesticide would selectively damage pests and not damage desirable organisms. If gross biologic dissimilarity exists (plant vs human) then this ideal is a reasonable goal. As biologic dissimilarity decreases, (insect vs human) absolute pesticidal selectivity becomes more difficult. Thus insecticides are generally more toxic to humans than herbicides. Insecticidal selectivity is usually dependent upon using amounts that are too small to poison larger organisms.

Organophosphates

Organophosphate insecticides inhibit cholinesterase by phosphorylating this enzyme. Poisoning with these agents results in cholinergic crisis. Both muscarinic and nicotinic effects may occur. The former includes miosis, lacrimation, salivation, diaphoresis, vomiting, diarrhea, bronchospasm, bronchorrhea, bradycardia, convulsions and coma. Nicotinic effects may include muscle cramps, fasciculations, muscular weakness and paralysis.

The lungs are the major target organs. The cause of the respiratory failure is multifactorial. Bronchoconstriction and bronchorrhea can be very severe. At times bronchoscopy for aspiration of secretions may be necessary despite heroic atropine therapy. Concomitant respiratory muscle paralysis contributes to the respiratory decompensation. Since many of these insecticides are solubilized in hydrocarbon solvents, an aspiration pneumonia may further aggravate respiratory function.

Atropine in large doses is the antidote of choice. If the diagnosis is in doubt, a therapeutic trial is indicated. Intravenously administer 1.0–2.0 mg to an adult or 0.25–0.5 to a child. If none of the usual effects of atropine are seen (mydriasis, increased heart rate or drying of mucosa) then the diagnosis is correct and more atropine is required. Atropine requirements in severe cases can be as high as several milligrams several times an hour and hundreds of milligrams over the entire course. Actual dosage requirements are titrated against the patient with the desired end point being clearing of pulmonary secretions and bronchospasm.

Pralidoxime is an adjunctive antidote to be used in addition to, and not instead of atropine. It counteracts the nicotinic effects of organophosphate poisoning and should be used if fasciculations, muscular weakness or paralysis is present. It reactivates cholinesterase by preventing irreversible phosphorylation by the insecticide. But, in order to be effective, administration must be begun within 36 hours of poisoning.

The above specific therapy does not diminish the need for meticulous supportive care. Supplemental oxygen, frequent respiratory suction and assisted ventilation may all be required. Other complications such as shock and convulsions may occur and require treatment. Measurement of cholinesterase levels may be interesting but they rarely contribute to patient management.

Carbamates

Carbamate insecticides are also cholinesterase inhibitors. The management of poisoning by these agents is identical to organophosphate poisoning. Since the carbamate-cholinesterase bond is more labile than the equivalent organophosphate bond, pralidoxime therapy is not indicated.

Organochlorines

The organochlorine insecticides are still in common usage. Apart from environmental pest control, one of them, lindane (Kwellada), is used as a drug for the control of human scabies. These insecticides are potent neurotoxins. They alter the electrophysiological properties of axonal membranes causing interference with transmission of nerve impulses. Poisoning may result in central nervous system excitement or depression. Symptoms and signs may include nervousness, agitation, tremor, convulsions, depression of vital centres and coma. Treatment is supportive.

Pyrethrins and Pyrethroids

Pyrethrins are refined from pyrethrum which is an extract from the chrysanthemum flower. Pyrethroids are synthetic compounds based structurally upon the pyrethrin molecule. Similar to the organochlorine insecticides, these substances rapidly paralyze the insect's nervous system by interfering with axonal transmission. Because mammals can rapidly catabolize these insecticides, they are remarkably resistant to them. These products are commonly formulated with piperonyl butoxide which synergistically enhances their effect upon the insect by blocking the metabolism of the insecticide. Such formulations are commonly found in over the counter "bug-bombs" (household aerosol insecticides). Although systemic toxicity is virtually unheard of, hypersensitivity to pyrethrins does occur. This is more of a problem for the workers involved in the manufacture of these products.

RODENTICIDES

The target organisms for these are usually mice or rats. As with insecticides, advantage is taken of the difference in size between target and nontarget organisms in order to produce products with selective toxicity.

Anticoagulants

The coumarin and indandione anticoagulants are the commonest rodenticides. They decrease the synthesis of various coagulation factors by the liver. Vitamin K counteracts the effects of both these groups.

The actual pesticide is usually grain or oatmeal that has been treated with one of these anticoagulants. However the concentration of active ingredient is very low. One of the more common mouse baits contains only 2.5 mg of active agent in the entire package of 50 gm of canary seed. In fact, the mouse must consume this bait on several days before being anticoagulated. If a toddler were to be anticoagulated from an acute ingestion, he would have to consume 10 packages or approximately one pound of mouse bait. Therefore ingestions of these products rarely, if ever, cause problems and the usual gastrointestinal decontamination procedures are not necessary.

Strychnine

Some rodent baits contain strychnine rather than anticoagulants. However, in Canada, strychnine poisoning from pesticides is rare because all such products bear the restricted classification and are therefore relatively

unavailable. Thus mouse or rat bait bought in the local supermarket will be anticoagulant and not strychnine based.

Strychnine is a potent and rapidly acting neurostimulant. Seizures may occur within 15–60 minutes of ingestion. It is probably unwise to administer ipecac after strychnine ingestion as seizures may occur during emesis. Activated charcoal alone is the safest decontamination procedure. Treat seizures with intravenous anticonvulsants. In many cases muscle paralysis with assisted ventilation will also be required.

Red Squill

Red squill is used for rat control. It contains cardiac glycosides and is a potent emetic. Human toxicity is quite uncommon because it is usually vomited after ingestion. However, rats cannot vomit and as a result they usually die of cardiac dysrhythmias and convulsions.

REPELLANTS

Repellants are not true pesticides in that they are not intended to kill the pest. But, because they are regulated by the PCP Act, we shall consider a few of the commoner ones.

Diethyltoluamide

N,N-diethyl-m-toluamide (DEET) is the commonest mosquito repellant in use today. It is available in many different types of physical formulations and concentrations. Although safe when used according to package instructions, purposeful ingestions of moderate amounts (e.g. 25 ml of 100% DEET) can have serious consequences. Depression of vital functions and toxic encephalopathy of rapid onset may occur. Respiratory depression, shock, convulsions and coma have been seen. Treatment is supportive.

Paradichlorobenzene

Paradichlorobenzene is the active ingredient in most mothballs. It is also used as a toilet bowl deodorant cake. Although it is an organochlorine, neurotoxicity is not a problem. Little other than nonspecific gastrointestinal complaints would be expected from a child who ingested 1–2 paradichlorobenzene mothballs.

Naphthalene

Some mothballs are still made of naphthalene which is more toxic than paradichlorobenzene. A hemolytic anemia may result which presents a few days after ingestion. Although normal individuals are at the most only mildly affected, those with G-6-PD deficiency are exquisitely sensitive to hemolysis. In these cases, less than one mothball may be fatal. Severe hemolysis with hemoglobinuria and renal failure can result. Naphthalene ingestors with Black, Oriental or Mediterranean ethnicity should be tested for G-6-PD deficiency on an emergent basis.

SUMMARY

The management of the patient presenting to the emergency department because of an acute exposure to a pesticide is simplified by an orderly approach to the problem. Careful history-taking with emphasis placed upon specifically identifying the product in question coupled with a basic understanding of pesticide classification will go a long way towards the management of most presentaions. Massive exposures, or exposures to less commonly encountered agents may require consultation with individuals or agencies with additional expertise.

The intent of this review is to discuss the commoner pesticides. More information can be obtained from Morgan's handbook[1] which is a very useful reference and should be purchased for most emergency departments. It is available from the Superintendent of Documents, U.S. Government Printing Office, Washington D.C. 20402. Hayes' book[2] is the definitive text for pesticide toxicology but is not suitable for the emergency department.

ADDITIONAL READING

1) Morgan DP: Recognition and management of pesticide poisonings. United States Environmental Protection Agency, 1982.
2) Hayes WJ: Pesticides studied in man. Williams and Wilkins; Baltimore, 1982.

Letters to the Editor

I am writing to bring to the attention of the Review's readers the creation of an International Section of the University Association for Emergency Medicine.

At its Annual Meeting earlier this spring, the University Association for Emergency Medicine identified as one of its priorities for the coming year the establishing and developing of ties with other national and international organizations and individuals interested and involved in Emergency Medicine Research.

UAEM's membership is comprised of individuals from varied backgrounds – surgery, anaesthesia, critical care, paediatrics, internal medicine and emergency medicine. Although diverse in background, they have been drawn together by their interest in the acutely ill and injured individuals.

There have been significant developments in Emergency Medicine in many countries around the world these past few years. Despite this "coming of age" of the specialty, there apparently exists no form for the dissemination of research in our specialty at an international level. There are a number of international societies in critical care and anaesthesia which address many of those issues which involve emergency medicine (viz. resuscitation, etc.); however, it is UAEM's intention to form an international forum specific to the needs and interests of Emergency Medicine. Although there is overlap between many of the traditional specialties and Emergency Medicine, the focus of UAEM is to promote research in the early evaluation and management of the emergent patient.

UAEM sponsors a number of activities including an annual meeting at which this past year 100 research papers in the field of Emergency Medicine were presented. As well, it hosts a yearly state-of-the-art symposium and is the co-sponsor of the "Annals of Emergency Medicine". With membership in UAEM, a free subscription is provided to this Journal and a reduced registration for the UAEM Annual Meeting is available.

Although the development of the Annual Meeting of the Royal College does provide us with a forum to present our research here in Canada – this does not preclude Canadian participation in UAEM. Indeed, I think it is particularly important that Canadian academics active in the field get involved in this larger forum. An international membership can only contribute to a greater dissemination of our research in Emergency Medicine.

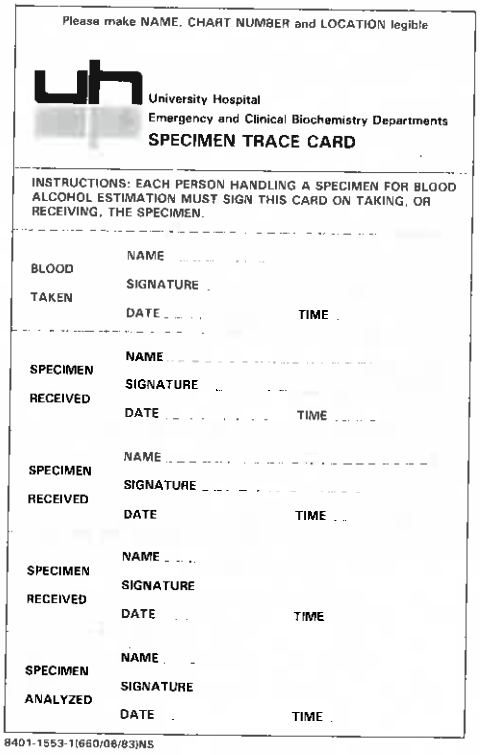
Should your readers be interested in joining UAEM and participating in its activities, they may contact me at the following address:

Dr. Bruce M.T. Rowat,
Chairman, Committee on International Affairs
University Association for Emergency Medicine
900 West Ottawa,
Lansing,
Michigan 48915

Until the recent past our Emergency nurses and Biochemistry Technologists have on occasion shared the frustration and embarrassment of having their testimony questioned regarding blood alcohol levels when subpoenaed for court cases. In recent discussion it has been brought to my attention that some centres are still having difficulty with this issue. For that reason I would submit for your consideration the form that was devised by the Emergency and Biochemistry Departments here at University Hospital.

The specimen trace card allows "continuity of evidence" by simply having everyone who touches the sample sign their name and the time at which the specimen was taken before handing it on to the next person in the chain. Obviously the easiest method would be to have only the technician draw the blood and take it directly to the machine but, as we are all aware, this is seldom a reality in a resuscitation room. This trace card allows a nurse or physician to draw the blood and simply to hold on to the alcohol sample or to any other specimen for that matter, until he is able to sign the card and pass on the sample.

Some may argue that the specimen taken for blood



Please make NAME, CHART NUMBER and LOCATION legible

University Hospital
Emergency and Clinical Biochemistry Departments
SPECIMEN TRACE CARD

INSTRUCTIONS: EACH PERSON HANDLING A SPECIMEN FOR BLOOD ALCOHOL ESTIMATION MUST SIGN THIS CARD ON TAKING, OR RECEIVING, THE SPECIMEN.

BLOOD TAKEN — NAME / SIGNATURE / DATE / TIME

SPECIMEN RECEIVED — NAME / SIGNATURE / DATE / TIME

SPECIMEN RECEIVED — NAME / SIGNATURE / DATE / TIME

SPECIMEN RECEIVED — NAME / SIGNATURE / DATE / TIME

SPECIMEN ANALYZED — NAME / SIGNATURE / DATE / TIME

8401-1553-1(660/06/83)NS

alcohol is no different from any other specimen but certainly in court cases it would appear that the handling of these samples is brought into question much more than others. Samples continue to be taken only with medical justification. This method simply gives support to hospital staff when they are subpoenaed to appear as witnesses in court cases.

I submit this specimen card for your perusal and consideration for distribution to your readers.

J.A. Cunningham MD
Director
Emergency Services
University Hospital
London, Ontario

Meetings to note

Title: Cardiology for the Primary Care Physician
Dates: March 22–24, 1985
Location: Palm Springs Spa Hotel, Palm Springs, CA
Accreditation: 18 hours, AMA/CMA, Prescribed AAFP, Category 2-D AOA
Course Coordinator: William A. Norcross, MD
For further information: CME Office, M-017, UCSD School of Medicine, La Jolla, CA 92093
Phone: (619) 452-3940

Title: Family Practice Refresher Course – 1985
Dates: March 25–29, 1985
Location: Palm Springs Spa Hotel, Palm Springs, CA
Accreditation: 30 hours, AMA/CMA, Prescribed AAFP, Category 2-D AOA
Course Coordinator: Howard D. Groveman, MD
For further information: CME Office, M-017, UCSD School of Medicine, La Jolla, CA 92093
Phone: (619) 452-3940

ATLS Provider Courses
Location: Sunnybrook Medical Centre, Toronto, Ontario
*Dates**: January 3–5 and 17–19, 1985
February 14–15 and February 28–March 2, 1985
March 28–30, 1985
April 11–13 and 25–27, 1985
May 9–11 and 23–25, 1985
June 6–8 and 20–22, 1985
September 5–7 and 19–21, 1985
October 3–5 and 17–19 and Oct. 31–November 2, 1985
November 14–16 and 28–30, 1985
December 12–14, 1985
Contact: Emergency Department Physicians Management Consultants Ltd. (416) 482-2247

*Subject to alteration

ACLS Provider Courses
Location: Sunnybrook Medical Centre, Toronto, Ontario.
*Dates**: January 18–20, 1985
February 1–3, 1985
March 29–31, 1985
April 26–28, 1985
May 24–26, 1985
June 21–23, 1985
Contact: Emergency Department Physicians Management Consultants Ltd. (416) 482-2247

*Subject to alteration

Title: 4th Annual Emergencies for Emergency Physicians
Dates: February 7–9, 1985
Location: Ottawa Civic Hospital
Contact: Dr. J.E. Devitt, Head, Medical Education, Ottawa Civic Hospital, 1053 Carling Ave., Ottawa, Ontario K1Y 4E9. Phone: (613) 725-4480

Title: Eighth Annual San Diego Postgraduate Assembly in Surgery
Dates: January 21–25, 1985
Location: Sheraton Harbor Island – East San Diego, California
Accreditation: Approximately 28 hours, AMA/CMA credit; 28 hours nursing credit
Course Coordinator: A. Gerson Greenburg, M.D.
For further information: Office of Continuing Medical Education, M-017, UC San Diego School of Medicine, La Jolla, CA 92093. Phone: (619) 452-3940

Title: Trauma Management 1985
Dates: January 28–30, 1985
Location: Vacation Village Resort, San Diego, California
Accreditation: Approximately 20 hours, AMA/CMA credit and nursing credit; other accreditation requests pending
Course Coordinator: Steven Shackford, M.D.
For further information: Office of Continuing Medical Education, M-017, UC San Diego School of Medicine, La Jolla, CA 92093. Phone: (619) 452-3940

EMERGENCY DOCTORS

Needed for the Emergency Room of the Welland County General Hospital, Niagara Peninsula. 42 hr. week, attractive salary and four week vacation.

Apply in writing to

Dr. E. Kouros, President
Welland Emergency Associates
c/o Welland County General Hospital
Third St., Welland
L3B 4W6

Director of Emergency Services
Grace General Hospital
Winnipeg, Manitoba

Applications are invited for the above full time salaried position which becomes vacant July 1, 1985. The Grace Hospital is a 300 bed secondary care hospital with approximately 34,000 emergency visits annually. The Director of Emergency Services is also Head of the Department of Emergency Medicine in the Hospital, and a member of the Medical Staff. There are 9 full time emergency physicians under the Director who are responsible for providing services in the Emergency Department, and acute care on the Hospital wards.

The Director works with and co-ordinates the activities of the emergency physicians including continuing education. Previous experience and/or training in Emergency Medicine is required, possession of specialist certification in emergency medicine, and administrative experience would be an asset.

Application accompanied by a curriculum vitae and names of two referees should be submitted to

Dr. J.H. Taylor, Medical Director
Grace General Hospital
300 Booth Drive, Winnipeg, Manitoba
R3J 3M7 (204) 837-0144

Full Time Emergency Physicians Required for Chedoke-McMaster Hospitals

McMASTER UNIVERSITY MEDICAL CENTRE DIVISION

This Unit has a moderate patient volume with additional in-hospital (cardiac arrests) and pre-hospital (i.e. Base Physician, etc.) responsibilities. It also functions as the Regional Sexual Assault Examination Centre, and it is planned to function as the Paediatric Trauma Centre.

The position involves limited clinical hours with a generous time allowance for academic pursuits related to all phases of education (i.e. including Royal College Program in Emergency Medicine, Family Medicine, undergraduate training, Paramedic training, ACLS/ATLS), administration and research. Applicants should be committed to teaching Emergency Medicine as a specialty, and also willing and able to develop and fulfil academic goals. This is a Faculty position in the Faculty of Health Sciences, Department of Family Medicine.

CHEDOKE HOSPITAL DIVISION

This position is a combined Emergency/Intensivist position made possible by the relatively low volume of activity in either Unit. Concurrently, the position requires active involvement in clinical care in the ICU/CCU, at cardiac arrests, and as back-up to base physician activities of the Pilot Paramedic Program.

This position is as part of five full time physicians covering the Emergency Medical needs of the Hospital 24 hours per day, 365 days of the year.

Applicants should have appropriate knowledge and skills to participate in basic critical care activities of a small hospital ICU/CCU.

Both positions are salaried and include 4 weeks vacation and 2 weeks conference leave per annum.
Preference will be given to those with higher qualifications in Emergency Medicine (e.g. F.R.C.P., C.C.F.P./E.R., A.B.E.M., etc.)

Applications to
F. Baillie, M.B., ChB., F.R.C.S.(C)
Head of Section, Emergency Services, Chedoke-McMaster Hospitals
1200 Main Street West, Hamilton, Ontario L8N 3Z5

In accordance with Canadian immigration requirements, this advertisement is directed to Canadian citizens and permanent residents.